Mid Term Conference of
Mumbai Hematology Group (MHG)
& Essence of EHA-2026

Charting the Future of Hematology: An In-Person Academic Odyssey

Featuring Distinguished International Guest Speakers

Friday-Sunday, 4th to 6th of September 2026

Venue:

Jio World Convention Centre
Bandra Kurla Complex
Bandra (East), Mumbai, India.
2nd Floor,
Room 206 A (Hall A), 206 B (Hall B),
203 (Hall C), 202 (Hall D), 207 (Hall E)

Click on the link underneath for registering:
https://registration.mhg-cme.com/

Mumbai Hematology Group

Executive committee

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Vice-President

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Dr. Vrinda Kulkarni

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Charting the Future of Hematology: An In-Person Academic Odyssey

Mumbai Hematology Group

Dear Colleagues & Students,

It is our distinct privilege to invite you to the Mid-Term Conference of the Mumbai Hematology Group (MHG) & Essence of EHA-2026.

In an era of digital fatigue, we are proud to announce this as a strictly in-person (physical) summit. This is a deliberate choice to foster the high-level scientific discourse, spontaneous networking, and personal camaraderie that only a face-to-face gathering can provide.

We have curated a faculty of eminent international experts to bridge the gap between global innovations and practical clinical application in both classical and malignant hematology.

Joining them is a "galaxy" of premier Indian faculty, bringing local context to the latest breakthroughs.

We also bring the most critical updates from the European Hematology Association (EHA) directly to you, distilled for maximum impact on your practice.

This is your premier opportunity to reconnect with mentors, engage with peers, and build the professional relationships that define our field.

We anticipate a record turnout this year. We urge you to register early and ensure your place at what promises to be the most vibrant and transformative educational event of 2026.

Let us gather in Mumbai to celebrate science, friendship, and the future of hematology.

Yours sincerely,

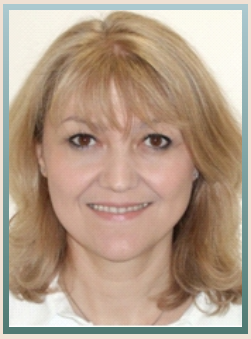
Dr. Rucha K. Patil
Secretary, MHG

Dr. M.B. Agarwal
President, MHG

Faculty - International


Dr. Cindy Neunert, NY


Dr. Daniel J. Hodson, UK


Dr. Dragana Milojkovic, UK

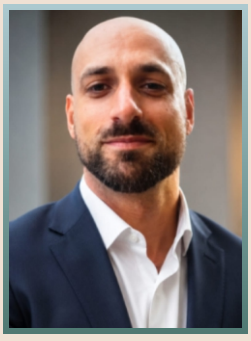

Dr. Karthik Ramasamy, UK


Dr. Mark Hertzberg, Sydney, Australia


Dr. Nicola Gökbuget, Germany


Dr. Rajshekhar Chakraborty, Columbia


Dr. Saad Z. Usmani, NY, USA


Dr. Matteo Piccini, Florence, Italy

Faculty - National

Dr. A.M.V.R. Narendra, Hyderabad
Dr. Aditya Jandial, Chandigarh
Dr. Amit Khurana, Surat
Dr. Aniket Mohite, Pune
Dr. Anjan Shrestha, Nepal
Dr. Anju Bharti, Varanasi
Dr. Ankit Batra, Dehradun
Dr. Anu Korula, Vellore
Dr. Anupama Borker, Goa
Dr. Anusree Prabhakaran, Pune
Dr. Arpita Bhattacharyya, Kolkata
Dr. Ashish Dixit, Bengaluru
Dr. Ashish Gupta, Jabalpur
Dr. Ashok Meshram, Kolkata
Dr. Asif Iqbal, Guwahati
Dr. Avinash Kumar Singh, Patna
Dr. Avriti Baveja, Dehradun
Dr. Biju George, Vellore
Dr. Dinesh Bhurani, Delhi
Dr. Dnyaneshwar Upase, Pune
Dr. Ganesh Jaishetwar, Hyderabad
Dr. Gaurav Dixit, Delhi
Dr. Gaurav Prakash, Chandigarh
Dr. Harshit Khurana, Delhi
Dr. J. M. Khunger, Delhi
Dr. Jasmina Ahluwalia, Chandigarh
Dr. Ramaswamy NV, Kochi
Dr. Javid Rasool Bhat, J & K
Dr. Jina Bhattacharyya, Guwahati
Dr. Kundan Mishra, Lucknow
Dr. Lalit Prashant Meena, Varanasi
Dr. Madhura Deshpande, Pune
Dr. Mahadeva Swamy BC, Goa
Dr. Mahesh Rajashekaraiah, Bengaluru
Dr. Mallikarjun Kalashetty, Bengaluru
Dr. Mayur Parihar, Kolkata
Dr. Naveen Gupta, Jaipur
Dr. Neeraj Arora, Ahmedabad
Dr. Neeraj Sidharthan, Kochi
Dr. Neha Kamble, Pune
Dr. Nehal Patel, Ahmedabad
Dr. Nikhil Kumar M, Kochi
Dr. Nilesh Wasekar, Nashik
Dr. Pankaj Tantia, Bikaner
Dr. Pavan Kumar Boyella, Hyderabad
Dr. Prakash Singh Shekhawat, Jaipur
Dr. Prashant Mehta, Faridabad
Dr. Prashant Sharma, Chandigarh
Dr. R. K. Jena, Cuttack
Dr. Rajan Kapoor, Leh
Dr. Rajib De, Kolkata
Dr. Ranjit Kumar Sahoo, Delhi
Dr. Rashmi Kushwaha, Lucknow
Dr. Rayaz Ahmed, Delhi
Dr. Reghu K. Sukumaran, Kolkata
Dr. Ritika Agrawal, Bikaner
Dr. Riya Ballikar, Nagpur
Dr. Rohan Haldar, Delhi
Dr. S. P. Shrivastava, Indore
Dr. Sanjeev, Lucknow
Dr. Sanjeevan Sharma, Delhi
Dr. Sanket Shah, Ahmedabad
Dr. Sayan Ghoshal, Howrah
Dr. Seema Bhatwardekar, Vadodara
Dr. Shailendra Prasad Verma, Lucknow
Dr. Sharat Damodar, Bengaluru
Dr. Shilpa Bhartia, Kolkata
Dr. Shivani Joshi, Nashik
Dr. Shouriyo Ghosh, Kolkata
Dr. Shruti Toshniwal, Aurangabad
Dr. Sohini Chattopadhyay, Nagpur
Dr. Sudarshan Pandit, Nashik
Dr. Sudha Sinha, Hyderabad
Dr. Suhani Barbhuiyan, Guwahati
Dr. Suman Mittal, Raipur
Dr. Sundar Shewale, Kolkata
Dr. Tathagata Chatterjee, Faridabad
Dr. Thejeswar N Prakasham, Hyderabad
Dr. Tribikram Panda, Bhubaneshwar
Dr. Uday Yanamandara, Pune
Dr. Upendra Sharma, Jaipur
Dr. Varun Bafna, Kolhapur
Dr. Venkatesh Ekbote, Sambhaji Nagar
Dr. Vijai Tilak, Varanasi
Dr. Vinay Bohara, Indore
Dr. Vinod Patil, Satara
Dr. Vipul Sheth, Delhi

Faculty - Local

Dr. Abhay Bhave
Dr. Aditi Shah Kaskar
Dr. Akshaya Mandloi
Dr. Alok Shetty
Dr. Ambreen Pandrowala
Dr. Amit Jain
Dr. Anant Gokarn
Dr. Anita Nadkarni
Dr. Ankita Pandey
Dr. Archana Swami
Dr. Archana Vazifdar
Dr. Asha Shah
Dr. Ashita Singhal
Dr. Ashok K. Yadav
Dr. Balkrishna Padate
Dr. Barnali Das
Dr. Bharat Agarwal
Dr. Bhausaheb Bagal
Dr. Bipin Kulkarni
Dr. Chetan Dhamne
Dr. Darshana Rane
Dr. Deep Gala
Dr. Digambar Panchal
Dr. Dipsha Kriplani
Dr. Farah Jijina
Dr. Gaurav Narula
Dr. Girish Rajadhyaksha
Dr. Hamza Dalal
Dr. Hasmukh Jain
Dr. Jai Juvekar
Dr. Jay Mehta
Dr. Jyoti Chirmade
Dr. Kalyani Bapat-Patil
Dr. Kanchanmala Ghorpade
Dr. Kiran A Ghodke
Dr. Kunal Goyal
Dr. Kunal Sehgal
Dr. Lingaraj Nayak
Dr. Mamta Manglani
Dr. Manisha Madkaikar
Dr. Manju Sengar
Dr. Meha Gupta
Dr. Namita Padwal
Dr. Naveen Khargekar
Dr. Naveen Vairamoorthy
Dr. Navin Khattry
Dr. Neha Garg
Dr. Nikhil Patkar
Dr. Nirmalya Roy Moulik
Dr. Nishant Jindal
Dr. P. G. Subramanian
Dr. Parth Ganatra
Dr. Prabhakar Kedar
Dr. Prashant Tembhare
Dr. Prashant Warang
Dr. Priti Mehta
Dr. Priyanka Moule
Dr. Punit Jain
Dr. Purva Kanvinde
Dr. Purvi Kadakia Kutty
Dr. Rajesh Patil
Dr. Rajesh Sawant
Dr. Reetu Jain
Dr. Ritika Khurana
Dr. Rucha Patil
Dr. Ruhi Mehra
Dr. S. D. Banavali
Dr. Sachin Punatar
Dr. Sameer Tulpule
Dr. Samir Shah
Dr. Santanu Sen
Dr. Shilpa Gupta
Dr. Shilpi Saxena
Dr. Shrinath Kshirsagar
Dr. Shrutika Java
Dr. Shubhangi Agale
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Dr. Sujata Sharma
Dr. Sumeet Mirgh
Dr. Swati Kanakia
Dr. Tapan Saikia
Dr. VA Arun
Dr. Varsha Vadera
Dr. Vidisha Mahajan
Dr. Vrinda Kulkarni

International faculty

Dr. Cindy Neunert, MD MSCS
Professor
Section Head
Pediatric Hematology
Columbia University Irving Medical Center
New York, NY, USA

- She did fellowship from University of Texas Southwestern Medical Center.
- She is an expert in benign hematological disorders.
- Her areas of research and specialization include immune thrombocytopenia, thrombosis and sickle cell disease.
- She is involved with the Intercontinental Cooperative ITP Study Group, which is an international study group advancing knowledge regarding the natural history of ITP in children and adults.
- She is author of the American Society of Hematology 2019 guidelines for immune thrombocytopenia and currently working on the committee for the 2026 update.
- She was past chair of the Pediatric ITP Consortium of North America (ICON) which is a group of 40 pediatric centers conducting studies on the biology of ITP as well as the conduct of patient-related and interventional trials.
- She is also the medical director for the Integrative Therapies Program at Columbia University Medical Center and Integrate Health Global.

International faculty

Dr. Daniel Hodson
CRUK Senior Research Fellow
Honorary Consultant Haematologist
Director, Clinical Academic Training Programme
CRUK Cambridge Centre
Cambridge Stem Cell Institute
University of Cambridge, Department of Haematology
The Jeffrey Cheah Biomedical Centre
Cambridge Biomedical Campus
Cambridge, UK

- Dr. Hodson studied medicine at Cambridge and Oxford Universities before training in clinical haematology in Cambridge. He undertook a University of Cambridge PhD in molecular immunology supervised by Dr Martin Turner at the Babraham Institute.
- In 2010 he moved as a Kay Kendall Leukaemia Fund (KKLF) Post-doctoral Fellow to the lab of Dr Louis Staudt at the National Cancer Institute, NIH, USA to study the functional genomics of aggressive B cell lymphoma.
- In 2015 he was awarded a Medical Research Council (MRC) Clinician Scientist Fellowship and returned to Cambridge to establish a research group in the Welcome MRC Cambridge Stem Cell Institute, University of Cambridge.
- In 2021 he was awarded a Cancer Research UK Senior Research Fellowship and in 2023 a European Research Council Consolidator award.
- The Hodson Group researches the molecular mechanisms that underlie lymphomagenesis with a view to identifying new targets for designer anti-lymphoma therapies.
- Key themes in his lab are molecular profiling (especially ctDNA) to resolve genetic heterogeneity of DLBCL, novel approaches to model the functional genetics of lymphoma, and the mechanistic contribution of RNA helicases in lymphomagenesis.
- Dr. Hodson is also a practicing clinician and an honorary Haematology consultant.
- He runs an active portfolio of clinical trials to study the effect of novel therapies in patients with lymphoma and chairs the UK NCRI Lymphoma Translational Science Subgroup.

International faculty

Dr. Dragana Milojkovic
Consultant Haematologist
Professor of Chronic Myeloid Malignancies
The Hammersmith Hospital
Imperial College
London, UK

- Dr. Dragana Milojkovic is a Consultant Haematologist and Professor of Chronic Myeloid Malignancies at The Hammersmith Hospital, Imperial College London, UK.
- She is a researcher and expertise focuses on chronic myeloid leukaemia, myeloproliferative neoplasms and transplantation.
- Professor Milojkovic, Chair of the UK CML sub-group 2019 - 2025.
- She is also Director of the Clinical Trials unit at The Hammersmith Hospital.
- She has published extensively in peer reviewed journals regarding the biology and clinical management of CML.
- She participates in CML ELN recommendations and BSH guidelines.
- She is a member of EBMT Chronic malignancies working party and EHA Specialized Working Group (SWG) for CML.

International faculty

Dr. Karthik Ramasamy, MBBS FRCP PhD FRCPath
Professor of Haematology
The University of Oxford and a Consultant Haematologist
Oxford University Hospitals NHS Foundation Trust

- He completed his general medical training in Nottingham, followed by initial haematology training at Glasgow Royal Infirmary.
- In 2002, he relocated to London to engage in an extensive haematology training programme at Kings College Hospital, covering all haematology subspecialties, including bone marrow transplantation.
- He further pursued his interest in cancer research by serving as a clinical research fellow at Kings College London for three years.
- Currently, Dr. Karthik Ramasamy practices at both Oxford University Hospital NHS Trust and Royal Berkshire, specializing in haematology.
- He is the myeloma lead for the Thames Valley Cancer Network and serves as Chief Investigator for UK multicentre trials.
- His dedication to research is evident in his authorship of over 75 peer-reviewed articles and contributions to haemato-oncology textbooks.
- This commitment ensures he remains at the forefront of the latest advancements, offering his patients optimal diagnosis and treatment.
- He is proficient in various medical procedures and treatments, including blood transfusion, coagulation, chemotherapy, and bone marrow transplantation.
- His expertise extends to managing conditions such as myeloma, thrombosis, blood disorders, blood cancer, and plasma cell dyscrasias, reflecting his comprehensive knowledge in haematological oncology and general haematology.

International faculty

Dr. Mark Hertzberg, AM, MBBS PhD FRACP FRCPA
Prof & Head
Dept of Clinical Haematology
Prince of Wales Hospital and University of NSW
Sydney, Australia

- Dr. Mark Hertzberg is an immediate previous Head of Clinical Haematology at Prince of Wales Hospital and University of NSW, Sydney.
- He is a past President of HSANZ (Haematology Society Australia New Zealand); a previous Chair of the Scientific Advisory Committee of the ALLG (Australasian Leukaemia Lymphoma Group) and ALLG High Grade Lymphoma Group; a current ALLG Board Member; an HSANZ Pitney Travelling Fellow.
- His research interests are in the areas of lymphoma, clinical trials, and molecular monitoring of blood cancers. He received his Order of Australia in January 2022 for services to Haematology research and education.

International faculty

Dr. Matteo Piccini
Consultant Hematologist
AOU Careggi University Hospital
Florence
Italy

- Dr. Matteo Piccini is a Consultant Hematologist at AOU Careggi University Hospital, Florence, Italy, a position he has held since 2020.
- He earned his MD and completed his specialization in Hematology from the University of Florence, graduating with the highest honors.
- Dr. Piccini's clinical and research interests focus on acute myeloid leukemia (AML), acute lymphoblastic leukemia (ALL), blastic plasmacytoid dendritic cell neoplasm (BPDCN), measurable residual disease (MRD), flow cytometry, and precision medicine.
- He is actively involved in the management of hematologic malignancies and serves as both a principal and sub-investigator in several Phase I-III international clinical trials.
- A recognized contributor to leukemia research, Dr. Piccini has authored and co-authored more than 50 peer-reviewed publications in leading international hematology journals.
- His work has focused on AML genomics, MRD-guided treatment strategies, targeted therapies, and innovative therapeutic approaches, helping advance the understanding and treatment of leukemia.

International faculty

Dr. Nicola Gökbuget
Haematologist and clinical researcher
Dept. of Medicine II
Goethe University Hospital
Frankfurt

- Dr Nicola Gökbuget has worked at the University Hospital Frankfurt, Germany, for more than 25 years and as senior physician, she heads the study center of the Department of Medicine II.
- She is task force director for clinical trials of the University Cancer Center, Principal Investigator of the Frankfurt Cancer Institute.
- She serves as Managing Director of the Central Clinical Trial Coordination of the Goethe University and University Hospital.
- She is coordinating or principal investigator of numerous academic or industry-sponsored trials in adult acute lymphoblastic leukemia (ALL) and related diseases such as lymphoblastic lymphoma or Burkitt's lymphoma.
- For more than 20 years, she has served as chair of the German Multicenter Study Group for ALL (GMALL) with more than 140 participating hospitals across Germany.
- She founded and established a national registry for ALL with an associated biobank and has chaired the European Hematology Association Scientific Working Group for ALL.
- She is a founding member and board member of the German Network for Acute and Chronic Leukemias and the European Leukemia Network (ELN).
- She is also a founding member of the ELN Working Group for Adult ALL and is corresponding author of the ELN guidelines for adult ALL.

International faculty

Dr. Rajshekhar Chakraborty, MD
Assistant Professor of Medicine
Columbia University Irving Medical Center
Herbert Irving Comprehensive Cancer Center
Multiple Myeloma and Amyloidosis Program
Columbia, New York, USA

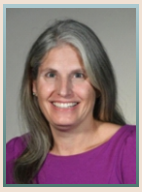
- Dr. Rajshekhar Chakraborty, MD is an assistant professor of medicine at Columbia University Irving Medical Center in the division of hematology/oncology.
- After receiving his medical degree from University of Delhi, India, he completed his residency in internal medicine at Icahn School of Medicine at Mount Sinai-Queens Hospital Center.
- Subsequently, he spent three years as a clinical researcher in plasma cell disorders at the Mayo Clinic, Rochester, followed by fellowship in hematology/oncology at the Cleveland Clinic.
- Dr. Chakraborty primarily cares for patients with plasma cells disorders, including multiple myeloma, amyloidosis, MGUS, POEMS syndrome, and WM.
- His research interests include clinical trials and outcomes research in plasma cell disorders.

International faculty

Dr. Saad Z. Usmani, MD, MBA, FACP
Hematologic Oncologist
Chief of the Myeloma Service
Memorial Sloan Kettering Cancer centre
New York, USA

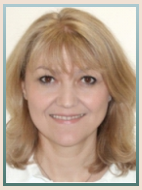
- Dr. Saad Z. Usmani is a hematologic oncologist with more than 14 years of experience specializing in the care of patients with multiple myeloma and other disorders affecting plasma cells.
- As a translational investigator, he has worked individually and as part of teams that have shed new light on the biology, pathogenesis, and therapy of these disorders and has been involved with the clinical development of several therapies approved by the FDA for patients with multiple myeloma and other blood cancers.
- His research has brought to the forefront the racial disparities impacting the treatment and response to multiple myeloma.
- Outside of the clinic and the lab, Dr. Usmani can be found training for a race or an endurance event to raise funds for cancer research.
- He completed the 2021 New York City Marathon as a member of MSK's Fred's Team and has previously run the Boston Marathon and completed the Mount Everest Base Camp trek.
- Dr. Usmani completed his medical education in Pakistan before completing an internal residency training at Sinai-Grace Hospital/Wayne State University, in Detroit, and a hematology and medical oncology fellowship at the University of Connecticut.
- He received his master's in business administration from the University of North Carolina Kenan-Flagler Business School.

Lectures by international faculty



Dr. Cindy Neunert, New York
a. When a low platelet count is not ITP
b. Secondary ITP: what do we know ?
c. Pediatric Thrombosis: Guidelines and Clinical Practice Updates
d. Hemophilia Guidelines: Application in the real world
e. Real-world data in Pediatric ITP
f. Case discussion session

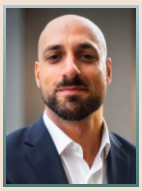
Dr. Daniel J. Hodson, Cambridge, UK
a. Is circulating tumour DNA set to revolutionize how we manage DLBCL ?
b. How can biology inform therapy in DLBCL?
c. Sex-specific roles of DDX3 RNA helices in aggressive B cell lymphomas
d. Case discussion session



Dr. Dragana Milojkovic, London
a. Management of the side-effects of TKI therapy
b. Balancing the choice of TKI therapy in CML- a new era
c. Treatment free remission in CML - the next frontier
d. Case discussion session

Dr. Karthik Ramasamy, UK
a. Dual targets, new possibilities:
BCMA & GPRC5D in relapsed refractory multiple myeloma
b. MRD in myeloma
c. Talquetamab - the new horizon
d. Close group meeting on Bispecific: New Era

Dr. Mark Hertzberg, Sydney, Australia
a. BrECADD decoded: practical insights for frontline HL management
b. Panel discussion: Adcetris expanded choice in frontline Hodgkin's Lymphoma



Dr. Matteo Piccini, Florence, Italy
a. Insights from Clinical Practice: Xospata Monotherapy in R/R FLT3+ AML
b. Case based panel discussion on Xospata monotherapy in R/R FLT3+ AML patients

Dr. Nicola Gökbuget, Germany
a. Treatment of B-precursor ALL - Ph-negative
b. Treatment of B-precursor ALL - Ph positive
c. Case discussion session
d. Treatment of T-ALL

Dr. Rajshekhar Chakraborty, Columbia
a. High-risk smoldering myeloma: how do I define and how do I treat ?
b. AL Amyloidosis: prognostic and predictive biomarkers.
c. Elranatamab in R/R Multiple Myeloma : current evidence and practical implications
d. Daratumumab is the game changer: from induction to maintenance.
e. Multiple myeloma: I do not want to kill my patient by infection.
f. Teclistamab: indications - today's and tomorrow's
g. Case discussion session

Dr. Saad Z. Usmani, New York, NY, USA
a. One Target, Multiple Possibilities: BCMA-Directed Therapies in
Multiple Myeloma with a Spotlight on Antibody-Drug Conjugates

Mid Term Conference of

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Scientific Agenda

Friday, 4th September 2026

Scientific Agenda

Friday, 4th September 2026, Hall A (Room 206 A)

Anchor: Dr. Amit Khurana

Time	Subject	Speaker
Session 1		
9.00 am - 9.30 am	**Panel discussion** **Sub:** Optimizing outcomes of HSCT: where have we reached in India ? **Moderator:** Dr. Biju George, Vellore	**Panelists:** Dr. Aditi Shah Kaskar, Mumbai Dr. Navin Khattry, Mumbai Dr. Sameer Tulpule, Mumbai Dr. Sanjeevan Sharma, Delhi
Session 2		
Sponsor: **Adley**		
Chair: Dr. Navin Khattry, Dr. Nikhil Patkar, Dr. Bhausaheb Bagal, Dr. Sameer Tulpule		
9.30 am - 10.00 am	Practice changes following ASH-25/EHA-26 in LMICs: My thoughts!	Dr. Ranjit Kumar Sahoo, Delhi
10.00 am - 10.30 am	The Molecular Underworld: Uncovering unusual Drivers in Myeloid Malignancies	Dr. Neha Kamble, Pune
10.30 am - 11.15 am	**Coffee**	
Session 3		
Sponsor: **Intas Aquila**		
Chair: Dr. Biju George, Dr. Ranjit Kumar Sahoo, Dr. Kunal Goyal		
11.15 am - 11.45 am	CAR-T cell therapy in B-ALL: our experience with a focus on those who received it as a consolidation	Dr. Hasmukh Jain, Mumbai
11.45 am - 12.15 pm	Beyond the Tail of the Curve: Engineering the Next Curative Frontier in NHL	Dr. Manju Sengar, Mumbai
12.15 pm - 1.15 pm	**Inauguration**	
1.15 pm - 2.00 pm	**Lunch**	
Session 4		
Sponsor: **Lucien**		
Chair: Dr. Sanjeevan Sharma, Dr. Rajan Kapoor, Dr. Aditi Shah Kaskar		
2.00 pm - 2.30 pm	When a low platelet count is not ITP !	Dr. Cindy Neunert, USA
2.30 pm - 3.30 pm	Dual targets, new possibilities: BCMA & GPRC5D in relapsed refractory multiple myeloma	Dr. Karthik Ramasamy, UK Dr. M. B. Agarwal, Mumbai

Scientific Agenda

Friday, 4th September 2026, Hall A (Room 206 A)

Anchor: Dr. Amit Khurana

Time	Subject	Speaker
Session 5		
Sponsor: **Zydus**		
Chair: Dr. Bhausaheb Bagal, Dr. Suman Mittal, Dr. VA Arun		
3.30 pm - 4.00 pm	Treatment of B-Precursor Ph-ve ALL	Dr. Nicola Gökbuget, Germany
4.00 pm - 4.30 pm	High risk smoldering myeloma: how do I define and how do I treat ?	Dr. Rajshekhar Chakraborty, USA
4.30 pm - 5.00 pm	**High Tea**	
Session 6		
Sponsor: **Natco**		
Chair: Dr. Bharat Agarwal, Dr. Sanjeevan Sharma, Dr. Archana Swami		
5.00 pm - 5.30 pm	Secondary ITP: What do we know ?	Dr. Cindy Neunert, USA
5.30 pm - 6.00 pm	Is circulating tumour DNA set to revolutionalize how we manage DLBCL ?	Dr. Daniel J. Hodson, UK
6.00 pm - 6.30 pm	Management of the side effects of TKI therapies	Dr. Dragana Milojkovic, UK
Session 7		
Sponsor: **Adley**		
Chair: Dr. Subhaprakash Sanyal, Dr. Bhausaheb Bagal, Dr. Kunal Goyal		
6.30 pm - 7.00 pm	Treatment of B-precursor Ph+ve ALL	Dr. Nicola Gökbuget, Germany
7.00 pm - 7.30 pm	AL Amyloidosis: Prognostic and predictive biomarkers	Dr. Rajshekhar Chakraborty, USA

Scientific Agenda

Friday, 4th September 2026, Hall B (Room 206 B)

Anchor: Dr. Bipin Kulkarni

Time	Subject	Speaker
Session 1		
9.00 am - 9.30 am	Panel discussion Sub: Refractory ITP Moderator: Dr. Mamta Manglani, Mumbai	Panelists: Dr. Amit Jain, Mumbai Dr. Anjan Shrestha, Nepal Dr. Ashita Singhal, Mumbai Dr. Jai Juvekar, Mumbai Dr. Parth Ganatra, Mumbai Dr. Purva Kanvinde, Mumbai Dr. Shweta Bansal, Mumbai
Session 2		
Sponsor: **Zydus**		
Chair: Dr. Mamta Manglani, Dr. Manisha Madkaikar, Dr. Purva Kanvinde		
9.30 am - 10.00 am	TPO mimetics beyond Pediatric ITP: Shifting Paradigms	Dr. Purvi Kadakia Kutty, Mumbai
10.00 am - 10.30 am	Neonatal Fc receptor: Biology & Therapeutics	Dr. Rajan Kapoor, Delhi
10.30 am - 11.15 am	**Coffee**	
11.15 am - 11.45 am	Combined Session in Hall A & B (Room 206 A & B)	
11.45 am - 12.15 pm	Combined Session in Hall A & B (Room 206 A & B)	
12.15 pm - 1.15 pm	**Inauguration in Hall A & B (Room 206 A & B)**	
1.15 pm - 2.00 pm	**Lunch**	
Session 3		
Sponsor: **Lucien**		
Chair: Dr. Swati Kanakia, Dr. Archana Swami, Dr. Purvi Kadakia Kutty		
2.00 pm - 2.30 pm	From Paediatric Oncologist to Infection Preventionist: My Journey as the Infection Control Officer at a Tertiary Cancer Centre	Dr. Arpita Bhattacharyya, Kolkata
2.30 pm - 3.00 pm	Childhood ALL: A decade of ICiCle & Immunotherapies- Lessons learnt & the Way Forward	Dr. Gaurav Narula, Mumbai
3.00 pm - 3.30 pm	Pattern of Asparaginase toxicities in Pediatric ALL	Dr. Chetan Dhamne, Mumbai

Scientific Agenda

Friday, 4th September 2026, Hall B (Room 206 B)

Anchor: Dr. Bipin Kulkarni

Time	Subject	Speaker
Session 4		
Sponsor: **Dr. Reddy's**		
Chair: Dr. Gaurav Narula, Dr. Chetan Dhamne, Dr. Ritika Khurana		
3.30 pm - 4.00 pm	Pharmacogenomics: day to day application	Dr. Swati Kanakia, Mumbai
4.00 pm - 4.30 pm	Survivorship in Pediatric Cancers	Dr. Suhani Barbhuiyan, Guwahati
4.30 pm - 5.00 pm	**High Tea**	
Session 5		
Sponsor: **Intas**		
Chair: Dr. Rajan Kapoor, Dr. Shweta Bansal, Dr. Parth Ganatra		
5.00 pm - 5.30 pm	Stem Cell Transplant in Metabolic Disorders	Dr. Santanu Sen, Mumbai
5.30 pm - 6.00 pm	Deficiency of ADA2: The Hematology spectrum	Dr. Ambreen Pandrowala, Mumbai
6.00 pm - 6.30 pm	Sip by Sip, Stronger Sickle Cells: The Power of Liquid Hydroxyurea	Dr. Ritika Khurana, Mumbai
Session 6		
Sponsor: **Dr. Reddy's**		
Chair: Dr. Santanu Sen, Dr. Ambreen Pandrowala, Dr. Suhani Barbhuiyan		
6.30 pm - 7.00 pm	Pediatric Hodgkin Lymphoma: Salvage therapy in R/R disease	Dr. Reghu K. Sukumaran, Kolkata
7.00 pm - 7.30 pm	Juvenile Myelomonocytic Leukemia	Dr. Ankita Pandey, Mumbai

Scientific Agenda

Friday, 4th September 2026, Hall C (Room 203)

Anchor: Dr. Rucha Patil

Time	Subject	Speaker
Session 1		
Sponsor: **RPG**		
Chair: Dr. Jasmina Ahluwalia, Dr. J. M. Khunger, Dr. Meha Gupta		
9.00 am - 9.30 am	Beyond Pyruvate Kinase Deficiency: Spectrum of Rare Red Cell Enzymopathies in Unexplained Hemolytic Anaemia - An NIIH Experience	Dr. Prabhakar Kedar, Mumbai
9.30 am - 10.00 am	Is Intracellular Calcium Dysregulation a Common Molecular Mechanism in Hereditary Hemolytic Anemias ? Implications for Novel Therapeutic Targets	Dr. Prashant Warang, Mumbai
10.00 am - 10.30 am	Understanding Immunothrombosis	Dr. Jasmina Ahluwalia, Chandigarh
10.30 am - 11.15 am	**Coffee**	
Session 2		
Sponsor: **Adley**		
11.15 am - 11.45 am	**Panel discussion** **Sub:** The Community Hematopathologist: Bridging the Gap Between Microscope and Bedside **Moderator**: Dr. Akshaya Mandloi, Mumbai	**Panelists:** Dr. Archana Vazifdar, Mumbai Dr. Dipsha Kriplani, Mumbai Dr. Shivani Joshi, Nashik Dr. Shrutika Java, Mumbai Dr. Vidisha Mahajan, Mumbai
11.45 am - 12.15 pm	**Panel discussion** **Sub:** Targeted therapy in B ALL **Moderator:** Dr. Kunal Sehgal, Mumbai	**Panelists:** Dr. Ambreen Pandrowala, Mumbai Dr. Gaurav Narula, Mumbai Dr. Prashant Tembhare, Mumbai Dr. Subhaprakash Sanyal, Mumbai
12.15 pm - 1.15 pm	**Inauguration in Hall A & B (Room 206 A & B)**	
1.15 pm - 2.00 pm	**Lunch**	
Session 3		
Sponsor: **Concord**		
Chair: Dr. Tathagata Chatterjee, Dr. Shivani Joshi, Dr. Shrutika Java		
2.00 pm - 2.30 pm	The Harmonization of Biomarker Reports of Monoclonal Gammopathies	Dr. Barnali Das, Mumbai
2.30 pm - 3.00 pm	Genomic work up in Relapsed Acute Lymphoblastic Leukaemia	Dr. Mayur Parihar, Kolkata
3.00 pm - 3.30 pm	Review of Guidelines and evidence for Diagnostic work up of Acute leukemia in India	Dr. P. G. Subramanian, Mumbai

Scientific Agenda

Friday, 4th September 2026, Hall C (Room 203)

Anchor: Dr. Rucha Patil

Time	Subject	Speaker
Session 4		
Sponsor: **Lucien**		
Chair: Dr. P. G. Subramanian, Dr. Mayur Parihar, Dr. Dipsha Kriplani		
3.30 pm - 4.00 pm	Rare but Relevant: Diagnostic Lessons from Challenging Malignant Hematopathology Cases	Dr. Akshaya Mandloi, Mumbai
4.00 pm - 4.30 pm	Slide seminars in Hematopathology: A Potpourri of exciting cases	Dr. Tathagata Chatterjee, Faridabad
4.30 pm - 5.00 pm	**High Tea**	
Session 5		
Sponsor: **Lucien**		
Chair: Dr. Neha Kamble, Dr. Anju Bharti, Dr. Akshaya Mandloi		
5.00 pm - 5.30 pm	Role of flow cytometry in platelet function disorders	Dr. Vidisha Mahajan, Mumbai
5.30 pm - 6.00 pm	Laboratory challenges in haemostasis: Case- based solutions	Dr. Bipin Kulkarni, Mumbai
6.00 pm - 6.30 pm	DOACs vs Traditional Anticoagulants: Current Guidelines & Controversies	Dr. J. M. Khunger, Delhi
Session 6		
Sponsor: **Zydus**		
Chair: Dr. Jasmina Ahluwalia, Dr. Rashmi Kushwah, Dr. Vidisha Mahajan		
6.30 pm - 7.00 pm	Need of chromogenic assay in the current era for haemophilia	Dr. Rucha Patil, Mumbai
7.00 pm - 7.30 pm	The most common yet missed bleeding disorder: Diagnosing Von Willebrand Disease	Dr. Anju Bharti, Varanasi

Scientific Agenda

Friday, 4th September 2026, Hall D (Room 202)

Anchor: Dr. Jay Sheth, Mr. Kamleshwar Dubey

Time	Subject	Speaker
Session 1		
8.45 am - 9.00 am	Quiz for UG	Dr. Shilpi Saxena, Mumbai
9.10 am - 9.25 am	Quiz for PG	Dr. Kunal Goyal, Mumbai
9.35 am - 9.50 am	Quiz for Superspeciality	Dr. VA Arun, Mumbai
10.00 am - 10.15 am	Quiz for Pediatric Hematology Oncology	Dr. Priti Mehta, Mumbai
10.15 am - 10.30 am	Quiz for Hematopathology	Dr. Vidisha Mahajan, Mumbai
10.30 am - 11.15 am	**Coffee**	
Session 2		
Sponsor: **Sun Pharma**		
11.15 am - 11.45 am	Panel discussion **Sub:** Venetoclax based panel discussion **Moderator:** Dr. Sameer Tulpule, Mumbai	Panelists: Dr. Balkrishna Padate, Mumbai Dr. Bhausaheb Bagal, Mumbai Dr. Digambar Panchal, Mumbai Dr. Navin Khattry, Mumbai Dr. Rajan Kapoor, Delhi Dr. Rajesh Patil, Mumbai
Session 3		
11.45 am - 12.15 pm	Panel discussion **Sub:** Case based discussion on CLL **Moderator:** Dr. VA Arun, Mumbai	Panelists: Dr. Alok Shetty, Mumbai Dr. Anant Gokarn, Mumbai Dr. Ashok Meshram, Kolkata Dr. Jai Juvekar, Mumbai Dr. Uday Yanamandra, Pune
12.15 pm - 1.15 pm	**Inauguration in Hall A & B (Room 206 A & B)**	
1.15 pm - 2.00 pm	**Lunch**	
Session 4		
Sponsor: **Adley**		
Chair: Dr. S. D. Banavali, Dr. Namita Padwal, Dr. Jai Juvekar		
2.00 pm - 2.30 pm	Acute Lymphoblastic Leukemia in the Era of NGS: An Indian Clinician's Perspective	Dr Tribikram Panda, Bhubaneswar
2.30 pm - 3.00 pm	Veno-occlusive disease: both in transplant and Chemo-Immunotherapy settings	Dr. Sohini Chattopadhyay, Nagpur
3.00 pm - 3.30 pm	Transplants in tier three cities	Dr. Venkatesh Ekbote, Sambhaji Nagar

Scientific Agenda

Friday, 4th September 2026, Hall D (Room 202)

Anchor: Dr. Jay Sheth, Mr. Kamleshwar Dubey

Time	Subject	Speaker
Session 5		
Sponsor: **Astellas**		
Chair: Dr. Subhaprakash Sanyal, Dr. Archana Vazifdar, Dr. Shivani Joshi		
3.30 pm - 4.20 pm	Astellas sponsored session Insights from Clinical Practice: Xospata Monotherapy in R/R FLT3+ AML	Dr. Matteo Piccini, Italy
4.30 pm - 5.00 pm	**High Tea**	
Session 6		
Sponsor: **Zydus**		
Chair: Dr. Tribikram Panda, Dr. Venkatesh Ekbote, Dr. Sohini Chattopadhyay		
5.00 pm - 5.30 pm	Hairy cell leukemia	Dr. Ramaswamy NV, Ernakulam
5.30 pm - 6.00 pm	Infections and immune dysfunction with bispecifics and CAR-T in Multiple Myeloma	Dr. Aditya Jandial, Chandigarh
Session 7		
Sponsor: **Intas**		
Chair: Dr. Ramaswamy NV, Dr. Aditya Jandial, Dr. Suman Mittal		
6.00 pm - 6.30 pm	Use of granulocyte transfusions in transplant	Dr. Anu Korula, Vellore
6.30 pm - 7.00 pm	Infections following BTK inhibitors	Dr. Mahesh Rajashekaraiah, Bengaluru
Session 8		
Sponsor: **Cipla**		
7.00 pm - 7.30 pm	Sponsored session by Cipla TBD	TBD

Scientific Agenda

Friday, 4th September 2026, Hall E (Room 207)

Anchor: Mr. Vivek Thorat / Mr. MSJ

Time	Subject	Speaker
Judges: Dr. Hamza Dalal, Dr. Shilpi Saxena, Dr. Sujata Sharma		
9.30 am - 10.30 am	Oral paper presentation session O-1 to O-6	
10.30 am - 11.15 am	**Coffee**	
11.15 am - 12.15 pm	Oral paper presentation session O--7 to O-12	
12.15 pm - 1.15 pm	**Inauguration in Hall A & B (Room 206 A & B)**	
1.15 pm - 2.00 pm	**Lunch**	
2.00 pm - 4.30 pm	Oral paper presentation session O-13 to O-27	
4.30 pm - 5.00 pm	**High Tea**	
5.00 pm - 7.30 pm	Oral paper presentation session O-28 to O-38	

Mid Term Conference of

Mumbai Hematology Group (MHG) & Essence of EHA-2026

Scientific Agenda

Saturday, 5th September 2026

Scientific Agenda

Saturday, 5th September 2026, Hall A (Room 206 A)

Anchor: Dr. Amit Khurana

Time	Subject	Speaker
Session 1		
9.00 am - 9.30 am	Panel discussion Sub: Myelofibrosis - how do I manage ? Moderator: Dr. Rajesh Patil, Mumbai	Panelists: Dr. A.M.V.R. Narendra, Hyderabad Dr. Ashok K. Yadav, Mumbai Dr. Kalyani Bapat-Patil, Mumbai Dr. Shruti Toshniwal, Aurangabad Dr. Suman Mittal, Raipur Dr. Varun Bafna, Kolhapur
Session 2		
Sponsor: **Adley**		
Chair: Dr. Manisha Madkaikar, Dr. Shilpa Gupta, Dr. Kalyani Bapat-Patil		
9.30 am - 10.00 am	Hydroxyurea Dosing in Indian Patients with Sickle Cell Disease: Results of a Six-Center Randomised Controlled Trial	Dr. Naveen Khargekar, Mumbai
Session 3		
Sponsor: **MICRO CRISPR**		
Chair: Dr. Gaurav Dixit, Dr. Mallikarjun Kalashetty		
10.00 am - 10.30 am	Transforming R/R B-cell Acute Lymphoblastic Leukemia treatment landscape: The evolving role of CAR-T therapy in clinical practice	Dr. Vipul Sheth, Delhi
10.30 am - 11.00 am	**Coffee**	
Session 4		
Sponsor**: Novartis**		
11.00 am - 11.40 am	Novartis sponsored session Panel discussion Sub: Choosing the Optimal Firstline treatment in CML Moderator:Dr. R K Jena, Cuttack	Panelists Dr. Ashok Meshram, Kolkata Dr. Nilesh Wasekar, Nashik Dr. Varun Bafna, Kolhapur
Session 5		
Sponsor**: BMS**		
11.40 am - 12.00 noon	BMS sponsored session From Transfusion Dependence to independence in Frontline MDS: COMMANDing Results with Rojuzda	Dr. Mahadeva Swamy BC, Goa
Session 6		
Sponsor**: Zydus**		
Chair: Dr. Venkatesh Ekbote, Dr. Ramaswamy NV, Dr. Aditya Jandial		
12.00 noon - 12.30 pm	Light chain Amyloidosis- diagnosis and treatment	Dr. Naveen Gupta, Jaipur
12.30 pm - 1.00 pm	Is low-dose nivolumab adequate for Hodgkin Lymphoma ?	Dr. Anusree Prabhakaran, Pune
1.00 pm - 2.00 pm	**Lunch**	

Scientific Agenda

Saturday, 5th September 2026, Hall A (Room 206 A)

Anchor: Dr. Amit Khurana

Time	Subject	Speaker
Session 7		
Sponsor: **Roche**		
2.00 pm - 3.00 pm	Panel discussion Sub: Navigating DLBCL in 2026: a case-based symposium Moderator: Dr. Bhausaheb Bagal, Mumbai	Panelists Dr. Gaurav Dixit, Delhi Dr. Pavan Kumar Boyella, Hyderabad Dr. Sanjeev, Lucknow Dr. Seema Bhatwadekar, Vadodara Dr. Shouriyo Ghosh, Kolkata Dr. Subhaprakash Sanyal, Mumbai
Session 8		
Sponsor: **Takeda**		
3.00 pm - 3.30 pm	BrECADD decoded: practical insights for frontline HL management	Dr. Mark Hertzberg, Australia
3.30 pm - 4.00 pm	Panel discussion Sub: Adcetris expanded choice in frontline Hodgkin's Lymphoma Moderator: Dr. M. B. Agarwal, Mumbai	Panelists Dr. Abhay Bhave, Mumbai Dr. Alok Shetty, Mumbai Dr. Ashish Dixit, Bengaluru Dr. Kunal Goyal, Mumbai Dr. Mark Hertzberg, Australia Dr. Sameer Tulpule, Mumbai Dr. Shouriyo Ghosh, Kolkata
Session 9		
4.00 pm - 4.30 pm	MRD in Myeloma	Dr. Karthik Ramasamy, UK Dr. Prashant Tembhare, Mumbai
4.30 pm - 5.00 pm	**High Tea**	
Session 10		
Sponsor: **Zydus**		
Chair: Dr. Reetu Jain, Dr. Bhausaheb Bagal, Dr. Darshana Rane		
5.00 pm - 5.30 pm	Recent Advances in management of Castleman's Disease	Dr. Amit Khurana, Surat
5.30 pm - 6.00 pm	POEMS Syndrome: Unique presentation of this rare paraneoplastic syndrome and its management	Dr. Priyanka Moule, Mumbai
Session 11		
Sponsor: **AstraZeneca**		
Chair: Dr. Reetu Jain, Dr. Bhausaheb Bagal, Dr. Darshana Rane		
6.00 pm - 6.30 pm	Lecture supported by AstraZeneca Eculizumab is the way to go for treating Paroxysmal Nocturnal Hemoglobinuria	Dr. Ashok Meshram, Kolkata
Session 12		
Chair: Dr. Reetu Jain, Dr. Bhausaheb Bagal, Dr. Darshana Rane		
6.30 pm - 7.30 pm	Talquetamab - the new horizon	Dr. Karthik Ramasamy, UK Dr. M. B. Agarwal, Mumbai

Scientific Agenda

Saturday, 5th September 2026, Hall B (Room 206 B)

Anchor: Dr. Bipin Kulkarni

Time	Subject	Speaker
Session 1		
Chair: Dr. Vinod Patil, Dr. Mahesh Rajashekaraiah, Dr. Anu Korula		
9.00 am - 9.30 am	Use of Anti-CD19 CAR-T in Primary CNS Lymphoma: A case-based discussion	Dr. Naveen Vairamoorthy, Mumbai
Session 2		
9.30 am - 10.00 am	Panel discussion Sub: Therapeutic implications of biology of DLBCL Moderator: Dr. Jay Mehta, Mumbai	Panelists: Dr. Gaurav Prakash, Chandigarh Dr. Manju Sengar, Mumbai Dr. Sanjeev, Lucknow Dr. Subhaprakash Sanyal, Mumbai Dr. Tathagata Chatterjee, Faridabad
Session 3		
Sponsor: **Novo Nordisk**		
10.00 am - 10.30 am	Innovation in Action: Enhancing Patient Autonomy and Haemostatic Control with Concizumab	
	Chair: Dr. Mamta Manglani, Mumbai	
	Lecture: Enabling Patient Autonomy and Normalcy with an Innovative Device	Dr. Amit Khurana, Surat
	Lecture: Normalizing Thrombin Generation with Steady-State Control and Precision Dosing	Dr. Dnyaneshwar Upase, Pune
10.30 am - 11.00 am	**Coffee**	
Session 4		
Sponsor: **Sanofi**		
11.00 am - 11.20 am	A new era of Hemophilia management - Rebalancing agents	Dr. Varun Bafna, Kolhapur
11.20 am - 11.35 am	Antithrombin Testing	TBD
11.35 am - 11.50 am	Panel discussion Sub: Unmet need and potential role of rebalancing agents in Haemophilia care Moderator: Dr. Varun Bafna, Kolhapur	Panelists: Dr. Jai Juvekar, Mumbai Dr. Nehal Patel, Ahmedabad Dr. Rucha Patil, Mumbai

Scientific Agenda

Saturday, 5th September 2026, Hall B (Room 206 B)

Anchor: Dr. Bipin Kulkarni

Time	Subject	Speaker
Session 5		
Sponsor: **Roche**		
12.00 noon - 12.30 pm	Session supported by Roche Panel discussion Sub: Framework for Selecting Better Therapies for Hemophilia A **Moderator:** Dr. Varun Bafna, Kolhapur	Panelists: Dr. Dnyaneshwar Upase, Pune Dr. Jai Juvekar, Mumbai Dr. Madhura Deshpande, Pune
Session 6		
Sponsor: **Abbvie**		
12.30 pm - 1.00 pm	Session supported by Abbvie Management of IC-Ineligible AML: Real-World Evidence and Clinical Insights	Dr. Subhaprakash Sanyal, Mumbai
1.00 pm - 2.00 pm	**Lunch**	
Session 7		
2.00 pm - 2.30 pm	Panel discussion **Sub:** All oral fixed duration therapy for ND CLL **Moderator:** Dr. Sharat Damodar, Bengaluru	Panelists: Dr. A.M.V.R. Narendra, Hyderabad Dr. Lingaraj Nayak, Mumbai Dr. Sanjeev, Lucknow Dr. Shilpa Bhartia, Kolkata Dr. Varun Bafna, Kolhapur Dr. Vinod Patil, Satara
Session 8		
Sponsor: **Accuren**		
2.30 pm - 3.00 pm	Panel discussion **Sub:** Lower toxicity approaches for B-ALL **Moderator:** Dr. Prashant Mehta, Faridabad	Panelists: Dr. A.M.V.R. Narendra, Hyderabad Dr. Deep Gala, Mumbai Dr. Hamza Dalal, Mumbai Dr. Thejeswar N Prakasham, Hyderabad Dr. Vinay Bohara, Indore
Session 9		
Chair: Dr. Hamza Dalal, Dr. Kunal Goyal, Dr. Deep Gala		
3.00 pm - 3.30 pm	Molecular Subtyping to Immune Redirection: Integrating LymphGen, POLARIX, Bispecifics, and CAR-T in High-Risk DLBCL	Dr. Mallikarjun Kalashetty, Bengaluru
3.30 pm - 4.00 pm	NHL Updated: What's New, What's Next, and What's Unusual ?	Dr. Gaurav Prakash, Chandigarh

Scientific Agenda

Saturday, 5th September 2026, Hall B (Room 206 B)

Anchor: Dr. Bipin Kulkarni

Time	Subject	Speaker
Session 10		
4.00 pm - 4.30 pm	Panel discussion Sub: Maintenance therapy in AML Moderator: Dr. Gaurav Dixit, Delhi	Panelists: Dr. Avinash Kumar Singh, Patna Dr. Kunal Goyal, Mumbai Dr. Prakash Singh Shekhawat, Jaipur Dr. Shilpa Bhartia, Kolkata Dr. Vinod Patil, Satara
4.30 pm - 5.00 pm	**High Tea**	
Session 11		
Chair: Dr. Gaurav Dixit, Dr. Avinash Kumar Singh, Dr. Shilpa Bhartia		
5.00 pm - 5.30 pm	Treatment free remission in CML: the next frontier	Dr. Dragana Milojkovic, UK
5.30 pm - 6.00 pm	Daratumumab is the game changer: from induction to maintenance	Dr. Rajshekhar Chakraborty, USA
6.00 pm - 6.30 pm	Multiple myeloma: I do not want to kill my patient by infection	Dr. Rajshekhar Chakraborty, USA
Session 12		
Sponsor: **Astellas** Chair: Dr. Girish Rajadhyaksha, Dr. Javid Rasool Bhat, Dr. Priyanka Moule		
6.30 pm - 7.00 pm	Sponsored session by Astellas Panel discussion Sub: Case based panel discussion on Xospata monotherapy in R/R FLT3+ AML patients Moderator: Dr. Punit Jain	Panelists: Dr. Asif Iqbal, Guwahati Dr. Matteo Piccini, Italy
Session 13		
Sponsor: **Unipath** Chair: Dr. Girish Rajadhyaksha, Dr. Javid Rasool Bhat, Dr. Priyanka Moule		
7.00 pm - 7.15 pm	Liquid biopsy in lymphoma	Dr. Neeraj Arora, Ahmedabad
7.15 pm - 7.30 pm	Comprehensive Genomic Profiling	Dr. Neeraj Arora, Ahmedabad

Scientific Agenda

Saturday, 5th September 2026, Hall C (Room 203)

Anchor: Dr. Rucha Patil

Time	Subject	Speaker
Session 1		
9.00 am - 9.30 am	Panel discussion **Sub:** Hemophagocytic lymphohistiocytosis **Moderator:** Dr. Sujata Sharma, Mumbai	Panelists: Dr. Aditi Shah Kaskar, Mumbai Dr. Archana Swami, Mumbai Dr. Purva Kanvinde, Mumbai Dr. Shubhangi Agale, Mumbai
Session 2		
Chair: Dr. Vijai Tilak, Dr. Rucha Patil, Dr. Ritika Agrawal		
9.30 am - 10.00 am	Fifth edition WHO classification: strategies for application in resource-limited settings	Dr. Prashant Tembhare, Mumbai
10.00 am - 10.30 am	The Immune Interface in Hemophilia: Biology to Bedside	Dr. Rashmi Kushwaha, Lucknow
10.30 am - 11.00 am	**Coffee**	
Session 3		
Chair: Dr. Javid Rasool Bhat, Dr. Shilpi Saxena, Dr. Dipsha Kriplani		
11.00 am - 11.30 am	Clinical Utility of New Hematology Parameters	Dr. Ritika Agrawal, Bikaner
11.30 am - 12.00 noon	Common pitfalls in NGS interpretation in benign hematology	Dr. Prashant Sharma, Chandigarh
Session 4		
Chair: Dr. Varsha Vadera, Dr. Nishant Jindal, Dr. Shivani Joshi		
12.00 noon - 12.30 pm	Molecular genetic landscape in MPN	Dr. Vijai Tilak, Varanasi
12.30 pm - 1.00 pm	Spectrum of patients with elevation of gamma delta T cells in routine clinical practice	Dr. Shilpi Saxena, Mumbai
1.00 pm - 2.00 pm	**Lunch**	
Session 5		
2.00 pm - 2.30 pm	Panel discussion **Sub:** Stem Cell Enumeration: From Flow Cytometry to Engraftment - Science, Standardization and Clinical Reality **Moderator:** Dr. Varsha Vadera, Mumbai	Panelists: Dr. Kiran A. Ghodke, Mumbai Dr. Nishant Jindal, Mumbai Dr. Ruhi Mehra, Mumbai
Session 6		
Chair: Dr. Seema Bhatwadekar, Dr. Prakash Singh Shekhawat, Dr. Sayan Ghoshal		
2.30 pm - 3.00 pm	From Morphology to Molecules: Integrating Morphology, Flow Cytometry, and Genomics in Hematology	Dr. Kiran A. Ghodke, Mumbai
3.00 pm - 3.30 pm	Rheumatology & Hematology: the interface	Dr. Farah Jijina, Mumbai

Scientific Agenda

Saturday, 5th September 2026, Hall C (Room 203)

Anchor: Dr. Rucha Patil

Time	Subject	Speaker
Session 7		
Sponsor: **Sun Pharma**		
3.30 pm - 4.00 pm	Panel discussion **Sub:** Curing/Eliminating Thalassaemia in India: A realistic approach **Moderator:** Dr. Rajib De, Kolkata	Panelists: Dr. Anita Nadkarni, Mumbai Dr. Mamta Manglani, Mumbai Dr. Prakash Singh Shekhawat, Jaipur Dr. Shivani Joshi, Nashik
Session 8		
4.00 pm - 4.30 pm	Panel discussion **Sub:** Transforming the Sickle Cell Landscape in India **Moderator:** Dr. R. K. Jena, Cuttack	Panelists: Dr. Anita Nadkarni, Mumbai Dr. Ritika Khurana, Mumbai Dr. Seema Bhatwadekar, Vadodara Dr. Shivani Joshi, Nashik Dr. Soniya Nagyal, Hedri, Gadchiroli Dr. Vinay Bohara, Indore
4.30 pm - 5.00 pm	**High Tea**	
Session 9		
5.00 pm - 5.30 pm	Panel discussion **Sub:** Paraproteinemic neuropathies **Moderator:** Dr. Shilpa Gupta, Mumbai	Panelists: Dr. A.M.V.R. Narendra, Hyderabad Dr. Ashish Gupta, Jabalpur Dr. Lingaraj Nayak, Mumbai Dr. Uday Yanamandra, Pune Dr. Varun Bafna, Kolhapur
5.30 pm - 6.00 pm	Panel discussion **Sub:** Plasma cell dyscrasia with renal involvement **Moderator:** Dr. Seema Bhatwadekar, Vadodara	Panelists: Dr. Ashish Gupta, Jabalpur Dr. S. P. Shrivastava, Indore Dr. Kunal Goyal, Mumbai Dr. Neha Garg, Mumbai Dr. Shruti Toshniwal, Aurangabad
6.00 pm - 6.30 pm	Panel discussion **Sub:** Defining cure in myeloma **Moderator:** Dr. Uday Yanamandra, Pune	Panelists: Dr. Ashish Gupta, Jabalpur Dr. Lingaraj Nayak, Mumbai Dr. Sanjeev, Lucknow Dr. Shruti Toshniwal, Aurangabad Dr. Thejeswar N Prakasham, Hyderabad
Session 10		
Chair: Dr. Farah Jijina, Dr. S. P. Shrivastava, Dr. Thejeswar N. Prakasham		
6.30 pm - 7.00 pm	Waldenström Macroglobulinemia	Dr. Sayan Ghoshal, Howrah
7.00 pm - 7.30 pm	Immunotherapy in Pediatric Oncology in India: A Nationwide Survey Assessing Practices, Perceptions, and Barriers Among Pediatric Oncologists	Dr. Nirmalya Roy Moulik, Mumbai

Saturday, 5th September 2026, Hall D (Room 202)

Anchor: Dr. Jay Sheth, Mr. Kamleshwar Dubey

Time	Subject	Speaker
Session 1		
Chair: Dr. Pavan Kumar Boyella, Dr. Naveen Gupta, Dr. Anusree Prabhakaran		
9.00 am - 9.30 am	How can biology inform therapy in DLBCL ?	Dr. Daniel J. Hodson, UK
9.30 am - 10.00 am	Balancing the choice of TKI therapy in CML: a new era	Dr. Dragana Milojkovic, UK
Session 2		
Chair: Dr. Gaurav Prakash, Dr. Sanjeev, Dr. Shilpa Gupta		
10.00 am - 10.30 am	Case discussion session **Case presenter:** Dr. Ashish Dixit, Bengaluru	Discussant: Dr. Nicola Gökbuget, Germany
10.30 am - 11.00 am	**Coffee**	
Session 3		
Sponsor: **Glenmark**		
11.00 am - 11.30 am	Session Sponsored by Glenmark Panel discussion **Sub:** Smarter BTK Targeting: The Zanubrutinib Era in B-Cell Lymphomas **Moderator:** Dr. M. B. Agarwal, Mumbai	Panelists: Dr. Ashish Dixit, Bengaluru Dr. Bhausaheb Bagal, Mumbai Dr. Mallikarjun Kalashetty, Bengaluru Dr. Prashant Mehta, Faridabad Dr. Sanjeevan Sharma, Delhi Dr. Shilpa Bhartia, Kolkata
Session 4		
Sponsor: **Natco**		
Chair: Dr. Darshana Rane, Dr. Varun Bafna, Dr. Ashita Singhal		
11.30 am - 12.00 noon	Treatment of T-ALL	Dr. Nicola Gökbuget, Germany
12.00 noon - 12.30 pm	Sex-specific roles of DDX3 RNA helices in aggressive B cell lymphomas	Dr. Daniel J. Hodson, UK
12.30 pm - 1.00 pm	Pediatric Thrombosis: Guidelines and Clinical Practice Updates	Dr. Cindy Neunert, USA
1.00 pm - 2.00 pm	**Lunch**	

Saturday, 5th September 2026, Hall D (Room 202)

Anchor: Dr. Jay Sheth, Mr. Kamleshwar Dubey

Time	Subject	Speaker
Session 5		
Sponsor: **Pfizer**		
Theme : Optimizing outcomes with BCMA specific antibodies in relapsed/refractory multiple myeloma		
2.00 pm - 2.05 pm	Welcome & opening remarks	Dr. M. B. Agarwal, Mumbai
2.05 pm - 2.30 pm	Elranatamab in R/R Multiple Myeloma : current evidence and practical implications	Dr. Rajshekhar Chakraborty, USA
2.30 pm - 2.45 pm	**Case-based discussion:** Management of a triple-class exposed / penta-refractory RRMM patient treated with BCMA - directed therapy	Dr. M. B. Agarwal, Mumbai
2.45 pm - 3.15 pm	Panel discussion **Sub:** Best practices in therapy management of bispecifics antibodies in RRMM: adverse events management, infection prevention, supportive care and patient monitoring **Moderator:** Dr. M. B. Agarwal, Mumbai	Panelists Dr. Harshit Khurana, Delhi Dr. Mallikarjun Kalashetty, Bengaluru Dr. Rajshekhar Chakraborty, USA Dr. Upendra Sharma, Jaipur
3.15 pm - 3.25 pm	Q&A	All faculty members
3.25 pm - 3.30 pm	Future of management of MM in India - Key take-home messages	Dr. Rajshekhar Chakraborty, USA Dr. M. B. Agarwal, Mumbai
Session 6		
Chair: Dr. Bharat Agarwal, Dr. Archana Swami		
3.30 pm - 4.00 pm	Hemophilia Guidelines: Application in the real world	Dr. Cindy Neunert, USA
4.00 pm - 4.30 pm	Real-world data in Pediatric ITP	Dr. Cindy Neunert, USA
4.30 pm - 5.00 pm	**High Tea**	
Session 7		
5.00 pm - 6.00 pm	One Target, Multiple Possibilities: BCMA-Directed Therapies in Multiple Myeloma with a Spotlight on Antibody-Drug Conjugates	Dr. Saad Usmani, USA
Session 8		
Sponsor: **GSK**		
6.00 pm - 6.30 pm	Session sponsored by GSK Eye related side effects with novel hematologic anti-cancer drugs: Predict, Prevent and Preside	Dr. Simona Esposti, UK
6.30 pm - 7.30 pm	Session sponsored by GSK Panel discussion **Sub:** Optimizing patient outcomes in second line multiple myeloma treatment landscape **Moderator:** Dr. Rayaz Ahmed, Delhi	Panelists Dr. Abhay Bhave, Mumbai Dr. Mallikarjun Kalashetty, Bengaluru Dr. Prashant Mehta, Faridabad Dr. Rohan Haldar, Delhi Dr. Simona Esposti, UK Dr. Smita Gavade, Mumbai

Scientific Agenda

Saturday, 5th September 2026, Hall E (Room 207)

Anchor: Mr. Vivek Thorat / Mr. MSJ

Time	Subject	
Session 1		
Judges : Dr. Rajan Kapoor, Dr. Neha Kamble, Dr. Ambreen Pandrowala		
9.00 am - 10.30 am	Discussion on poster presentation	
10.30 am - 11.00 am	**Coffee**	
Session 2		
11.00 am - 11.30 am	Session sponsored by Servier Panel discussion Sub: Precise 1 for mIDH1: Unlocking the Precision with TIBSOVO Moderator: Dr. Subhaprakash Sanyal, Mumbai	Panelists Dr. Jina Bhattacharya, Guwahati Dr. Kundan Mishra, Lucknow Dr. Prakash Singh Shekhawat, Jaipur Dr. Sanjeev, Lucknow
11.30 am - 12.00 noon	Sponsored session by GSK (Vaccine division) Integrating Shingles Prevention into Hemato-Oncology Care	Dr. Seema Bhatwadekar, Vadodara
Session 3		
12.00 noon - 1.00 pm	The role of Asciminib in Early-Line CML (Newly Diagnosed and 1L Failure CML) Moderator: Dr. M. B. Agarwal, Mumbai	Participants TBD
1.00 pm - 2.00 pm	**Lunch**	
Session 4		
Sponsor: **Micro CRISPR**		
2.00 pm - 4.30 pm	Workshop by MICRO CRISPR Sub: CAR-T patient selection workshop Moderator: TBD	Participants Dr. A.M.V.R. Narendra, Hyderabad Dr. Bhausaheb Bagal, Mumbai Dr. Gaurav Dixit, Delhi Dr. Kunal Goyal, Mumbai Dr. Lingaraj Nayak, Mumbai Dr. Mallikarjun Kalashetty, Bengaluru Dr. Manju Sengar, Mumbai Dr. Nishant Jindal, Mumbai Dr. Prakash Singh Shekhawat, Jaipur Dr. Prashant Mehta, Faridabad Dr. Rajib De, Kolkata Dr. Reetu Jain, Mumbai Dr. S. P. Shrivastava, Indore Dr. Sharat Damodar, Bengaluru Dr. Subhaprakash Sanyal, Mumbai Dr. Uday Yanamandra, Pune Dr. Vinay Bohara, Indore Dr. Vipul Sheth, Delhi
4.30 pm - 5.00 pm	**High Tea**	
5.00 pm - 5.30 pm	Workshop by MICRO CRISPR (Cont..)	
Session 5		
5.30 pm - 6.30 pm	Close group meeting On Bispecific: New Era	Dr. Karthik Ramasamy, UK
Session 6		
Sponsor: **Bayer**		
6.30 pm - 7.00 pm	Navigating Cancer-Associated Thrombosis: Evidence and Clinical Considerations with Rivaroxaban	Dr. Hamza Dalal, Mumbai

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Scientific Agenda

Sunday, 6th September 2026

Scientific Agenda

Sunday, 6th September 2026, Hall A (Room 206 A)

Anchor: Dr. Amit Khurana

Time	Subject	Speaker
Session 1		
Sponsor: **Bharat Serums & Vaccines**		
9.00 am - 9.30 am	Panel discussion Sub: Non-transplant treatment of aplastic anemia Moderator: Dr. Sameer Tulpule, Mumbai	Panelists: Dr. Kundan Mishra, Lucknow Dr. Rajesh Patil, Mumbai Dr. Shailendra Prasad Verma, Lucknow Dr. Subhaprakash Sanyal, Mumbai Dr. Vrinda Kulkarni, Mumbai
Session 2		
Sponsor: **Natco**		
Chair: Dr. Prashant Mehta, Dr. Kundan Mishra, Dr. Nikhil Kumar M		
9.30 am - 10.00 am	Tackling NRM of Haplo identical transplants in the Indian Scenario	Dr. Harshit Khurana, Delhi
10.00 am - 10.30 am	Where does BMT stand in the era of CARs and BITEs ?	Dr. Reetu Jain, Mumbai
10.30 am - 11.00 am	**Coffee**	
Session 3		
Sponsor: **Intas**		
Chair: Dr. R. K. Jena, Dr. Reetu Jain, Dr. Sanket Shah		
11.00 am - 11.30 am	Breaking Free: Calcineurin-Free GVHD Prophylaxis & Steroid-Sparing First-Line Therapy EHA 2026 Updates	Dr. Sundar Shewale, Kolkata
11.30 am - 12.00 noon	Monoclonal Gammopathy of Renal Significance (MGRS): Current Concepts and Evolving Therapeutic Strategies	Dr. Dinesh Bhurani, Delhi
Session 4		
Sponsor: **Adley**		
Chair: Dr. Asha Shah, Dr. Sanket Shah, Dr. Shrinath Kshirsagar		
12.00 noon - 12.30 pm	Case discussion session Case presenter: Dr. Kundan Mishra, Lucknow	Dr. Dragana Milojkovic, UK
12.30 pm - 1.00 pm	Teclistamab: indications - today's and tomorrow's	Dr. Rajshekhar Chakraborty, USA

Scientific Agenda

Sunday, 6th September 2026, Hall A (Room 206 A)

Anchor: Dr. Amit Khurana

Time	Subject	Speaker
1.00 pm - 2.00 pm	**Lunch**	
Session 5		
Sponsor: **Natco**		
Chair: Dr. Shubhangi Agale, Dr. Sameer Tulpule, Dr. Shailendra Prasad Verma		
2.00 pm - 2.30 pm	Integrated global scientific advances in hematology	Dr. Sanket Shah, Ahmedabad
2.30 pm - 3.00 pm	Case discussion session Case presenter: Dr. Nikhil Kumar, Kochi	Dr. Daniel J. Hodson, UK
Session 6		
Sponsor: **Lucien**		
Chair: Dr. Jina Bhatacharyya, Dr. Namita Padwal, Dr. Riya Ballikar		
3.00 pm - 3.30 pm	Case discussion session Case presenter: Dr. VA Arun, Mumbai	Dr. Cindy Neunert, USA
3.30 pm - 4.00 pm	Case discussion session Case presenter: Dr. Shailendra Prasad Verma, Lucknow	Dr. Rajshekhar Chakraborty, USA
Session 7		
4.00 pm - 4.30 pm	**Valedictory function & prize distribution**	
4.30 pm - 5.00 pm	**High Tea**	

Scientific Agenda

Sunday, 6th September 2026, Hall B (Room 206 B)

Anchor: Dr. Bipin Kulkarni

Time	Subject	Speaker
Session 1		
Sponsor: **Adley**		
Chair: Dr. Rajesh Sawant, Dr. Samir Shah, Dr. Ruhi Mehra		
9.00 am - 9.30 am	Complement Inhibition: Beyond PNH to Kidney and Rare Disease	Dr. Shrinath Kshirsagar, Mumbai
9.30 am - 10.00 am	Management of Acute immune mediated TTP	Dr. Asha Shah, Mumbai
10.00 am - 10.30 am	Endotheliopathies in HSCT - TMA and SOS	Dr. Sachin Punatar, Mumbai
10.30 am - 11.00 am	**Coffee**	
Session 2		
Sponsor: **Adley**		
Chair: Dr. Jina Bhattacharyya, Dr. Mallikarjun Kalashetty, Dr. Nikhil Kumar		
11.00 am - 11.30 am	New advances in thrombosis	Dr. Abhay Bhave, Mumbai
11.30 am - 12.00 noon	The History of Hemophilia Through the Ages: From Ancient Observations to Modern Therapies	Dr. Lalit Prashant Meena, Varanasi
Session 3		
Sponsor: **Adley**		
Chair: Dr. Abhay Bhave, Dr. Riya Ballikar, Dr. Kunal Goyal		
12.00 noon - 12.30 pm	Challenging Scenarios in Therapeutic anticoagulation - Case based approach	Dr. Nikhil Kumar M, Kochi
12.30 pm - 1.00 pm	Evidence Based Transfusion Practices	Dr. Rajesh Sawant, Mumbai
1.00 pm - 2.00 pm	**Lunch**	
Session 4		
Sponsor: **Lucien**		
Chair: Dr. Neeraj Sidharthan, Dr. Lalit Prashant Meena, Dr. Avinash Kumar Singh		
2.00 pm - 2.30 pm	Myeloproliferative Neoplasms: Indian scenario	Dr Jina Bhattacharyya, Guwahati
2.30 pm - 3.00 pm	The changing face of MDS: from transfusions to targeted therapy	Dr. Riya Ballikar, Nagpur
Session 5		
Sponsor: **Dr. Reddy's**		
Chair: Dr. Rajesh Sawant, Dr. Ganesh Jaishetwar, Dr. Gaurav Dixit		
3.00 pm - 3.30 pm	CAR-T: toxicity mitigation	Dr. Neeraj Sidharthan, Kochi
3.30 pm - 4.00 pm	Next-Generation engineering: From Autologous to universal and in vivo CAR-T	Dr. Kunal Goyal, Mumbai
Session 6		
4.00 pm - 4.30 pm	**Valedictory function & prize distribution in Hall A (Room 206 A)**	
4.30 pm - 5.00 pm	**High Tea**	

Scientific Agenda

Sunday, 6th September 2026, Hall C (Room 203)

Anchor: Dr. Rucha Patil

Time	Subject	Speaker
Session 1		
Sponsor: **Natco**		
Chair: Dr. Tapan Saikia, Dr. Dinesh Bhurani, Dr. Balkrishna Padate		
9.00 am - 9.30 am	The End of 7+3: Venetoclax & the new era of precision in treatment of AML	Dr Ganesh Jaishetwar, Hyderabad
9.30 am - 10.00 am	MRD directed therapy in adult AML: are we there yet ?	Dr. Sudha Sinha, Hyderabad
10.00 am - 10.30 am	Induction in acute leukemia: survival determined by pathogens and not cytogenetics	Dr. Asif Iqbal, Guwahati
10.30 am - 11.00 am	**Coffee**	
Session 2		
Sponsor: **Sun Pharma**		
Chair: Dr. Kanchanmala Ghorpade, Dr. Sudha Sinha, Dr. Pankaj Tantia, Dr. Neha Garg		
11.00 am - 11.30 am	How do we sequence therapy in CLL in 2026? from BTKi to BCL2i and beyond	Dr. Upendra Sharma, Jaipur
11.30 am - 12.00 noon	BTKi and BCL2i: The New Frontline in Mantle Cell Lymphoma (MCL)	Dr. Pankaj Tantia, Bikaner
Session 3		
Sponsor: **Natco**		
Chair: Dr. Samir Shah, Dr. Ganesh Jaishetwar, Dr. Upendra Sharma		
12.00 noon - 12.30 pm	Where does ABVD fit in for advanced stage Hodgkin Lymphoma in 2026?	Dr. Tapan Saikia, Mumbai
12.30 pm - 1.00 pm	T-Cell Lymphomas: How do we treat in 2026?	Dr. Balkrishna Padate, Mumbai
1.00 pm - 2.00 pm	**Lunch**	
Session 4		
Sponsor: **Natco**		
Chair: Dr. Priti Mehta, Dr. Avitri Baveja, Dr Ankita Pandey		
2.00 pm - 2.30 pm	Precision in Focus: Modern Radiotherapy Paradigms in Lymphoma	Dr. Ankit Batra, Dehradun
2.30 pm - 3.00 pm	Primary and secondary CNS lymphomas	Dr. Jyoti Chirmade, Mumbai
Session 5		
Sponsor: **Natco**		
Chair: Dr. Ankit Batra, Dr. Amit Jain, Dr. Jyoti Chirmade		
3.00 pm - 3.30 pm	Short Telomeres, Big Impact: Decoding the Latest in TBD Biology"	Dr. Avriti Baveja, Dehradun
3.30 pm - 4.00 pm	How to tackle DSA positivity prior to transplant ?	Dr. Priti Mehta, Mumbai
Session 6		
4.00 pm - 4.30 pm	**Valedictory function & prize distribution in Hall A (Room 206 A)**	
4.30 pm - 5.00 pm	**High Tea**	

Scientific Agenda

Sunday, 6th September 2026, Hall E (Room 207)

Anchor: Dr. Jay Sheth, Mr. Kamleshwar Dubey

Time	Subject	Speaker
10.30 am - 11.00 am	**Coffee**	
Session 1		
11.00 am - 12.30 pm	Close group meeting	J&J Representative
1.00 pm - 2.00 pm	**Lunch**	
Session 2		
4.00 pm - 4.30 pm	**Valedictory function & Prize distribution in Hall A (Room 206 A)**	
4.30 pm - 5.00 pm	**High Tea**	

Mid Term Conference of

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Program at a glance

Friday, 4th September 2026

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Friday, 4th September 2026

Time	Hall A (206 A)	Hall B (206 B)	Hall C (203)	Hall D (202)	Hall E (Room 207)
9.00 am - 9.30 am	**Panel discussion** **Sub:** Optimizing outcomes of HSCT: where have we reached in India ? **Moderator:** Dr. Biju George, Vellore **Panelists:** Dr. Aditi Shah Kaskar Dr. Navin Khattry Dr. Sameer Tulpule Dr. Sanjeevan Sharma	**Panel discussion** **Sub:** Refractory ITP **Moderators:** Dr. Mamta Manglani **Panelists:** Dr. Amit Jain Dr. Anjan Shrestha Dr. Ashita Singhal Dr. Jai Juvekar Dr. Parth Ganatra Dr. Purva Kanvinde Dr. Shweta Bansal	Beyond Pyruvate Kinase Deficiency: Spectrum of Rare Red Cell Enzymopathies in Unexplained Hemolytic Anaemia - An NIIH Experience Dr. Prabhakar Kedar	8.45 am - 9.00 am **Quiz for UG** Dr. Shilpi Saxena 9.10 am - 9.25 am **Quiz for PG** Dr. Kunal Goyal	
9.30 am - 10.00 am	Practice changes following ASH-25/EHA-26 in LMICs: My thoughts! Dr. Ranjit Kumar Sahoo	TPO mimetics beyond Pediatric ITP: Shifting Paradigms Dr. Purvi Kadakia Kutty	Is Intracellular Calcium Dysregulation a Common Molecular Mechanism in Hereditary Hemolytic Anemias ? Implications for Novel Therapeutic Targets Dr. Prashant Warang	9.35 am - 9.50 am **Quiz for Superspeciality** Dr. VA Arun	9.30 am - 10.30 am **Oral paper presentation session** **(O-1 to O-6**
10.00 am - 10.30 am	The Molecular Underworld: Uncovering unusual Drivers in Myeloid Malignancies Dr. Neha Kamble	Neonatal Fc receptor: Biology & Therapeutics Dr. Rajan Kapoor	Understanding Immunothrombosis Dr. Jasmina Ahluwalia	10.00 am - 10.15 am **Quiz for Pediatric Hematology Oncology** Dr. Priti Mehta 10.15 am - 10.30 am **Quiz for Hematopathology** Dr. Vidisha Mahajan	
10.30 am - 11.15 am	**Coffee**	**Coffee**	**Coffee**	**Coffee**	
11.15 am - 11.45 am	CAR-T cell therapy in B-ALL: our experience with a focus on those who received it as a consolidation Dr. Hasmukh Jain	Combined Session in **Hall A & B (Room 206 A & B)**	**Panel discussion** **Sub:** The Community Hematopathologist: Bridging the Gap Between Microscope and Bedside **Moderators:** Dr. Akshata Mandloi **Panelists:** Dr. Archana Vazifdar Dr. Dipsha Kriplani Dr. Shivani Joshi Dr. Shrutika Java Dr. Vidisha Mahajan	**Sponsored session by Sun Pharma** **Panel discussion** **Sub:** Venetoclax based panel discussion **Moderator:** Dr. Sameer Tulpule **Panelists:** Dr. Balkrishna Padate Dr. Bhausaheb Bagal Dr. Digambar Panchal Dr. Navin Khattry Dr. Rajan Kapoor Dr. Rajesh Patil	11.15 am - 12.15 pm **Oral paper presentation session** **O-7 to O-12**
11.45 am - 12.15 pm	Beyond the Tail of the Curve: Engineering the Next Curative Frontier in NHL Dr. Manju Sengar	Combined Session in **Hall A & B (Room 206 A & B)**	**Panel discussion** **Sub:** Targeted therapy in B ALL **Moderators:** Dr. Kunal Sehgal **Panelists:** Dr. Ambreen Pandrowala Dr. Gaurav Narula Dr. Prashant Tembhare Dr. Subhaprakash Sanyal	**Panel discussion** **Sub:** Case based discussion on CLL **Moderator:** Dr. VA Arun **Panelists:** Dr. Alok Shetty Dr. Anant Gokarn Dr. Ashok Meshram Dr. Jai Juvekar Dr. Uday Yanamandra	
12.15 pm - 1.15 pm	**Inauguration**	**Inauguration in Hall A & B (Room 206 A & B)**	**Inauguration in Hall A & B (Room 206 A & B)**	**Inauguration in Hall A & B (Room 206 A & B)**	**Inauguration in Hall A & B (Room 206 A & B)**

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Time	**Hall A** (206 A)	**Hall B** (206 B)	**Hall C** (203)	**Hall D** (202)	**Hall E** (Room 207)
1.15 pm - 2.00 pm	**Lunch**	**Lunch**	**Lunch**	**Lunch**	**Lunch**
2.00 pm - 2.30 pm	When a low platelet count is not ITP ! Dr. Cindy Neunert	From Paediatric Oncologist to Infection Preventionist: My Journey as the Infection Control Officer at a Tertiary Cancer Centre Dr. Arpita Bhattacharyya	The Harmonization of Biomarker Reports of Monoclonal Gammopathies Dr. Barnali Das	Acute Lymphoblastic Leukemia in the Era of NGS: An Indian Clinician's Perspective Dr. Tribikram Panda	2.00 pm - 4.30 pm **Oral paper presentation session** **O-13 to O-27**
2.30 pm - 3.00 pm	2.30 pm - 3.30 pm Dual targets, new possibilities: BCMA & GPRC5D in relapsed refractory multiple myeloma Dr. Karthik Ramasamy, UK	Childhood ALL: A decade of ICiCle & Immunotherapies- Lessons learnt & the Way Forward Dr. Gaurav Narula	Genomic work up in Relapsed Acute Lymphoblastic Leukaemia Dr. Mayur Parihar	Veno-occlusive disease: both in transplant and Chemo-Immunotherapy settings Dr. Sohini Chattopadhyay	
3.00 pm - 3.30 pm	Dr. M. B. Agarwal, Mumbai	Pattern of Asparaginase toxicities in Pediatric ALL Dr. Chetan Dhamne	Review of Guidelines and evidence for Diagnostic work up of Acute leukemia in India Dr. P. G. Subramanian	Transplants in tier three cities Dr. Venkatesh Ekbote	
3.30 pm - 4.00 pm	Treatment of B-Precursor Ph-ve ALL Dr. Nicola Gökbuget	Pharmacogenomics: day to day application Dr. Swati Kanakia	Rare but Relevant: Diagnostic Lessons from Challenging Malignant Hematopathology Cases Dr. Akshaya Mandloi	3.30 pm - 4.20 pm **Astellas sponsored session** Insights from Clinical Practice: Xospata Monotherapy in R/R FLT3+ AML Dr. Matteo Piccini	
4.00 pm - 4.30 pm	High risk smoldering myeloma: how do I define and how do I treat ? Dr. Rajshekhar Chakraborty	Survivorship in Pediatric Cancers Dr. Suhani Barbhuiyan	Slide seminars in Hematopathology: A Potpourri of exciting cases Dr. Tathagata Chatterjee		
4.30 pm - 5.00 pm	**High Tea**	**High Tea**	**High Tea**	**High Tea**	**High Tea**
5.00 pm - 5.30 pm	Secondary ITP: What do we know ? Dr. Cindy Neunert	Stem Cell Transplant in Metabolic Disorders Dr. Santanu Sen	Role of flow cytometry in platelet function disorders Dr. Vidisha Mahajan	Hairy cell leukemia Dr. Ramaswamy NV	5.00 pm - 7.30 pm **Oral paper presentation session** **O-28 to O-38**
5.30 pm - 6.00 pm	Is circulating tumour DNA set to revolutionalize how we manage DLBCL ? Dr. Daniel J. Hodson	Deficiency of ADA2: The Hematology spectrum Dr. Ambreen Pandrowala	Laboratory challenges in haemostasis: Case- based solutions Dr. Bipin Kulkarni	Infections and immune dysfunction with bispecifics and CAR-T in Multiple Myeloma Dr. Aditya Jandial	
6.00 pm - 6.30 pm	Management of the side effects of TKI therapies Dr. Dragana Milojkovic	Sip by Sip, Stronger Sickle Cells: The Power of Liquid Hydroxyurea Dr. Ritika Khurana	DOACs vs Traditional Anticoagulants: Current Guidelines & Controversies Dr. J. M. Khunger	Use of granulocyte transfusions in transplant Dr. Anu Korula	
6.30 pm - 7.00 pm	Treatment of B-precursor Ph+ve ALL Dr. Nicola Gökbuget	Pediatric Hodgkin Lymphoma: Salvage therapy in R/R disease Dr. Reghu K. Sukumaran	Need of chromogenic assay in the current era for haemophilia Dr. Rucha Patil	Infections following BTK inhibitors Dr. Mahesh Rajashekaraiah	
7.00 pm - 7.30 pm	AL Amyloidosis: Prognostic and predictive biomarkers Dr. Rajshekhar Chakraborty	Juvenile Myelomonocytic Leukemia Dr. Ankita Pandey	The most common yet missed bleeding disorder: Diagnosing Von Willibrand Disease Dr. Anju Bharti	**Sponsored session by Cipla** TBD	

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Program at a glance

Saturday, 5th September 2026

Saturday, 5th September 2026

Time	**Hall A** (206 A)	**Hall B** (206 B)	**Hall C** (203)	**Hall D** (202)	**Hall E** (Room 207)
9.00 am - 9.30 am	**Panel discussion** **Sub:** Myelofibrosis - how do I manage ? **Moderator:** Dr. Rajesh Patil **Panelists:** Dr. A.M.V.R. Narendra Dr. Ashok K. Yadav Dr. Kalyani Patil Dr. Shruti Toshniwal Dr. Suman Mittal Dr. Varun Bafna	Use of Anti-CD19 CAR-T in Primary CNS Lymphoma: **A case-based discussion** Dr. Naveen Vairamoorthy	**Panel discussion** **Sub:** Hemophagocytic lymphohistiocytosis **Moderator:** Dr. Sujata Sharma **Panelists:** Dr. Aditi Shah Kaskar Dr. Archana Swami Dr. Purva Kanvinde Dr. Shubhangi Agale	How can biology inform therapy in DLBCL ? Dr. Daniel J. Hodson	9.00 am - 10.30 am **Discussion on poster presentation** **Judges:** Dr. Rajan Kapoor Dr. Neha Kamble Dr. Ambreen Pandrowala
9.30 am - 10.00 am	Hydroxyurea Dosing in Indian Patients with Sickle Cell Disease: Results of a Six-Center Randomised Controlled Trial Dr. Naveen Khargekar	**Panel discussion** **Sub:** Therapeutic implications of biology of DLBCL **Moderator:** Dr. Jay Mehta **Panelists:** Dr. Gaurav Prakash Dr. Manju Sengar Dr. Sanjeev Dr. Subhaprakash Sanyal Dr. Tathagata Chatterjee	Fifth edition WHO classification: strategies for application in resource-limited settings Dr. Prashant Tembhare	Balancing the choice of TKI therapy in CML: a new era Dr. Dragana Milojkovic	
10.00 am - 10.30 am	**Session supported by MICRO CRISPR** Transforming R/R B-cell Acute Lymphoblastic Leukemia treatment landscape: The evolving role of CAR-T therapy in clinical practice Dr. Vipul Sheth	**Session supported by Novo Nordisk** Innovation in Action: Enhancing Patient Autonomy and Haemostatic Control with Concizumab **Chair:** Dr. Mamta Manglani **Lecture:** Enabling Patient Autonomy and Normalcy with an Innovative Device Dr. Amit Khurana **Lecture:** Normalizing Thrombin Generation with Steady-State Control and Precision Dosing Dr. Dnyaneshwar Upase	The Immune Interface in Hemophilia: Biology to Bedside Dr. Rashmi Kushwaha	**Case discussion session** **Case presenter:** Dr. Ashish Dixit **Discussant:** Dr. Nicola Gökbuget	
10.30 am - 11.00 am	**Coffee**	**Coffee**	**Coffee**	**Coffee**	**Coffee**

Saturday, 5th September 2026

Time	**Hall A** (206 A)	**Hall B** (206 B)	**Hall C** (203)	**Hall D** (202)	**Hall E** (Room 207)
11.00 am - 11.30 am	11.00 am - 11.40 am **Sponsored session by Novartis** **Panel discussion** **Sub:** Choosing the Optimal Firstline treatment in CML **Moderator:** Dr. R K Jena, Cuttack **Panelists** Dr. Ashok Meshram Dr. Damodar Das Dr. Varun Bafna	11.00 am - 11.50 am **Sponsored session by Sanofi** 11.00 am - 11.20 am A new era of Hemophilia management-Rebalancing agents Dr. Varun Bafna 11.20 am - 11.35 am Antithrombin Testing TBD 11.35 am - 11.50 am **Panel discussion** **Sub:** Unmet need and potential role of rebalancing agents in Haemophilia care **Moderator:** Dr. Varun Bafna **Panelists:** Dr. Jai Juvekar Dr. Nehal Patel Dr. Rucha Patil	Clinical Utility of New Hematology Parameters Dr. Ritika Agrawal	**Session sponsored by Glenmark** **Panel discussion** **Sub:** Smarter BTK Targeting: The Zanubrutinib Era in B-Cell Lymphomas **Moderator:** Dr. M. B. Agarwal **Panelists** Dr. Ashish Dixit Dr. Bhausaheb Bagal Dr. Mallikarjun Kalashetty Dr. Prashant Mehta Dr. Sanjeevan Sharma Dr. Shilpa Bhartia	**Session sponsored by Servier** **Panel discussion** **Sub:** Precise 1 for mIDH1: Unlocking the Precision with TIBSOVO **Moderator:** Dr. Subhaprakash Sanyal **Panelists** Dr. Jina Bhattacharya Dr. Kundan Mishra Dr. Prakash Singh Shekhawat Dr. Sanjeev
11.30 am - 12.00 noon	11.40 am - 12.00 noon **Sponsored session by BMS** From Transfusion Dependence to independence in Frontline MDS: COMMANDing Results with Rojuzda Dr. Mahadeva Swamy BC		Common pitfalls in NGS interpretation in benign hematology Dr. Prashant Sharma	Treatment of T-ALL Dr. Nicola Gökbuget	**Sponsored session by GSK (Vaccine division)** Integrating Shingles Prevention into Hemato-Oncology Care Dr. Seema Bhatwadekar
12.00 noon - 12.30 pm	Light chain Amyloidosis-diagnosis and treatment Dr. Naveen Gupta	**Panel discussion** **Sub:** Framework for Selecting Better Therapies for Hemophilia A **Moderator:** Dr. Varun Bafna **Panelists:** Dr. Dnyaneshwar Upase Dr. Jai Juvekar Dr. Madhura Deshpande	Molecular genetic landscape in MPN Dr. Vijai Tilak	Sex-specific roles of DDX3 RNA helices in aggressive B cell lymphomas Dr. Daniel J. Hodson	12.00 noon - 1.00 pm **Sponsored session by Novartis** The role of Asciminib in Early-Line CML (Newly Diagnosed and 1L Failure CML) **Moderator:** Dr. M. B. Agarwal, Mumbai **Participants** TBD
12.30 pm - 1.00 pm	Is low-dose nivolumab adequate for Hodgkin Lymphoma ? Dr. Anusree Prabhakaran	**Session supported by Abbvie** Management of IC-Ineligible AML: Real-World Evidence and Clinical Insights Dr. Subhaprakash Sanyal	Spectrum of patients with elevation of gamma delta T cells in routine clinical practice Dr. Shilpi Saxena	Pediatric Thrombosis: Guidelines and Clinical Practice Updates Dr. Cindy Neunert	
1.00 pm - 2.00 pm	**Lunch**	**Lunch**	**Lunch**	**Lunch**	**Lunch**

Saturday, 5th September 2026

Time	Hall A (206 A)	Hall B (206 B)	Hall C (203)	Hall D (202)	Hall E (Room 207)
2.00 pm - 2.30 pm	2.000 pm - 3.00 pm **Session supported by Roche** **Panel discussion** **Sub:** Navigating DLBCL in 2026: a case-based symposium **Moderator:** Dr. Bhausaheb Bagal **Panelists:** Dr. Gaurav Dixit Dr. Pavan Kumar Boyella Dr. Sanjeev Dr. Seema Bhatwadekar Dr. Shouriyo Ghosh Dr. Subhaprakash Sanyal	**Session supported by Abbvie** **Panel discussion** **Sub:** All oral fixed duration therapy for ND CLL **Moderator:** Dr. Sharat Damodar **Panelists:** Dr. A.M.V.R. Narendra Dr. Lingaraj Nayak Dr. Sanjeev Dr. Shilpa Bhartia Dr. Varun Bafna Dr. Vinod Patil	**Panel discussion** **Sub:** Stem Cell Enumeration: From Flow Cytometry to Engraftment - Science, Standardization and Clinical Reality **Moderator:** Dr. Varsha Vadera **Panelists:** Dr. Kiran A. Ghodke Dr. Nishant Jindal Dr. Ruhi Mehra	2.00 pm - 3.30 pm **Session sponsored by Pfizer** Optimizing outcomes with BCMA specific antibodies in R/R MM 2.00 pm - 2.05 pm **Welcome & opening remarks** Dr. M. B. Agarwal 2.05 pm - 2.30 pm Elranatamab in R/R MM : current evidence and practical implications Dr. Rajshekhar Chakraborty 2.30 pm - 2.45 pm **Case-based discussion:** Management of a triple-class exposed / penta-refractory RRMM patient treated with BCMA - directed therapy Dr. M. B. Agarwal 2.45 pm - 3.15 pm **Panel discussion** **Sub:** Best practices in therapy management of bispecifics antibodies in RRMM: adverse events management, infection prevention, supportive care & patient monitoring **Moderator:** Dr. M. B. Agarwal, Mumbai **Panelists** Dr. Harshit Khurana Dr. Mallikarjun Kalashetty Dr. Rajshekhar Chakraborty Dr. Upendra Sharma 3.15 pm - 3.25 pm **Q&A** All faculty members 3.25 pm - 3.30 pm Future of management of MM in India : Key take-home messages Dr. Rajshekhar Chakraborty Dr. M. B. Agarwal	2.00 pm - 5.30 pm **Workshop by MICRO CRISPR** **Sub:** CAR-T patient selection workshop **Moderator:** TBD
2.30 pm - 3.00 pm		**Panel discussion** **Sub:** Lower toxicity approaches for B-ALL **Moderator:** Dr. Prashant Mehta **Panelists:** Dr. A.M.V.R. Narendra Dr. Deep Gala Dr. Hamza Dalal Dr. Thejeswar N Prakasham Dr. Vinay Bohara	From Morphology to Molecules: Integrating Morphology, Flow Cytometry, and Genomics in Hematology Dr. Kiran A. Ghodke		
3.00 pm - 3.30 pm	**Session supported by Takeda** BrECADD decoded: practical insights for frontline HL management Dr. Mark Hertzberg	Molecular Subtyping to Immune Redirection: Integrating LymphGen, POLARIX, Bispecifics, and CAR-T in High-Risk DLBCL Dr. Mallikarjun Kalashetty	Rheumatology & Hematology: the interface Dr. Farah Jijina		

Saturday, 5th September 2026

Time	Hall A (206 A)	Hall B (206 B)	Hall C (203)	Hall D (202)	Hall E (Room 207)
3.30 pm - 4.00 pm	**Session supported by Takeda** **Panel discussion** **Sub:** Adcetris expanded choice in frontline Hodgkin's Lymphoma **Moderator:** Dr. M. B. Agarwal, Mumbai **Panelists** Dr. Abhay Bhave Dr. Alok Shetty Dr. Ashish Dixit Dr. Kunal Goyal Dr. Mark Hertzberg Dr. Sameer Tulpule Dr. Shouriyo Ghosh	NHL Updated: What's New, What's Next, and What's Unusual ? Dr. Gaurav Prakash	**Panel discussion** **Sub**: Curing/Eliminating Thalassaemia in India: A realistic approach **Moderator:** Dr. Rajib De **Panelist** Dr. Anita Nadkarni Dr. Mamta Manglani Dr. Prakash Singh Shekhawat Dr. Shivani Joshi	Hemophilia Guidelines: Application in the real world Dr. Cindy Neunert	**Workshop by MICRO CRISPR (Cont..)**
4.00 pm - 4.30 pm	MRD in Myeloma Dr. Karthik Ramasamy Dr. Prashant Tembhare	**Panel discussion** **Sub**: Maintenance therapy in AML **Moderator:** Dr. Gaurav Dixit **Panelist** Dr. Avinash Kumar Singh Dr. Kunal Goyal Dr. Prakash Singh Shekhawat Dr. Shilpa Bhartia Dr. Vinod Patil	**Panel discussion** **Sub**: Transforming the Sickle Cell Landscape in India **Moderator:** Dr. R. K. Jena **Panelist** Dr. Anita Nadkarni Dr. Ritika Khurana Dr. Seema Bhatwadekar Dr. Shivani Joshi Dr. Soniya Nagyal Dr. Vinay Bohara	Real-world data in Pediatric ITP Dr. Cindy Neunert	
4.30 pm 5.00 pm	**High Tea**	**High Tea**	**High Tea**	**High Tea**	**High Tea**
5.00 pm - 5.30 pm	Recent Advances in management of Castleman's Disease Dr. Amit Khurana	Treatment free remission in CML: the next frontier Dr. Dragana Milojkovic	**Panel discussion** **Sub**: Paraproteinemic neuropathies **Moderator:** Dr. Shilpa Gupta **Panelist** Dr. A.M.V.R. Narendra Dr. Ashish Gupta Dr. Lingaraj Nayak Dr. Uday Yanamandra Dr. Varun Bafna	5.00 pm - 6.00 pm One Target, Multiple Possibilities: BCMA-Directed Therapies in Multiple Myeloma with a Spotlight on Antibody-Drug Conjugates Dr. Saad Usmani, USA	**Workshop by MICRO CRISPR (Cont..)**
5.30 pm - 6.00 pm	POEMS Syndrome: Unique presentation of this rare paraneoplastic syndrome and its management Dr. Priyanka Moule	Daratumumab is the game changer: from induction to maintenance Dr. Rajshekhar Chakraborty	**Panel discussion** **Sub:** Plasma cell dyscrasia with renal involvement **Moderator:** Dr. Seema Bhatwadekar **Panelists:** Dr. Ashish Gupta Dr. Kunal Goyal Dr. Neha Garg Dr. S. P. Shrivastava Dr. Shruti Toshniwal		5.30 pm - 6.30 pm **Close group meeting** On Bispecific: New Era Dr. Karthik Ramasamy

Saturday, 5th September 2026

Time	Hall A (206 A)	Hall B (206 B)	Hall C (203)	Hall D (202)	Hall E (Room 207)
6.00 pm - 6.30 pm	**Session supported by AstraZeneca** Eculizumab is the way to go for treating Paroxysmal Nocturnal Hemoglobinuria Dr. Ashok Meshram	Multiple myeloma: I do not want to kill my patient by infection Dr. Rajshekhar Chakraborty	**Panel discussion** **Sub:** Defining cure in myeloma **Moderator:** Dr. Uday Yanamandra **Panelists:** Dr. Ashish Gupta Dr. Lingaraj Nayak Dr. Sanjeev Dr. Shruti Toshniwal Dr. Thejeswar N Prakasham	**Sponsored session by GSK** Eye related side effects with novel hematologic anti-cancer drugs: Predict, Prevent and Preside Dr. Simona Esposti	
6.30 pm - 7.00 pm	6.30 pm - 7.30 pm Talquetamab - the new horizon Dr. Karthik Ramasamy Dr. M. B. Agarwal	**Sponsored session by Astellas** **Panel discussion** **Sub:** Case based panel discussion on Xospata monotherapy in R/R FLT3+ AML patients **Moderator:** Dr. Punit Jain **Panelists:** Dr. Asif Iqbal Dr. Matteo Piccini	Waldenström Macroglobulinemia Dr. Sayan Ghoshal, Howrah	**Sponsored session by GSK** **Panel discussion** **Sub:** Optimizing patient outcomes in second line multiple myeloma treatment landscape **Moderator:** Dr. Rayaz Ahmed **Panelists** Dr. Abhay Bhave Dr. Mallikarjun Kalashetty Dr. Prashant Mehta Dr. Rohan Haldar Dr. Simona Esposti Dr. Smita Gavade	**Sponsored session by Bayer** Navigating Cancer-Associated Thrombosis: Evidence and Clinical Considerations with Rivaroxaban Dr. Hamza Dalal
7.00 pm - 7.30 pm		**Session supported by Unipath** 7.00 pm - 7.15 pm Liquid biopsy in lymphoma Dr. Neeraj Arora 7.15 pm - 7.30 pm Comprehensive Genomic Profiling Dr. Neeraj Arora	Immunotherapy in Pediatric Oncology in India: A Nationwide Survey Assessing Practices, Perceptions, and Barriers Among Pediatric Oncologists Dr. Nirmalya Roy Moulik	**Sponsored session by GSK**	

Mid Term Conference of

Mumbai Hematology Group (MHG) & Essence of EHA-2026

Program at a glance

Sunday, 6th September 2026

Mid Term Conference of
Mumbai Hematology Group (MHG) & Essence of EHA-2026

Sunday, 6th September 2026

Time	**Hall A** (206 A)	**Hall B** (206 B)	**Hall C** (203)	**Hall E** (Room 207)
9.00 am - 9.30 am	**Sponsored by BSV** **Panel discussion** **Sub:** Non-transplant treatment of aplastic anemia **Moderator:** Dr. Sameer Tulpule **Panelists:** Dr. Kundan Mishra Dr. Rajesh Patil Dr. Shailendra Prasad Verma Dr. Subhaprakash Sanyal Dr. Vrinda Kulkarni	Complement Inhibition: Beyond PNH to Kidney and Rare Disease Dr. Shrinath Kshirsagar	The End of 7+3: Venetoclax & the new era of precision in treatment of AML Dr. Ganesh Jaishetwar	
9.30 am - 10.00 am	Tackling NRM of Haplo identical transplants in the Indian Scenario Dr. Harshit Khurana	Management of Acute immune mediated TTP Dr. Asha Shah	MRD directed therapy in adult AML: are we there yet ? Dr. Sudha Sinha	
10.00 am - 10.30 am	Where does BMT stand in the era of CARs and BITEs ? Dr. Reetu Jain	Endotheliopathies in HSCT - TMA and SOS Dr. Sachin Punatar	Induction in acute leukemia: survival determined by pathogens and not cytogenetics Dr. Asif Iqbal	
10.30 am - 11.00 am	**Coffee**	**Coffee**	**Coffee**	**Coffee**
11.00 am - 11.30 am	Breaking Free: Calcineurin-Free GVHD Prophylaxis & Steroid-Sparing First-Line Therapy EHA 2026 Updates Dr. Sundar Shewale	New advances in thrombosis Dr Abhay Bhave	How do we sequence therapy in CLL in 2026? from BTKi to BCL2i and beyond Dr. Upendra Sharma	11.00 am - 12.30 pm **Close group meeting** J&J Representative
11.30 am - 12.00 noon	Monoclonal Gammopathy of Renal Significance (MGRS): Current Concepts and Evolving Therapeutic Strategies Dr. Dinesh Bhurani	The History of Hemophilia Through the Ages: From Ancient Observations to Modern Therapies Dr. Lalit Prashant Meena	BTK and BCL2: The New Frontline in Mantle Cell Lymphoma (MCL) Dr. Pankaj Tantia	
12.00 noon - 12.30 pm	**Case discussion session** **Case presenter:** Dr. Kundan Mishra **Discussant:** Dr. Dragana Milojkovic	Challenging Scenarios in Therapeutic anticoagulation - Case based approach Dr. Nikhil Kumar M	Where does ABVD fit in for advanced stage Hodgkin Lymphoma in 2026? Dr. Tapan Saikia	
12.30 pm - 1.00 pm	Teclistamab: indications - today's and tomorrow's Dr. Rajshekhar Chakraborty	Evidence Based Transfusion Practices Dr. Rajesh Sawant	T-Cell Lymphomas: How do we treat in 2026? Dr. Balkrishna Padate	
1.00 pm - 2.00 pm	**Lunch**	**Lunch**	**Lunch**	**Lunch**

Sunday, 6th September 2026

Time	**Hall A** (206 A)	**Hall B** (206 B)	**Hall C** (203)	**Hall E** (Room 207)
2.00 pm - 2.30 pm	Integrated global scientific advances in hematology Dr. Sanket Shah	Myeloproliferative Neoplasms: Indian scenario Dr. Jina Bhattacharyya	Precision in Focus: Modern Radiotherapy Paradigms in Lymphoma Dr. Ankit Batra	
2.30 pm - 3.00 pm	**Case discussion session** **Case presenter:** Dr. Nikhil Kumar **Discussant:** Dr. Daniel J. Hodson	The changing face of MDS: from transfusions to targeted therapy Dr. Riya Ballikar	Primary and secondary CNS lymphomas Dr. Jyoti Chirmade	
3.00 pm - 3.30 pm	**Case discussion session** **Case presenter:** Dr. VA Arun **Discussant:** Dr. Cindy Neunert	CAR-T: toxicity mitigation Dr. Neeraj Sidharthan	Short Telomeres, Big Impact: Decoding the Latest in TBD Biology" Dr. Avriti Baveja	
3.30 pm - 4.00 pm	**Case discussion session** **Case presenter:** Dr. Shailendra Prasad Verma **Discussant:** Dr. Rajshekhar Chakraborty	Next-Generation engineering: From Autologous to universal and in vivo CAR-T Dr. Kunal Goyal	How to tackle DSA positivity prior to transplant ? Dr. Priti Mehta	
4.00 pm - 4.30 pm	**Valedictory function & prize distribution**	**Valedictory function & prize distribution in Hall A (Room No. 206 A)**	**Valedictory function & prize distribution in Hall A (Room No. 206 A)**	**Valedictory function & prize distribution in Hall A (Room No. 206 A)**
4.30 pm - 5.00 pm	**High Tea**	**High Tea**	**High Tea**	**High Tea**

Annexe 1

Abstracts

Dr. H. M. Bhatia & Dr. L. D. Sanghvi oral paper presentation session

Oral paper presentation session

Friday, 4th September 2026, Hall E (Room 207), 9.30 am to 7.30 pm

Judges: Dr. Sujata Sharma, Dr. Hamza Dalal, Dr. Shilpi Saxena

O-1

Ibrutinib-Induced Cardiotoxicity in B-Cell Lymphoma: An In Silico Electrophysiological Analysis Revealing Nav1.5 Sodium Current Suppression and Sinoatrial Node Dysfunction

Presenting Author: Dr. Chitaranjan Mahapatra[1]

Co-Authors: [2]Dr. Arshdeep Kaur, University of California San Francisco, USA

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O-2

Real-World Clinical Profile, Treatment Patterns and Outcomes of Waldenström Macroglobulinemia: A Single-Centre Experience from North India

Presenting Author: Dr. Nikhil Nagpal

Co-Authors: Dr Manoj Saini , Dr Saiprasath J, Dr Kavya Ronanki, Dr Bibhant Shah, Dr Eswer Subbaiyan, Dr Anshika Sinha, Dr. Uttam Kumar Nath

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O-3

Frontline ICE therapy for Hepatosplenic T-cell lymphoma: a real-world single center experience from India

Presenting Author: Dr. Ayush Agarwal

Co-Authors: Dr. Abhinav Sengupta[1], Dr. Shubham Goswami[1], Dr. Jasmita Dass[2], Dr. Rishi Dhawan[2], Dr. Mukul Aggarwal[2], Dr. Tulika Seth[3], Dr. Manoranjan Mahapatra[3]

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Oral paper presentation session

Friday, 4th September 2026, Hall E (Room 207), 9.30 am to 7.30 pm

Judges: Dr. Sujata Sharma, Dr. Hamza Dalal, Dr. Shilpi Saxena

O-4

Clinical Outcomes in Very Elderly (Age ≥80 years) Patients with Non-Hodgkin's Lymphoma: Experience from Tertiary care centre

Presenting Author: Dr. Nimmya SK

Co-Authors: Dr Nikhil Krishna Haridas, Dr Sourabh Radhakrishnan, Dr Rakesh MP, Dr Anjali, Dr Wesley Jose

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O-5

Outcomes of Adult and Adolescent Acute Lymphoblastic Leukemia Treated with Paediatric inspired protocol: A Single-Center Retrospective Analysis of Toxicity, MRD Response, and SurvivalCentre

Presenting Author: Dr. Devireddy Raj Mohan Reddy

Co-Authors: Anant Gokarn, Sachin Punatar, Sumeet Mirgh, Yamuna Naik, Akanksha Chichra, Lingaraj Nayak, Nishant Jindal, Avinash Bonda, Bhausaheb Bagal and Navin Khattry.

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O-6

Molecular landscape and treatment outcomes of acute myeloid leukemia: A 2-year single-center experience from a tertiary care hospital in South India

Presenting Author: Dr. Aakash Rajendra Gupta

Co-Authors: Dr. Neeraj Siddharthan[1], Dr. Manoj Unni[1], Dr. Nikhil Haridas[1], Dr. Rema[1], Dr. Suchitra[2], Dr. Thomas Kuncheria[2]

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Oral paper presentation session

Friday, 4th September 2026, Hall E (Room 207), 9.30 am to 7.30 pm

Judges: Dr. Sujata Sharma, Dr. Hamza Dalal, Dr. Shilpi Saxena

O-7

The Pre-engraftment Window:
Bloodstream Infection following Allogeneic Haematopoietic Stem Cell Transplantation

Presenting Author: Dr. Goh Jun Yan

Co-Authors: Dr. Hany Ariffin

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O-8

Standardization of Skin Fibroblast Culture for Germline Testing in Acute Myeloid Leukemia

Presenting Author: Swastik Sastri

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O-9

Age-Stratified Molecular Profiling and ELN 2022 Risk Distribution in Acute Myeloid Leukemia Patients Younger Than 60 Years: A Single-Center Retrospective Analysis

Presenting Author: Neerukonda Sriteja

Co-Authors: Ashish Dixit, Mallikarjun Kalashetty

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Oral paper presentation session

Friday, 4th September 2026, Hall E (Room 207), 9.30 am to 7.30 pm

Judges: Dr. Sujata Sharma, Dr. Hamza Dalal, Dr. Shilpi Saxena

O-10

Clinical Outcomes of Hypomethylating Agents in Acute Myeloid Leukemia: A Single-Centre Retrospective Study from South India

Presenting Author: Moiz Bashir Doctor

Co-Authors: Mallikarjun Kalashetty, Ashish Dixit, Santhosh Subramanian, Apeksha Kanakiya, Sriteja Neerukonda

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O-11

Podoplanin Expression Improves the Diagnostic Accuracy of Acute Promyelocytic Leukemia

Presenting Author: Ananya Ghosh

Co-Authors: Akash Maity, Syed K. Hasan

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O-12

Life Beyond TKI: Real-World Experience of Treatment-Free Remission in Chronic Myeloid Leukemia from North India

Presenting Author: Dr. Raj Kumar Maurya

Co-Authors: Dr. S. P. Verma[2], Dr. Swasti Sinha[3], Dr. P. Raghuveer[3], Dr. Aritra Saha[1], Dr. Middinti Akshay[1], Dr. Khushboo Kumari[1], Dr. Manish Kumar[1]

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Oral paper presentation session

Friday, 4th September 2026, Hall E (Room 207), 9.30 am to 7.30 pm

Judges: Dr. Sujata Sharma, Dr. Hamza Dalal, Dr. Shilpi Saxena

O-13

Clinical profile and response to TKI of paediatric chronic myeloid leukemia: an experience from a tertiary care hospital from the Himalayan foothills, India

Presenting Author: Dr. Manoj Saini

Co-Authors: Dr Nagpal Nikhil, Dr Janarthan Saiprasath , Dr Ronanki Kavya, Dr Shah Bibhant, Dr Subbaiyan Eswer, Dr Sinha Anshika, Prof Nath Uttam Kumar

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O-14

The utility of NGS-based chimerism analysis for the interpretation of STR-PCR results in patients after a second allogeneic hematopoietic cell transplantation

Presenting Author: Ewa Karakulska-Prystupiuk

Co-Authors: Marcelina Grabowska, Martyna Brzoza, Mateusz Brzozowski, Agnieszka Tomaszewska, Grzegorz Władysław Basak

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O-15

Outcomes of donor lymphocyte infusion for pre-emptive or therapeutic use after allogeneic HSCT; a single-center experience

Presenting Author: Ewa Karakulska-Prystupiuk

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Oral paper presentation session

Friday, 4th September 2026, Hall E (Room 207), 9.30 am to 7.30 pm

Judges: Dr. Sujata Sharma, Dr. Hamza Dalal, Dr. Shilpi Saxena

O-16

Treosulfan-based conditioning is associated with better outcomes in adolescents and young adults undergoing matched related donor transplantation for Thalassemia major

Presenting Author: Mithun Abraham Prakash

Co-Authors: Uday Kulkarni, Sujith K, Sushil Selvarajan, Sharon Lionel, Anu Korula, Fouzia NA, Kavitha M Lakshmi, Eunice Sindhuvi, Aby Abraham, Vikram Mathews, Biju George

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O-17

**From Vision to Reality:
Establishing a Pediatric Bone Marrow Transplant Center in a Government Hospital in Central India**

Presenting Author: Dr. Shweta Pathak

Co-Authors: Dr Meena Singh[1], Dr Monica Lazarus[2], Dr Vidya Kumari[3]

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O-18

**Real-World Experience with CAR-T Cell Therapy in Lymphoma:
Insights from a Tertiary Care Centre in South India**

Presenting Author: Dr. Mohit Rajora

Co-Authors: N. Siddharthan[1], Manoj Unni[1], Rema[1], Nikhil K Haridas[2], Radhakrishnan S[3], Rakesh Mp[3]

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Oral paper presentation session

Friday, 4th September 2026, Hall E (Room 207), 9.30 am to 7.30 pm

Judges: Dr. Sujata Sharma, Dr. Hamza Dalal, Dr. Shilpi Saxena

O-19

CAR-T cell therapy- a single center experience

Presenting Author: Dr. Ganga R

Co-Authors: Dr. Paul Sonal, Dr Bhogaria Akash, Mr Das Mihir, Mrs Hemam Sarla Devi, Mrs Keisam Sanjita, Dr Chakrabartty Joydeep

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O-20

Spectrum of plasma cell dyscrasias: clinicopathological study in a tertiary care hospital

Presenting Author: Dr. Jay Y. Sheth

Co-Author: Dr. M. B. Agarwal

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O-21

Frailty in Newly Diagnosed Multiple Myeloma: Prevalence, Determinants and Prognostic Impact in a Real-World Indian Cohort Using the Simplified IMWG Frailty Score.

Presenting Author: Ms. Khadija Saifee

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Oral paper presentation session

Friday, 4th September 2026, Hall E (Room 207), 9.30 am to 7.30 pm

Judges: Dr. Sujata Sharma, Dr. Hamza Dalal, Dr. Shilpi Saxena

O-22

Attrition rates in multiple myeloma: a report from Indian patients at a Tertiary Cancer Centre

Presenting Author: Dr. Dhyey R. Mishra

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O-23

Comparison of differential count in leukopenic samples by the regular and low WBC mode on an automated hematology analyzer with the manual differential count

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O-24

Dry Tap Dilemma

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Oral paper presentation session

Friday, 4th September 2026, Hall E (Room 207), 9.30 am to 7.30 pm

Judges: Dr. Sujata Sharma, Dr. Hamza Dalal, Dr. Shilpi Saxena

O-25

Repeat 3+7 versus FLAG re-induction in acute myeloid leukaemia with residual disease after standard 3+7 induction: a real-world single-centre analysis

Presenting Author: Dr. Shubham Goswami

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O-26

The Cartwheel Chronicles

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O-27

Marrow Beyond Malignancy: : A Window into Systemic Disease

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O-28

Oral paper presentation session

Friday, 4th September 2026, Hall E (Room 207), 9.30 am to 7.30 pm

Judges: Dr. Sujata Sharma, Dr. Hamza Dalal, Dr. Shilpi Saxena

O-29

Co-inheritance of α-Globin Gene Multiplication in β-Globin Disorders: A Single-Centre Molecular Diagnostic Experience

Presenting Author: Dr. Manasvi J. Shah

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O-30

Clinical Profile, Prognostic Stratification and Treatment Outcomes of Patients with Chronic Lymphocytic Leukemia: A Single-Center Real-World Experience

Presenting Author: Dr. Bhageerath Kumar

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O-31

Innovative bridge in Ph positivity: Efficacy of combination therapy with Inotuzumab ozogamicin and Ponatinib in Ph positive and Ph-like acute lymphoblastic leukemia in children

Presenting Author: Dr. Kavitha Ganesan

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O-32

Clinical, Laboratory Profile and Early Outcomes of Pediatric Hyperleukocytosis in Acute Leukemia: Retrospective comparative analytic Study from Central India

Presenting Author: Dr. Sunil Jondhale

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Oral paper presentation session

Friday, 4th September 2026, Hall E (Room 207), 9.30 am to 7.30 pm

Judges: Dr. Sujata Sharma, Dr. Hamza Dalal, Dr. Shilpi Saxena

O-33

Caught in the Overlap: Immune Mediated Thrombotic Thrombocytopenic Purpura in Systemic Lupus Erythematosus–Sjögren's Syndrome

Presenting Author: Dr. Amruta B Bagul

Co-Authors: Amol Dube[1], Ishan Verma[2], Archit Pandharipande[3], Naila Khan[3], Vinay Rautiya[4], Sanjana Nalmas[5]

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O-34

Timing matters: Predictors of outcome in severe aplastic anemia

Presenting Author: Dr. Asmita K. Gawde

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O-35

Emergency Splenectomy in Immune Thrombocytopenia Presenting with Intracranial Hemorrhage

Presenting Author: Mithun Abraham Prakash

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Oral paper presentation session

Friday, 4th September 2026, Hall E (Room 207), 9.30 am to 7.30 pm

Judges: Dr. Sujata Sharma, Dr. Hamza Dalal, Dr. Shilpi Saxena

O-36

Thalidomide in pediatric thalassemia with hypersplenism and transfusion burden: a single center experience

Presenting Author: Dr. Ritamoni C. Baruah

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O-37

Clinico-etiological and management profile of Acquired Hemophilia A: A single centre experience from India

Presenting Author: Dr. Priyamvadha Ramesh[1]

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O-38

Clinical, microbiological profile and outcomes of febrile neutropenia in acute leukemia patients undergoing induction therapy in a tertiary care centre from North India

Presenting Author: Dr. Vaishali Sharai

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Annexe 2

Abstracts

Dr. A. J. Desai & Dr. L. H. Hiranandani poster paper presentations

Poster presentation session

Saturday, 5th September 2026, Hall E (Room 207), 9.00 am - 10.30 am

Judges: Dr. Rajan Kapoor, Dr. Ambreen Pandrowala, Dr. Neha Kamble

P-1

HLH-Spectrum Hyperinflammation During Induction Therapy for Acute Monocytic Leukemia: A Stereotyped Syndrome of Persistent Fever, Conjugated Hyperbilirubinemia, Platelet Transfusion Refractoriness and Dramatic Corticosteroid Responsiveness

Presenting Author: Dr Aniruddha P. Burli

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P-2

A phase II study to evaluate the role of Pomalidomide in maintaining treatment-free remission post TKI discontinuation in patients with chronic myeloid leukemia-chronic phase

Presenting Author: Dr. Bikash Sourav[1]

Co-Authors: Manju Sengar[1], Alok Shetty[1], Lingaraj Nayak[1], Bhausaheb Bagal[1], Thomas Eipe[1], Prashant Tembhare[1], Avinash Bonda[1], Neha Sharma[1], Santhosh Devadas[2], Dhanlaxmi Shetty[1], P.G.Subramanian[1], Anant Gokarn[1], Sachin Punatar[1], Navin Khattry[1], Smruti Mokal[1], Hasmukh Jain[1]*

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P-3

A Storm in the Blood: Unravelling the Cause of Hyperleukocytosis

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Poster presentation session

Saturday, 5th September 2026, Hall E (Room 207), 9.00 am - 10.30 am

Judges: Dr. Rajan Kapoor, Dr. Ambreen Pandrowala, Dr. Neha Kamble

P-4

Daratumumab Based Quadruplet Therapy in Newly Diagnosed Multiple Myeloma: Outcomes from a Tertiary Care Centre in India

Presenting Author: Mr. Aabhran Singh

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P-5

Acute leukemia during pregnancy: a single-center retrospective cohort study of maternal & fetal outcomes in India

Presenting Author: Dr. Ayush Agarwal

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P-6

Short term outcomes of Flu/Cy/Mel + TBI Conditioning in Haplo-Identical Hematopoietic Stem Cell Transplantation

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Poster presentation session

Saturday, 5th September 2026, Hall E (Room 207), 9.00 am - 10.30 am

Judges: Dr. Rajan Kapoor, Dr. Ambreen Pandrowala, Dr. Neha Kamble

P-7

Multimodal Salvage Therapy for Severe Graft-versus-Host Disease: A Single-Centre Experience

Presenting Author: Dr. Vishwa Makadia

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P-8

The vitamin that made the difference: A case of Thiamine-responsive megaloblastic anemia

Presenting Author: Dr. Ahmad Fikri Ahmad Zafrullah Afham

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P-9

Etiology of Pyrexia of Unknown Origin (PUO) with pancytopenia hidden in bone marrow

Presenting Author: Dr. Aniket Sonawane

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Poster presentation session

Saturday, 5th September 2026, Hall E (Room 207), 9.00 am - 10.30 am

Judges: Dr. Rajan Kapoor, Dr. Ambreen Pandrowala, Dr. Neha Kamble

P-10

Beyond the ovary: an uncommon presentation of paediatrics diffuse large B-cell lymphoma

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P-11

Thymoma-Associated PRCA Revealing a KIF23-Related CDA

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P-12

Unmasking genetic complexity in Beta thalassaemia trait

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Poster presentation session

Saturday, 5th September 2026, Hall E (Room 207), 9.00 am - 10.30 am

Judges: Dr. Rajan Kapoor, Dr. Ambreen Pandrowala, Dr. Neha Kamble

P-13

Dual Autoimmune Crisis: Active SLE with concurrent severe Aplastic Anaemia successfully treated with Horse Anti-Thymocyte Globulin

Presenting Author: Dr. Udaya Kumari Challa

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P-14

When the Counts Recover but the Virus Does Not: EBV-Driven Secondary Hemophagocytic Lymphohistiocytosis Evolving to Chronic Active EBV Disease in an Immunocompetent Young Adult

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P-15

**Individualizing the Venetoclax Window:
Balancing Toxicity and Response in Acute Myeloid Leukemia: A Single-Centre Real-World Experience**

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Poster presentation session

Saturday, 5th September 2026, Hall E (Room 207), 9.00 am - 10.30 am

Judges: Dr. Rajan Kapoor, Dr. Ambreen Pandrowala, Dr. Neha Kamble

P-16

Correlative diagnosis of multiple myeloma using bone marrow morphology serum protein electrophoresis and CRAB criteria: A case report

Presenting Author: Dr. Arunima Bhati

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P-17

HLH-Macrophage Activation Syndrome in Juvenile Rheumatoid Syndrome

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P-18

Extramedullary plasmacytoma - a rare case

Presenting Author: Dr. Dhvani Thakker

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Poster presentation session

Saturday, 5th September 2026, Hall E (Room 207), 9.00 am - 10.30 am

Judges: Dr. Rajan Kapoor, Dr. Ambreen Pandrowala, Dr. Neha Kamble

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**When itching precedes the diagnosis:
classical Hodgkin Lymphoma presenting with prurigo nodularis**

Presenting Author: Dr. Prianuz Dewraja

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P-20

When MDS Hides a Second Clone IgM - κ Monoclonal Gammopathy with Cold Agglutinin Disease: a Diagnostic Challenge

Presenting Author: Neerukonda Sriteja

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P-21

Unmasking a Rare Telomeropathy: Homozygous CTC1 Mutation Presenting as Transfusion: Dependent Pancytopenia and Multisystem Disease in a Young Adult: A case of Coats Plus Syndrome

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Poster presentation session

Saturday, 5th September 2026, Hall E (Room 207), 9.00 am - 10.30 am

Judges: Dr. Rajan Kapoor, Dr. Ambreen Pandrowala, Dr. Neha Kamble

P-22

Determining stability of complete blood count samples stored in the Hematology laboratory at recommended temperature

Presenting Author: Dr. Uma Ramanathan

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Oral Papers

O-1

Ibrutinib-Induced Cardiotoxicity in B-Cell Lymphoma: An In Silico Electrophysiological Analysis Revealing Nav1.5 Sodium Current Suppression and Sinoatrial Node Dysfunction

Presenting Author: Dr. Chitaranjan Mahapatra[1]

Co-Authors: [2]Dr. Arshdeep Kaur, University of California San Francisco, USA

Institution: 1. SRM Institute of Science and Technology Delhi NCR Campus, Ghaziabad, India.
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Background:

Ibrutinib, an irreversible Bruton's tyrosine kinase (BTK) inhibitor, has revolutionized the therapeutic landscape for B-cell lymphoma. Despite its established clinical efficacy, emerging reports of cardiotoxicity—including arrhythmias, atrial fibrillation, and sudden cardiac death—have raised significant safety concerns. The precise electrophysiological mechanisms underlying Ibrutinib-induced cardiac dysfunction remain incompletely characterized, particularly regarding voltage-gated sodium channel (Nav1.5) modulation in the sinoatrial node (SAN), the primary cardiac pacemaker. Understanding these mechanisms is critical for optimizing dosing strategies and mitigating life-threatening adverse events in haematology patients receiving long-term Ibrutinib therapy.

Objective:

To elucidate the concentration-dependent effects of Ibrutinib on cardiac electrophysiological properties, with specific emphasis on Nav1.5-mediated sodium current modulation and its consequent impact on SAN pacemaker activity.

Method:

An in silico electrophysiological model of the human sinoatrial node was employed to simulate acute Ibrutinib exposure across clinically relevant concentrations, ranging from 0.1 mol/L to 10 mol/L, over a 200 ms simulation window. The conductance of the voltage-gated sodium channel Nav1.5 was systematically altered to reflect documented drug-channel interactions. Both current-clamp and voltage-clamp protocols were applied to record electrophysiological parameters, including action potential (AP) firing characteristics and current-voltage (I-V) relationships under control and drug-exposed conditions.

Results:

Current-clamp simulations demonstrated robust action potential generation across all stimulus intensities (0.1-0.10 nA, 10-50 ms). Voltage-clamp analysis revealed a striking, concentration-dependent suppression of inward sodium currents. At the highest tested concentration of 10 mol/L, peak inward sodium current was reduced to merely 26% of baseline control values. This was accompanied by a 20% rightward shift in the I-V curve and a 28% increase in the half-activation potential, indicating altered channel gating kinetics. At elevated concentrations, action potentials exhibited marked morphological alterations including broadened waveforms, significantly slowed upstroke velocity (dV/dt max), and prolonged repolarization phases. Critically, the overall SAN firing frequency was substantially diminished, demonstrating impaired intrinsic pacemaker activity and predisposition to bradyarrhythmia.

Conclusion:

This study provides the first in-silico evidence that higher concentrations of Ibrutinib exert potent suppressive effects on Nav1.5-mediated sodium currents, leading to reduced sinoatrial node pacemaker activity, slowed conduction, and prolonged repolarization. These findings offer a mechanistic basis for the clinical cardiotoxicity observed with Ibrutinib therapy and underscore the urgent need for rigorous dose optimization, therapeutic drug monitoring, and prospective cardiac surveillance in B-cell lymphoma patients. Further translational studies integrating in-silico predictions with clinical electrophysiology are warranted to establish evidence-based

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cardioprotective protocols.

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O-2

Real-World Clinical Profile, Treatment Patterns and Outcomes of Waldenström Macroglobulinaemia: A Single-Centre Experience from North India

Presenting Author: Nikhil Nagpal

Co-Authors: Manoj Saini, Saiprasath Janarthan, Kavya Ronanki, Bibhant Shah, Prisla Dalton, Eswer Subbaiyan, Anshika Sinha, Arjun Kachhwaha, Reshma Benson, Adamya Gupta, Uttam Kumar Nath

Institution: Department of Medical Oncology Haematology, All India Institute of Medical Sciences, Rishikesh, Uttarakhand, India

Background:

Waldenström Macroglobulinemia (WM) is a rare indolent lymphoplasmacytic lymphoma characterized by bone marrow infiltration and monoclonal IgM paraproteinaemia. Despite advances in therapy, real-world data from India remain limited. We evaluated the clinical profile, treatment patterns, response, and outcomes of patients with WM treated at a tertiary care centre in North India.

Objective:

To describe the clinical characteristics, treatment patterns, therapeutic responses, and outcomes of patients with Waldenström Macroglobulinemia.

Method:

This retrospective observational study included consecutive patients diagnosed with WM between 2014 and 2026. Demographic, clinical, laboratory, treatment, response, relapse, second-line therapy, and survival data were retrieved from electronic medical records. Treatment responses were assessed according to the International Workshop on Waldenström's Macroglobulinemia (IWWM) response criteria.

Results:

Sixteen patients were included. The median age at diagnosis was 61.5 years (range 36–77 years), and 75% were male. Anaemia (87.5%), constitutional symptoms (62.5%), and hyperviscosity syndrome (56.3%) were the most common presenting manifestations. Chemoimmunotherapy was administered as first-line treatment in 68.8% of patients, while 18.8% received bortezomib-based regimens. Among 13 evaluable patients, complete response was achieved in 38.5%, very good partial response in 23.1%, partial response in 15.4%, minor response in 7.7%, and stable disease in 7.7%. During follow-up, 18.8% experienced disease progression, 37.5% required second-line therapy, including BTK inhibitor-based treatment in 18.8%. At last follow-up, 75% of patients were alive.

Conclusion:

Indian patients with WM frequently present with symptomatic disease requiring treatment, with anaemia and hyperviscosity as the predominant manifestations. Chemoimmunotherapy remains an effective first-line strategy, while BTK inhibitors are increasingly incorporated in the relapsed setting. Larger multicentre studies with longer follow-up are required to better define long-term outcomes and optimize treatment sequencing in the Indian population.

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O-3

Frontline ICE therapy for Hepatosplenic T-cell lymphoma: a real-world single center experience from India

Presenting Author: Dr. Ayush Agarwal

Co-Authors: Dr. Abhinav Sengupta[1], Dr. Shubham Goswami[1], Dr. Jasmita Dass[2], Dr. Rishi Dhawan[2], Dr. Mukul Aggarwal[2], Dr. Tulika Seth[3], Dr. Manoranjan Mahapatra[3]

Institution: 1. Senior Resident, Dept of Hematology, AIIMS, Delhi
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3. Professor, Dept of Hematology, AIIMS, Delhi

Background:

Hepatosplenic T-cell lymphoma (HSTCL) is a rare and highly aggressive peripheral T-cell lymphoma with poor outcomes. Anthracycline-based regimens yield modest response rates, while platinum-containing intensive chemotherapy may improve remission rates and facilitate hematopoietic stem cell transplantation (HSCT). However, real-world data from low- and middle-income countries are scarce. We evaluated the feasibility, efficacy and outcomes of frontline ifosfamide, carboplatin and etoposide (ICE) in patients with HSTCL at a tertiary care center in India.

Method:

We performed a retrospective analysis of consecutive patients diagnosed with HSTCL between 2021 and 2026. Patients receiving frontline ICE were evaluated for treatment response, toxicity, HSCT, progression-free survival (PFS), and overall survival (OS). Complete response (CR) was defined by clearance of bone marrow disease by morphology and flow cytometry together with resolution of organomegaly. Survival was estimated using the Kaplan-Meier method.

Results:

Twenty-one patients were diagnosed during the study period. Median age was 27 years (IQR, 24–30), and 85.7% were male. Bone marrow involvement was present in all patients, while splenomegaly and hepatomegaly occurred in 84% and 55%, respectively. Four patients (19%) presented with secondary hemophagocytic lymphohistiocytosis. Three patients died before treatment initiation and one was lost to follow-up. Fifteen patients received frontline ICE, with a median of 4 (IQR, 2.5–5.5) cycles, including 53% managed using an outpatient schedule. Twelve patients who completed at least three cycles were evaluable for response. Seven achieved CR, yielding an overall response rate of 58.3%, with no partial responses observed. One patient experienced treatment-related mortality secondary to febrile neutropenia. Two patients subsequently underwent autologous HSCT and remain in ongoing complete remission at 19 and 32.5 months, respectively. At a median follow-up of 5 months (IQR, 1.3–7.8), median PFS and OS for the entire cohort were 11.3 months and 12.8 months, respectively, with estimated 1-year and 2-year overall survival rates of 54.1% and 20.3%, respectively. Among relapsed/refractory patients, responses to salvage therapies including GDP, DHAP, IVAC and hypomethylating agent-based regimens were uniformly poor.

Conclusion:

Frontline ICE is feasible in both inpatient and outpatient settings and achieves encouraging remission rates in HSTCL within a resource-limited setting. However, long-term outcomes remain poor, primarily because few patients are able to proceed to consolidative HSCT. Strategies that improve timely transplant access and incorporate novel targeted therapies are urgently needed to improve survival.

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O-4

Clinical Outcomes in Very Elderly (Age ≥80 years) Patients with Non-Hodgkin's Lymphoma: Experience from Tertiary care centre

Presenting Author: Dr. Nimmya SK

Co-Authors: Dr Nikhil Krishna Haridas, Dr Sourabh Radhakrishnan, Dr Rakesh MP, Dr Anjali, Dr Wesley Jose

Institution: Department of Medical Oncology, Amrita Institute of Medical Sciences, Kochi

Background:

Non-Hodgkin lymphoma (NHL) comprises a heterogeneous group of lymphoid malignancies that predominantly affect the elderly. With increasing life expectancy and improved diagnostic capabilities, the incidence of NHL in patients aged ≥80 years has risen. Management of this oldest-old population remains challenging because of frailty, multiple comorbidities, poor performance status, and under-representation in prospective clinical trials. Real-world data regarding this population remain limited.

Objective:

To evaluate the clinical characteristics, treatment patterns, and outcomes of patients aged ≥80 years with Non-Hodgkin lymphoma.

Method:

A retrospective review of the institutional Hospital Cancer Registry was performed from 2019 to 2025. Among 1,389 patients with lymphoma, 1,121 had Non-Hodgkin lymphoma. Patients aged ≥80 years were identified and analyzed for their clinicopathological characteristics, treatment received, and clinical outcomes.

Results:

Forty-five patients were analyzed, with a median age of 81 years (range 80–89 years); 86% (39) belonged to the 80–84-year age group and 77% (35) were male. Twenty-four percent had more than three comorbidities, while 44% had more than one comorbidity. Hypertension (51%) was the commonest comorbidity, followed by diabetes mellitus (33%). Cardiovascular comorbidities, including coronary artery disease, atrial fibrillation, and cardiomyopathy, were present in 24%. The most common presenting manifestation was lymphadenopathy with pain (31%). Mature B-cell lymphomas constituted the majority (82%, n=37), followed by NK/T cell lymphoma (9%, n=4), while primary CNS lymphoma and lymphoma NOS accounted for two cases each. Among B-cell lymphomas, 15% were GCB and 22.2% were non-GCB/ABC subtype. Stage IV disease was present in 57.8% and stage III disease in 22%. ECOG performance status was 3 in 42% of patients, while only 20% had an ECOG score of ≤1. Upfront best supportive care alone was recommended for 33.3% because of poor performance status and advanced disease. Twenty-two patients (48%) received systemic treatment, although only 13 completed the planned six cycles. Treatment included rituximab monotherapy, R CHOP, R-CVP, R-mini-CHOP, bendamustine-rituximab, lenalidomide-based therapy, and chlorambucil-rituximab, with dose modifications based on performance status and tolerance. Five patients who completed planned treatment achieved a sustained response and remained alive at last follow-up. Three patients were presented with perforation peritonitis requiring emergency surgery, while three received palliative radiotherapy alone. Overall, 73% (33) of patients died, with disease progression being the leading cause of death, followed by infection and sepsis. The Median overall survival was 2 months and Mean Overall Survival was 6 months (range 0–31 months).

Conclusion:

Patients aged ≥80 years with Non-Hodgkin lymphoma represent a highly vulnerable population characterized by advanced-stage disease, poor performance status, and a substantial burden of comorbidities. Treatment completion rates and survival remain poor despite individualized therapy, highlighting the need for careful patient selection, comprehensive geriatric assessment, and treatment strategies that balance disease control with quality of life.

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O-5

Outcomes of Adult and Adolescent Acute Lymphoblastic Leukemia Treated with Paediatric inspired protocol: A Single-Center Retrospective Analysis of Toxicity, MRD Response, and Survival

Presenting Author: Devireddy Raj Mohan Reddy

Co-Authors: Anant Gokarn, Sachin Punatar, Sumeet Mirgh, Yamuna Naik, Akanksha Chichra, Lingaraj Nayak, Nishant Jindal, Avinash Bonda, Bhausaheb Bagal, and Navin Khattry.

Institution: Dept. of Medical Oncology, Advanced Centre for Treatment, Education and Research (ACTREC), Tata Memorial Centre, Kharghar, Navi Mumbai – 410210

Background:

Adult acute lymphoblastic leukemia (ALL) carries inferior outcomes compared with pediatric disease, driven by adverse biology, treatment-related toxicity, and residual disease burden. Real-world outcome data from intensified pediatric-inspired protocols in resource-constrained settings remain limited.

Objective:

To describe baseline demographic and disease characteristics, induction-phase toxicity, measurable residual disease (MRD) response, and survival outcomes in a single-center adult/adolescent ALL cohort, and to evaluate the prognostic impact of post-induction MRD status on relapse and overall survival.

Materials and Method:

This retrospective analysis included patients with ALL or MPAL treated with intensified multi-agent pediatric inspired chemotherapy regimen at ACTREC, Tata Memorial Centre between 1st January 2007 and 31st December 2023. Demographic, treatment-toxicity, MRD (multiparameter flow cytometry), relapse, and follow-up data were extracted from institutional records. Overall survival (OS) was estimated using the Kaplan-Meier method from date of diagnosis to death or last follow-up; subgroups were compared using the log-rank test, and categorical associations using the chi-square test, with $p<0.05$ were considered significant.

Results:

Median age was 30.5 years (range 13–65) with a male predominance (68%). Subtypes included B-cell precursor ALL (38%), Ph-positive B-ALL (21%), T-ALL (21%), early T-precursor ALL (13%), and mixed-phenotype leukemia (7%). During induction, bacterial sepsis occurred in 44% (culture proven in 14%), fungal infection in 20%, and thrombotic events in 8% of patients. Post-induction MRD was positive in 54% of evaluable patients and was significantly associated with higher relapse (48% vs. 19%, $p=0.002$) and inferior 2-year OS (55% vs. 80%, log-rank $p=0.0015$; Figure 1). Marrow, CNS, and testicular relapse occurred in 33%, 7%, and 2%

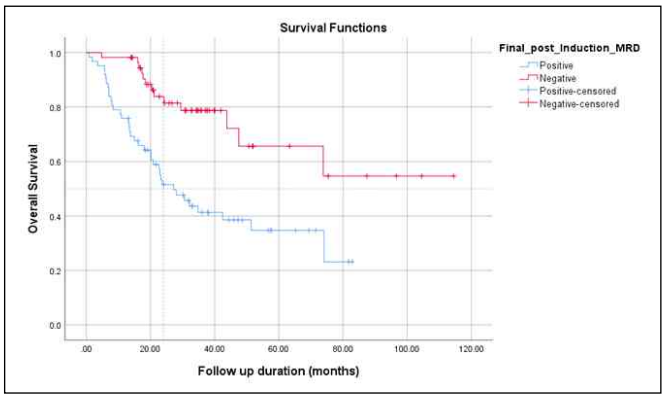

Figure 1:
Impact of MRD post induction on survival

respectively. Only 14% underwent allogeneic transplant. At median follow-up of 23 months, 1- year, 2-year, 5-year OS were 82%, 63% and 45% (Figure 2).

Conclusion:

Post-induction MRD status is a strong independent predictor of relapse and survival in this real-world adult ALL cohort, supporting wider use of allogeneic HSCT in patients who remain MRD-positive after induction. Infectious and thrombotic complications during induction remain substantial, underscoring the need for risk-adapted supportive care alongside MRD-directed treatment intensification, including timely allogeneic transplant where indicated.

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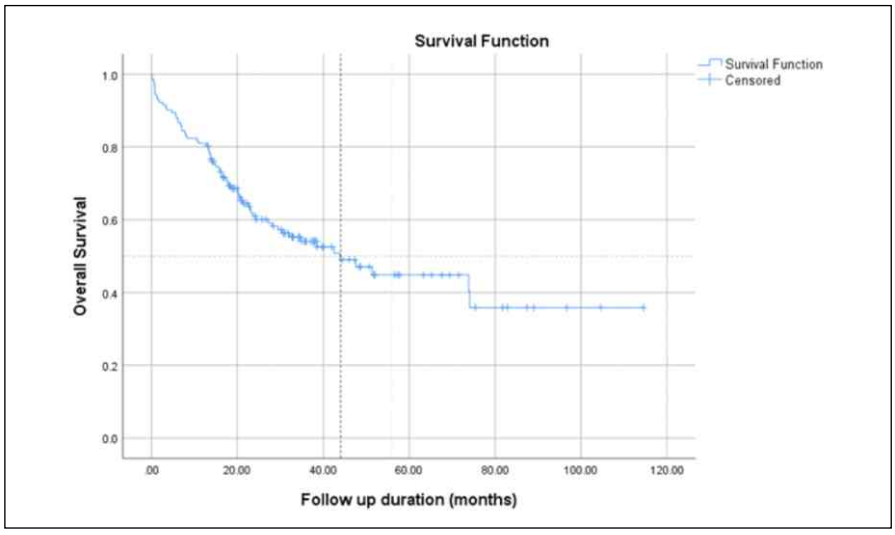
Figure 2:
Overall survival of study population



O-6

Molecular landscape and treatment outcomes of acute myeloid leukemia: A 2-year single-center experience from a tertiary care hospital in South India

Presenting Author: Dr. Aakash Rajendra Gupta

Co-Authors: Dr. Neeraj Siddharthan[1], Dr. Manoj Unni[1], Dr. Nikhil Haridas[1], Dr. Rema[1], Dr. Suchitra[2], Dr. Thomas Kuncheria[2]

Institution: 1. Division of Stem Cell Transplantation, Department of Clinical Haematology, Amrita Institute of Medical Sciences, Kochi
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Background:

Acute Myeloid Leukemia (AML) is a genetically heterogeneous hematological malignancy in which molecular and cytogenetic abnormalities determine disease biology, risk stratification, treatment selection, and prognosis. Real-world data integrating molecular profiles with treatment patterns and outcomes from Indian tertiary care centers remain limited.

Objective:

To evaluate the baseline demographics, spectrum of molecular genetic testing, first-line induction choices, subsequent consolidation strategies, and clinical outcomes (transplant rates, remission, and survival status) in a real-world cohort of AML patients

Method:

A retrospective observational study was conducted including consecutive AML patients diagnosed and treated over a two-year period at a tertiary care center. Clinical records were reviewed for demographic characteristics, disease status, ELN 2022 molecular risk classification, molecular genetic profile, first-line induction therapy, consolidation strategy, allogeneic hematopoietic stem cell transplantation (allo-HSCT), treatment response, measurable residual disease (MRD) status, and survival outcomes.

Results:

47 AML patients were included, with a median age of 54 years (range, 1–73 years); 55.3% were male. De novo AML accounted for 83.0% of cases, while 42.6%, 34.0%, and 23.4% belonged to the adverse-, intermediate-, and favourable-risk ELN categories, respectively. Aza-ven (42.6%) and 7+3 induction (36.2%) were the most frequently administered frontline regimens. Best documented responses included complete remission (CR/CR1/CR2) in 26 patients, with additional patients achieving MRD-negative remission. 9 patients underwent allogeneic HSCT, predominantly haploidentical transplantation, with all transplanted patients remaining in MRD-negative remission during last follow-up. At last follow-up, 24 patients were alive of which 21 were MRD-negative, 8 had died, 9 were lost to follow-up, and 3 remained on on-going treatment. Patients with favourable-risk molecular abnormalities, including core-binding factor rearrangements and isolated NPM1/CEBPA mutations, demonstrated superior remission rates and durable disease control, whereas adverse-risk lesions, including TP53 mutations, complex karyotype, KMT2A rearrangements, FLT3-associated disease, and myelodysplasia-related mutations, were associated with refractory disease, relapse, and inferior outcomes.

Conclusion:

This real-world cohort highlights the prognostic significance of comprehensive molecular profiling in AML. ELN 2022 risk stratification closely correlated with treatment response and clinical outcomes, while integration of molecular diagnostics enabled risk-adapted therapeutic strategies, including venetoclax-based regimens and allogeneic stem cell transplantation. These findings support the routine incorporation of molecular testing into AML management in resource-constrained settings.

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O-7

The Pre-engraftment Window: Bloodstream Infection following Allogeneic Haematopoietic Stem Cell Transplantation

Presenting Author: Dr. Goh Jun Yan

Co-Authors: Dr. Hany Ariffin

Institution: Haematology-Oncology and Bone Marrow Transplantation Unit, Universiti Malaya Medical Centre, Kuala Lumpur, Malaysia

Background:

Pre-engraftment bloodstream infection (BSI) is a life-threatening complication following allogeneic haematopoietic stem cell transplantation (allo-HSCT), occurring during a period of profound immunosuppression and heightened susceptibility to sepsis. Despite its clinical significance, local epidemiological data remain limited, hindering the development of targeted infection prevention and management strategies.

Objective:

To determine the incidence, risk factors, and clinical outcomes of pre-engraftment BSI in our unit.

Method:

We retrospectively reviewed allo-HSCT recipients transplanted between January 2016 and December 2025. Demographic, clinical and microbiological data were extracted from medical records. Pre-engraftment was defined as the period from stem cell infusion until an absolute neutrophil count $>0.5 \times 10^9$/L was achieved for three consecutive days.

Results:

A total of 151 consecutive patients were included, of whom 92 (60.9%) were male. The median age at transplantation was 6.2 years (IQR 3.4 – 11.1). Indications for HSCT included haematological malignancies (n=85, 56.3%), inborn errors of immunity and bone marrow failure (n=46, 30.5%), and haemoglobinopathies (n=20, 13.2%). The median pre-engraftment duration was 15 days (IQR 13 – 17) among 135 patients. Graft failure occurred in 16 patients (10.6%), of whom 10 (62.5%) were successfully salvaged and subsequently achieved engraftment. Pre-engraftment BSI occurred in 36 patients (23.8%), with a median onset of 9.5 days (IQR 8 – 11) following stem cell infusion. Gram-negative organisms accounted for the majority of infections (n=22, 61.1%), with Klebsiella spp. predominating. On multivariable analysis, HLA-haploidentical donor transplantation was independently associated with an increased risk of pre-engraftment BSI (OR 5.71, 95% CI 1.88 – 17.33, p=0.002). Patients who developed pre-engraftment BSI had significantly higher 100-day all-cause mortality (OR 4.41, 95% CI 1.47 – 13.19, p=0.008).

Conclusion:

Pre-engraftment BSI remains a significant complication, affecting nearly one-quarter of allo-HSCT recipients and was associated with approximately fourfold higher odds of 100-day mortality. Patients undergoing HLA-haploidentical transplantation represent a key high-risk group for targeted preventative strategies.

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O-8

Standardization of Skin Fibroblast Culture for Germline Testing in Acute Myeloid Leukemia

Presenting Author: Swastik Sastri

Co-Authors: Swastik Sastri, Sweta Rajpal*, Ishita Mathur, Roshni Lal, Nikhil Patkar, Syed K. Hasan*

Institution: Hasan Lab, KS-256, Khanolkar Shodhika, Advanced Centre for Treatment, Research and Education in Cancer (ACTREC), Kharghar, Navi Mumbai, Maharashtra 410210

Background:

Acute Myeloid Leukemia (AML) is a genetically heterogeneous myeloid neoplasm characterized by the aberrant proliferation of hematopoietic stem cells (HSCs) and impaired differentiation of myeloid lineage cells. Advancements in high-throughput technologies, such as next-generation sequencing (NGS), have established that approximately 5–10% of myeloid neoplasms (MNs), including AML, harbour pathogenic germline variants in hematopoietic regulatory genes. Hereditary myeloid malignancy syndromes (HMMS) are inherited genetic predispositions caused by germline mutations transmitted across generations, predisposing individuals to the development of blood disorders such as MDS or AML. Most HMMS-associated genes are tumor suppressors; therefore, affected individuals are initially asymptomatic carriers, possessing one mutant germline allele while the second allele remains wild type. With increasing age, loss of the wild-type allele or acquisition of a somatic second hit, followed by cooperating mutations, leads to loss of gene function and progression to AML or MDS.

Objective:

To establish a highly enriched fibroblast culture from skin biopsies of Indian AML patients for screening of germline variants.

Method:

Skin biopsy-derived fibroblasts were established from MPAL patient and characterized by morphology and flow cytometry using CD133, CD44, CD45, CD34, CD326, and intracellular vimentin. Targeted next-generation sequencing (NGS) was performed on matched peripheral blood and fibroblast samples from the same patient to distinguish somatic from germline variants. The established fibroblast models will subsequently be utilized for CRISPR-Cas9-mediated knock-in of germline variants for their functional characterization.

Results:

Patient-derived fibroblasts were successfully established from skin biopsy samples and showed characteristic spindle-shaped morphology with good proliferative capacity. Flow cytometric analysis confirmed enrichment of fibroblasts based on positive expression of CD133, CD44 and intracellular vimentin, with negative expression of CD45, CD34, and CD326. Targeted NGS identified a heterozygous TP53 mutation (VAF ~41%) in peripheral blood, whereas the mutation was absent in matched skin biopsy-derived fibroblasts, confirming its somatic origin and supporting fibroblasts as a suitable source for germline variant screening.

Conclusion:

Patient-derived fibroblasts provide a reliable source of germline DNA by eliminating interference from somatic mutations present in peripheral blood. Successful establishment of patient-derived fibroblast models provides the foundation for future CRISPR-mediated functional characterization of germline variants to investigate their role in the pathobiology of hereditary myeloid malignancy syndromes.

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O-9

Age-Stratified Molecular Profiling and ELN 2022 Risk Distribution in Acute Myeloid Leukemia Patients Younger Than 60 Years: A Single-Center Retrospective Analysis

Presenting Author: Neerukonda Sriteja

Co-Authors: Ashish Dixit, Mallikarjun Kalashetty

Institution: Department of Clinical Haematology, Manipal Hospital, No. 98, HAL Old Airport Road, Kodihalli, Bengaluru, Karnataka, 560017, India.

Background:

Acute myeloid leukemia (AML) in younger adults is a biologically heterogeneous disease with age-dependent variation in cytogenetic and molecular architecture. The European Leukemia Net (ELN) 2022 recommendations integrate these genetic abnormalities into a three-tier risk classification, but real-world data describing how molecular profiles and ELN risk categories evolve across successive decades of life in patients under 60 years remain limited, particularly from resource-constrained settings.

Objective:

To characterize the clinical, demographic, and molecular landscape of AML across sequential age subgroups in patients younger than 60 years and to evaluate age- related patterns in ELN 2022 risk distribution.

Method:

We conducted a single-center retrospective analysis of 119 patients aged 10–59 years diagnosed with AML. Patients were stratified into five decade-based subgroups: 10– 19, 20–29, 30–39, 40–49, and 50–59 years. Targeted mutational testing included FLT3, NPM1, CEBPA, TP53, IDH1, IDH2, DNMT3A, and NRAS. Risk classification followed the ELN 2022 criteria. Categorical comparisons across age subgroups were performed using the Chi-square test (or Fisher's exact test where appropriate), with two-sided $p<0.05$ considered significant.

Results:

The cohort comprised 11 (9.2%) patients aged 10–19 years, 15 (12.6%) aged 20– 29, 36 (30.3%) aged 30–39, 34 (28.6%) aged 40–49, and 23 (19.3%) aged 50–59 years; median age was 38 years and 47.1% were male, with no significant sex difference across subgroups ($p=0.181$). Secondary AML rose progressively with age, from 0–9% in patients <30 years to 21.7–38.2% in those ≥40 years ($p=0.040$). Among tested patients, mutation positivity was 43.5% for FLT3, 47.7% for NPM1, 48.0% for CEBPA, 60.0% for TP53, 52.9% for IDH1, and 61.1% for IDH2. CEBPA mutations clustered in the 30–39 year group (85.7% positivity) and were absent in the 50–59 year group ($p=0.064$), whereas TP53 mutations were absent below 30 years and concentrated in patients 40–59 years. ELN risk distribution differed significantly across subgroups ($p=0.008$): favorable-risk disease predominated in the 20–29 year group (90.0%) and 10–19 year group (55.6%), while adverse-risk disease predominated in the 40–49 (50.0%) and 50–59 year (46.7%) subgroups.

Conclusion:

AML in patients under 60 years exhibits distinct, age-dependent molecular and ELN risk profiles, with a clear shift from favorable-risk, CEBPA-enriched disease in adolescents and young adults to adverse-risk, TP53-enriched, and secondary AML in older subgroups. These findings underscore the value of routine, comprehensive molecular profiling and age-aware ELN 2022 risk assignment to inform individualized prognostication in younger AML populations.

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Clinical Outcomes of Hypomethylating Agents in Acute Myeloid Leukemia: A Single-Centre Retrospective Study from South India

Presenting Author: Moiz Bashir Doctor

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Background:

Hypomethylating agents (HMAs) have become the standard low-intensity therapeutic option for older adults and medically unfit patients with acute myeloid leukemia (AML). However, real-world data evaluating treatment outcomes and prognostic factors in the Indian population remain scarce. This study evaluated clinical characteristics, treatment response, survival outcomes, and predictors of response among AML patients receiving HMA-based therapy at a tertiary care centre.

Objective:

To characterize the clinical, demographic, and molecular landscape of AML across sequential age subgroups in patients younger than 60 years and to evaluate age- related patterns in ELN 2022 risk distribution.

Method:

This retrospective observational study included treatment-naïve AML patients who received at least one cycle of azacitidine or decitabine, with or without venetoclax, at Manipal Comprehensive Cancer Centre, Bengaluru. Patients with relapsed/refractory AML, acute promyelocytic leukemia, prior intensive chemotherapy, or concomitant solid organ malignancies were excluded. Demographic, clinical, hematological, cytogenetic, molecular, treatment, and toxicity data were retrieved from electronic medical records. Responses were assessed according to ELN 2022 criteria. Overall survival (OS) and relapse-free survival (RFS) were estimated using Kaplan–Meier analysis, while factors associated with response and survival were evaluated using univariate logistic regression.

Results:

Fifty-three patients were included, with a median age of 64 years (IQR 57–70); 56.6% were male. Intermediate-, adverse-, and favourable-risk ELN categories comprised 47.2%, 22.6%, and 15.1% of patients, respectively. Azacitidine and decitabine were administered in 43.4% and 41.5% of patients, while 17.0% received venetoclax in combination with HMAs. The median number of HMA cycles was six.

Following three treatment cycle, the overall response rate (CR/CRh/CRi/PR) was 47.2%, including complete remission (CR) in 24.5%, CRh/CRi in 15.1%, and partial response (PR) in 7.5%. At six cycles, the overall response rate was 28.3%, with CR achieved in 13.2% of patients; 15.1% had refractory disease and 11.3% experienced relapse. Median overall survival was 14.3 months, while responders demonstrated superior survival compared with non-responders (18.9 vs. 7.9 months). Median relapse-free survival among responders was 13.4 months.

On univariate analysis, increasing age, adverse ELN risk, myelodysplasia-related changes, elevated baseline white blood cell count, and peripheral blood blasts were associated with inferior response and survival, whereas venetoclax addition and a greater number of HMA cycles were associated with improved treatment response.

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Conclusion:

HMA-based therapy provides meaningful clinical benefit in treatment-naïve AML patients who are unsuitable for intensive chemotherapy, with nearly half of patients achieving an initial response and a median overall survival exceeding one year. Baseline disease biology and incorporation of venetoclax significantly influence treatment outcomes. These real-world data support individualized HMA-based treatment strategies and reinforce the role of venetoclax-containing regimens in appropriately selected patients.

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O-11

Podoplanin Expression Improves the Diagnostic Accuracy of Acute Promyelocytic Leukemia

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Background:

Acute Promyelocytic Leukemia is the M3 subtype of Acute Myeloid Leukemia (AML) characterised by the accumulation of abnormal promyelocytes in both blood and bone marrow, caused by a balanced reciprocal translocation between the promyelocytic leukemia (PML) and retinoic acid receptor alpha (RAR) genes. APL is considered a medical emergency and early diagnosis and treatment is critical, as delays lead to a unique bleeding diathesis and elevated risk of early mortality. APL is diagnosed using microscopy, immunophenotyping and FISH/PCR for PML-RARA. Although the current diagnostic method immunophenotyping is reliable but morphological overlapping with AML-M5 and AML-M2 often misdiagnose APL cases. This study investigates the role of Podoplanin (transmembrane glycoprotein) that interacts with the C-type lectin-like receptor 2 (CLEC-2) on platelets, triggering their activation and contributing to thrombotic processes, as a potential diagnostic biomarker for APL.

Objective:

To evaluate the expression levels, transcriptional regulation mechanisms and the clinical utility of Podoplanin (PDPN) as a diagnostic biomarker to accurately distinguish APL from non-APL AML.

Method:

73 clinical samples (52 APL and 21 non-APL AML) collected from bone marrow or peripheral blood at the time of diagnosis were analyzed. PDPN expression was evaluated with mRNA levels using Real-Time Quantitative PCR (RQ-PCR) and cell surface protein expression via 10-color multiparametric flow cytometry. The panel included monoclonal antibodies against CD11b, CD14, CD15, CD33, CD34, CD36, CD45, CD117, CD123, HLA-DR, and Podoplanin. Furthermore, computational analysis of PDPN gene expression using the TCGA-LAML (14 APL and 137 non-APL AML) and BEATAML1.0-COHORT (20 APL and 647 non-APL AML) datasets were also performed. Transcriptional regulation was investigated by identifying transcription factor binding sites on the PDPN promoter region with the JASPAR (2022) database, followed by Chromatin Immunoprecipitation (ChIP-qPCR) in NB4 APL cells to confirm physical binding by SMAD proteins. The TGF-β/SMAD signalling pathway was further validated by treating NB4 cells with a recombinant TGF-β1 ligand and the inhibitor Luspatercept, observing the downstream effects on PDPN expression and SMAD activation via immunoblotting. Enzyme-Linked Immunosorbent Assay (ELISA) was utilized to quantify activated TGF-β1 ligand levels in the serum of the APL vs non-APL AML patients.

Results:

PDPN mRNA copy numbers and protein surface expression (median 9.34% PDPN positive cells vs. 0.40%) were significantly higher in APL patients than in non-APL AML patients ($p < 0.0001$). Flow cytometry evaluating PDPN surface protein expression showed 92.86% sensitivity and 100% specificity (AUC = 0.9592, $p < 0.0001$) at a 1.4% cutoff. RQ-PCR evaluation showed 80.7% sensitivity and 71.43% specificity (AUC = 0.84, $p < 0.0001$) at a cutoff of 10 mRNA copies. PDPN expression displayed a significant negative correlation with patient platelet counts consistent with known function of PDPN in promoting platelet aggregation. ChIP-qPCR analysis confirmed a direct physical interaction (five- to seven-fold) enrichment of SMAD proteins binding to the PDPN

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upstream promoter region following TGF-β1 stimulation. APL cells when treated with TGF-β1 ligand increased SMAD phosphorylation and upregulated PDPN. Alternately, using the inhibitor Luspatercept significantly reduced both SMAD phosphorylation and PDPN levels. APL patients also had significantly elevated levels of TGF-β1 ligand in their serum compared to non-APL AML patients.

Conclusion:

The study establishes Podoplanin (PDPN) overexpression in APL as a downstream effect driven by the TGF-β/SMAD signalling pathway. The surface expression of PDPN highlights its role as a diagnostic biomarker due to its high sensitivity and specificity. PDPN could be incorporated as a positive diagnostic marker alongside standard immunophenotyping panels to significantly improve the accuracy of APL detection and reduce the risk of misdiagnosis in clinical settings.

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O-12

Life Beyond TKI: Real-World Experience of Treatment-Free Remission in Chronic Myeloid Leukemia from North India

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Background:

Treatment-free remission (TFR) has emerged as an important therapeutic goal in chronic-phase chronic myeloid leukemia (CML), allowing selected patients with sustained deep molecular response (DMR) to discontinue tyrosine kinase inhibitor (TKI) therapy without compromising disease control. However, real-world data from India remain limited.

Method:

We retrospectively analyzed twenty-four adult patients with chronic-phase CML who discontinued tyrosine kinase inhibitor (TKI) therapy after sustained DMR between January 2016 and December 2024 at a tertiary care center in North India. The primary outcome was successful TFR, defined as maintenance of major molecular response (MMR) without TKI therapy. Secondary endpoints included molecular relapse-free survival (MRFS), recovery of MMR after TKI re-initiation, predictors of successful TFR, and safety outcomes.

Results:

After a median follow-up of 18 months (IQR, 10–32), 14 patients (58.3%) maintained TFR, whereas 10 (41.7%) experienced molecular relapse requiring TKI re-initiation; all regained major molecular response (MMR) (100%). Kaplan–Meier analysis demonstrated estimated molecular relapse-free survival rates of 69.8% at 6 months, 58.2% at 12 and 24 months, and 46.6% at 36 months. Longer TKI duration before discontinuation (median 90.0 vs. 70.5 months; P=0.106), prolonged sustained DMR (59.5 vs. 43.0 months; P=0.095), low ELTS risk (62.5% vs. 33.3%), and second-generation TKI therapy (75.0% vs. 55.0%; P=0.615) were associated with higher TFR rates, although none reached statistical significance. TKI withdrawal syndrome occurred in 8 patients (33.3%) and was limited to mild musculoskeletal symptoms. No patient experienced loss of hematologic response, progression to accelerated/blast phase, or death during follow-up.

Conclusion:

TKI discontinuation is a feasible and safe strategy for carefully selected patients with chronic-phase CML and sustained DMR. Most relapses occurred within the first year and responded successfully to TKI re-initiation, supporting the implementation of TFR in routine clinical practice with standardized molecular monitoring.

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Clinical profile and response to TKI of paediatric chronic myeloid leukemia: an experience from a tertiary care hospital from the Himalayan foothills, India

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Background:

Pediatric CML is a rare disease entity and patients usually present with severe clinical manifestations in comparison to adults. The present study was conducted to assess the clinical profile and response to standard tyrosine kinase inhibitors therapy in pediatric CML patients.

Objective:

To assess the clinical characteristics and early hematological and molecular response to standard TKI therapy in pediatric CML patients.

Method:

A retrospective analysis was done in 18 patients of CML aged less than 18 years at the time of diagnosis. Recruitment of patients was done from Medical Oncology Haematology out-patient department of a tertiary care hospital in the foothills of Himalayas. Sociodemographic profile, baseline characteristics, disease specific risk scores and bone marrow aspiration biopsy cytogenetics were done at the baseline. Quantitative BCR- ABL was done at baseline and at 3 months.

Results:

Median age of patients was 16 years and majority of them were males. Fever and abdominal heaviness were the most common presenting symptoms. Splenomegaly was associated in 88.9% with a median spleen size of 16cm and hepatomegaly was found in 27.8%patients. Majority of patients were of Sokal high grade, Hasford intermediate and EUTOS high grade. Early molecular response was seen in 66.7% and complete haematological response was seen in 81.25% patients.

Conclusion:

Treatment with Tyrosine kinase inhibitors produces a sub-optimal early molecular response in pediatric CML patients. Appropriate treatment strategies are needed to be formulated for effective management of pediatric CML.

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O-14

The utility of NGS-based chimerism analysis for the interpretation of STR-PCR results in patients after a second allogeneic hematopoietic cell transplantation

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Background:

Chimerism monitoring after allogeneic hematopoietic cell transplantation (allo-HCT) is an important tool for assessing engraftment and treatment efficacy. Interpretation of chimerism results may be challenging in patients undergoing consecutive transplants from different donors.

Aim:

To evaluate the usefulness of next-generation sequencing (NGS)-based chimerism analysis in clarifying ambiguous STR-PCR chimerism results after a second allo-HCT.

Materials and Method:

Two female patients with acute myeloid leukemia (AML) who underwent two consecutive allo-HCT procedures from male donors were analyzed. Following the second transplantation, peripheral blood STR-PCR demonstrated mixed chimerism. Results were compared with bone marrow FISH analysis, bone marrow immunophenotypic assessment and NGS-based chimerism analysis.

Results:

In both patients, FISH analysis demonstrated complete donor chimerism, and bone marrow immunophenotyping confirmed complete remission with negative measurable residual disease. Despite these findings, STR-PCR indicated mixed chimerism. NGS analysis revealed different patterns; in the patient after reduced-intensity conditioning (RIC), mixed chimerism reflected the coexistence of hematopoiesis derived from two both transplant donors, without evidence of recipient-derived cells. In contrast, in the patient receiving myeloablative conditioning (MAC), NGS demonstrated complete replacement by the second donor (100%). NGS findings resolved the apparent discrepancy between STR-PCR and other monitoring methods and excluded autologous hematopoietic recovery in both cases.

Patient	1	2
age	48	28
Recipient sex	female	female
Donor 1 sex	male	male
Donor 2 sex	male	male
1st allo-HCT	20.07.23	13.08.19
2nd allo-HCT	31.07.24	3.09.25
conditioning before 2nd allo-HCT	RIC (Flu/Treo)	MAC (Flu/Bu4)
FISH XY day+100	100%	100%
BM immunophenotype	CR	CR
STR-PCR PB Day +30	99%	98%
STR-PCR PB Day +180	72%	98%
NGS PB chimerism	Recipient 0%; 1 Donor 25%, 2 Donor 75%	Recipient 0%, 1 Donor 0%, 2 Donor 100%

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Conclusion:

Similar STR-PCR results after a second allo-HCT may reflect distinct biological situations. NGS-based chimerism analysis allows accurate identification of the origin of hematopoiesis and may improve interpretation of discordant post-transplant monitoring results.

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O-15

Outcomes of donor lymphocyte infusion for pre-emptive or therapeutic use after allogeneic HSCT: a single-center experience

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Background:

Relapse of hematological malignancies remains the leading cause of mortality after allogeneic hematopoietic stem cell transplantation (allo-HSCT). Donor lymphocyte infusion (DLI) enhances graft-versus-leukemia effects.

Objective:

To assess the clinical characteristics and early hematological and molecular response to standard TKI therapy in pediatric CML patients.

Method:

We retrospectively evaluated the effectiveness and safety of DLI administered at our center between 2015 and 2025. Treatment response was assessed using STR-PCR chimerism and minimal residual disease (MRD) monitoring by flow cytometry and cytogenetic studies using disease-specific FISH probes.

Results:

A total of 105 DLI infusions were administered to 42 patients; 23 preemptively (pre-DLI) and 19 therapeutically (t-DLI). Underlying diseases were mainly myeloid malignancies, including AML (n=24) and MDS (n=5). Donors were matched unrelated in 59% and cconditioning was myeloablative in 65% of patients.

The median lymphocyte dose was 1×10^6/kg (5×10^5–1×10^8), with a median first dose of 5×10^5/kg (5×10^5–5×10^6). Patients received a median of 2 DLI infusions (range 1–7).

Twenty-two AML/MDS patients received azacitidine.

In the pre-DLI group, median follow-up from first DLI was 40 months (range 2–132). Thirteen patients (56%) remain in continuous complete remission (CR), three (13%) relapsed, two (9%) died and five (22%) proceeded to a second allo-HSCT.

Among AML/MDS patients receiving pre-DLI ± AZA, CR was achieved in 12/15 (80%).

In the t-DLI group, CR was achieved in 4/19 patients (21%), including 4/13 AML/MDS patients (30%) treated with azacitidine. Remissions lasted 2, 9, 12, and 15 months; two patients subsequently relapsed, while two remain in ongoing remission at 9 and 12 months. Median survival from first DLI was 5 months (range 0.5–35).

Among relapsed AML/MDS patients, clonal evolution was detected in 4/7 (57%) and new cytogenetic clones in two additional cases. GvHD occurred in four patients, including one fatal case of acute gastrointestinal GvHD.

Conclusion:

Pre-emptive DLI, particularly in combination with azacitidine, induces durable remissions in AML/MDS patients. Outcomes remain limited in overt relapse. Clonal evolution or new cytogenetic abnormalities appear to reduce the likelihood of response. Overall, DLI was well tolerated, with a low incidence of clinically significant GvHD.

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O-16

Treosulfan-based conditioning is associated with better outcomes in adolescents and young adults undergoing matched related donor transplantation for Thalassemia major

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Background:

Outcomes of allogeneic transplantation for Thalassemia Major (TM) in adolescents and young adults (AYA) remain suboptimal in resource-limited settings due to late referral, iron overload, and high-risk disease biology. Evidence comparing Treosulfan-based regimens with conventional Busulfan-based conditioning in this age group is limited.

Method:

We performed a retrospective analysis of AYA patients (≥12 years) with TM who underwent matched sibling or family donor transplantation at our centre between 1994–2022. Conditioning consisted of either Busulfan–Cyclophosphamide (Bu/Cy ± ATG) or Treosulfan–Fludarabine–Thiotepa (FTT). Standard institutional protocols were followed for GVHD prophylaxis, supportive care, and engraftment monitoring. Overall survival (OS) and thalassemia-free survival (TFS) were estimated using Kaplan–Meier analysis. Multivariable Cox regression was used to assess prognostic factors.

Results:

A total of 124 AYA patients underwent MRD transplant. Median age was 15 years (13–25); 88.7% were Lucarelli Class III, and 65.3% belonged to the Vellore high-risk subgroup. The median ferritin level was 3490 ng/mL (range: 361 – 16761). Conditioning comprised Bu/Cy in 46 (37.1%) and FTT in 73 (58.9%) patients. Peripheral blood stem cells were used in 94.5% of FTT recipients versus 6.5% in the Bu/Cy group.

Engraftment occurred in 111 patients (89.5%); median neutrophil and platelet engraftment times were 16(range: 8 - 43) and 21(range: 10 - 66) days, respectively. Primary graft failure occurred in 3.2% and secondary graft failure in 6.4%. Treatment-related mortality by Day 100 was significantly lower with FTT (15%) compared to Bu/Cy (39.1%, p=0.002).

Sinusoidal obstruction syndrome (SOS) occurred in 38.7%, with significantly lower incidence in the FTT group (31.5% vs 54.3%, p=0.021). Hemorrhagic cystitis was also less frequent with FTT (2.7% vs 19.6%, p=0.003). Grade II–IV acute GVHD occurred in 34.2% and chronic GVHD in 31.4% of evaluable patients.

The median duration of follow-up after excluding early deaths was 59 months (range: 1 - 289 months). For the entire cohort, 5-year OS and TFS were 63.6+ 4.5% and 60.1+ 4.5% respectively. FTT was associated with significantly superior survival: OS 72.2% vs 54.8% (p=0.017) and TFS 68.7% vs 51.1% (p=0.018). Platelet engraftment was faster, complete chimerism at Day 30 was higher (92.8% vs 78.6%, p=0.023), and regimen-related toxicity was reduced in the FTT cohort.

In Lucarelli Class III patients overall, FTT showed significantly improved survival, with a 5-year OS of 71.9% vs 52.5% for Bu/Cy (p=0.014) and a 5-year TFS of 69.3% vs 52.5% (p=0.029). In the Vellore High-Risk subset, the benefit was even more, with 5-year OS of 75.2% vs 40.2% (p=0.002) and 5-year TFS of 69.0% vs 40.2% (p=0.003) for FTT compared with Bu/Cy. Outcomes were comparable between patients aged 12–18 and those >18 years within the FTT group.

Conclusion:

Treosulfan-based conditioning significantly reduces early toxicity and improves long-term OS and TFS in AYA patients with TM undergoing MRD transplantation, including those in high-risk subgroups. These findings support Treosulfan-based conditioning as the preferred regimen for AYA patients in resource-constrained settings.

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From Vision to Reality: Establishing a Pediatric Bone Marrow Transplant Center in a Government Hospital in Central India

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Background:

Pediatric hematopoietic stem cell transplantation (HSCT) in India is largely limited to metropolitan cities; hence, access is restricted to only those who can afford to travel and stay in these cities for almost six months. Consequently, the expenditure remains predominantly out-of-pocket despite bone marrow transplantation being supported by various government schemes and non-governmental, non-profit organizations. Even when travel and treatment expenses are covered, the entire family suffers because of prolonged loss of income and absence from work. Therefore, it is of utmost importance to establish such centres in every state, preferably one in smaller states and two to three in larger states, to make treatment affordable and accessible to all. Although training programs in BMT do exist, they need to be expedited, and adequate resources must be provided to ensure job satisfaction and retention of trained personnel.

Objective:

To describe the establishment of a government-supported pediatric HSCT program in a tier-2 city and evaluate its early clinical outcomes using the Donabedian Structure–Process–Outcome framework.

Method:

This single-center descriptive case series included consecutive pediatric HSCT procedures performed between March 2025 and May 2026 at a newly established pediatric HSCT unit in central India. Program development involved phased infrastructure creation, personnel training, blood bank upgradation, implementation of infection-control measures, and collaborative diagnostic partnerships. Clinical outcomes, engraftment, transplant-related complications, and survival were analyzed using descriptive statistics.

Results:

Nine children underwent HSCT, including six allogeneic (66.7%) and three autologous (33.3%) transplants. The median age was 5 years (range, 3–12 years). Indications included sickle cell disease, sickle beta-thalassemia, transfusion-dependent beta-thalassemia major, refractory acute myeloid leukemia, and high-risk neuroblastoma. Median neutrophil engraftment occurred at 12 days (range, 10–20 days), and the median duration of hospitalization was 31 days (range, 24–34 days). Acute graft-versus-host disease (GVHD) occurred in two patients (22.2%), while cytomegalovirus reactivation, veno-occlusive disease, and documented sepsis occurred in one patient each (11.1%). Febrile neutropenia was observed in three patients (33.3%). No cases of invasive fungal infection, graft failure, chronic GVHD, or transplant-related mortality were recorded. With a median follow-up of 6 months (range, 2–10 months), overall survival at last follow-up was 100%.

Conclusion:

Performing pediatric HSCT is no longer the major challenge it once was, as the number of trained transplant physicians in India continues to increase. However, the greatest challenge today is making HSCT accessible and

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affordable for all children in need across the country. A substantial gap exists between the number of transplant centres in metropolitan cities and those in smaller tier-2 and tier-3 cities, where the burden of disease is often highest. The experience at Netaji Subhash Chandra Bose Medical College demonstrates that, with strategic planning, government funding, workforce training, robust infection-control infrastructure, and collaborative partnerships, pediatric HSCT programs can be successfully established in tier-2 cities. Our journey addresses a critical implementation gap in the literature and provides a practical roadmap for expanding transplant access in India and other resource-limited settings, thereby strengthening care for children requiring hematopoietic stem cell transplantation.

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Real-World Experience with CAR-T Cell Therapy in Lymphoma: Insights from a Tertiary Care Centre in South India

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Background:

Chimeric antigen receptor (CAR)-T cell therapy has transformed the management of relapsed/refractory (R/R) B-cell lymphomas. However, real-world data from India remain limited.

Objective:

To evaluate the efficacy, safety, and early survival outcomes of CAR-T cell therapy in patients with R/R B-cell lymphoma treated at a tertiary care centre in South India.

Method:

We conducted a retrospective observational study of consecutive patients with R/R B-cell lymphoma who received commercial CAR-T cell therapy at our centre. Demographic, disease, treatment, toxicity, response, and survival data were extracted from medical records. Responses were assessed using the Lugano criteria, while cytokine release syndrome (CRS) and immune effector cell-associated neurotoxicity syndrome (ICANS) were graded according to ASTCT consensus criteria.

Results:

Thirteen patients with relapsed/refractory B-cell lymphoma underwent CAR-T cell therapy at our centre. The median age was 53 years (range, 43–71 years), and 10 (76.9%) were male. Diffuse large B-cell lymphoma was the predominant indication (8/13, 61.5%), followed by follicular lymphoma (3/13, 23.1%) and transformed follicular lymphoma (2/13, 15.4%). NexCAR19 was administered to 10 (76.9%) patients and Qartemi to 3 (23.1%). Bridging therapy was required in 10 (76.9%) patients, while two (15.4%) required intensive care support. Among nine patients with evaluable day-30 PET-CT, the overall response rate was 88.9%, comprising complete metabolic response in three (33.3%) and partial metabolic response in five (55.6%) patients; one patient (11.1%) had progressive disease. Cytokine release syndrome occurred in nine (69.2%) patients and was limited to grade 1 (23.1%) and grade 2 (46.2%), with no grade ≥3 CRS or ICANS. At the time of analysis, one patient had relapsed and four patients had died.

Conclusion:

Our early real-world experience demonstrates that commercial CAR-T cell therapy is feasible and safe in a tertiary care centre in South India, with high early response rates and manageable toxicity. The absence of grade ≥3 CRS and ICANS is encouraging, although longer follow-up in a larger cohort is required to define the durability of response and long-term survival outcomes.

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O-19

CAR-T cell therapy: A single center experience

Presenting Author: Dr Ganga R

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Background:

Chimeric antigen receptor T-cell (CAR-T) therapy has emerged as a promising treatment for patients with relapsed/refractory (R/R) haematological malignancies who have failed multiple lines of therapy. More recently, adoptive cellular therapies are also being explored in advanced solid tumours. However, real-world data from our country is limited. We present our institutional experience evaluating the safety and clinical outcomes of CAR-T therapy across various haematological malignancies and selected solid tumours.

Objective:

To evaluate the clinical characteristics, treatment-related toxicities, and outcomes of patients undergoing CAR-T cell therapy at a tertiary care centre.

Method:

A retrospective analysis was performed on 19 consecutive patients who received CAR-T therapy between December 2022 and July 2026. Patient demographics, underlying diagnosis, prior therapies, bridging treatment, lymphodepletion regimen, CAR-T construct, treatment-related toxicities including cytokine release syndrome (CRS) and immune effector cell-associated neurotoxicity syndrome (ICANS), disease response, complications, maintenance therapy, and survival outcomes were analysed.

Results:

Nineteen patients with relapsed/refractory malignancies underwent CAR-T therapy, including B-cell acute lymphoblastic leukaemia (n=4), diffuse large B-cell lymphoma/high-grade B-cell lymphoma and primary CNS lymphoma (n=8), multiple myeloma (n=5), Burkitt lymphoma (n=1), and metastatic renal cell carcinoma treated with TCR-T therapy (n=1). Most patients had received multiple prior lines of chemotherapy and targeted therapies before CAR-T infusion. The majority underwent fludarabine and cyclophosphamide-based lymphodepletion, while selected patients also received bendamustine and/or rituximab. Dual-target CD19/CD22 CAR-T therapy was the most commonly administered product, followed by CD19/BCMA, BCMA, CD19 alone, CD19/CD20/CD22, and TCR-T therapy.

Cytokine release syndrome occurred in several patients, predominantly Grade 1–2, while Grade 3–4 CRS was observed in a small proportion and was successfully managed with tocilizumab, corticosteroids, anakinra, and supportive care. ICANS was uncommon and occurred in one patient with fatal neurotoxicity. Infectious complications, prolonged cytopenia's, coagulopathy, hypofibrinogenemia, and viral reactivation were observed in selected patients. The majority of evaluable patients achieved complete metabolic, molecular, or marrow remission following CAR-T therapy, whereas a minority developed persistent disease, disease progression, or CAR-T refractory disease. In addition, the patient with metastatic renal cell carcinoma and localised colorectal carcinoma was treated with TCR-T therapy achieved a metabolic response, demonstrating the potential applicability of adoptive cellular immunotherapy beyond haematological malignancies. Mortality was primarily related to progressive disease, severe infectious complications, transplant-related complications, or severe CRS/ICANS. Many unknown or lesser-known complications like NK cell clonal expansion causing cytopenia's, pseudo tumour progression were all seen.

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Conclusion:

CAR-T cell therapy demonstrated encouraging clinical activity in heavily pre-treated patients with relapsed/refractory haematological malignancies, with acceptable and manageable toxicity in most patients. Solid tumours also respond to TCR gene therapy.

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Spectrum of plasma cell dyscrasias: clinicopathological study in a tertiary care hospital

Presenting Author: Dr. Jay Y. Sheth

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Background:

Plasma cell dyscrasias by definition include a wide range of disorders represented by excessive proliferation of a single clone of plasma cells producing entire immunoglobulins, immunoglobulin fragments, heavy chains or light chains.

Materials and Method:

This study was carried out in a tertiary care hospital (Bombay Hospital, Mumbai) prospectively for a period of nine months from December 2024 to August 2025. All the cases diagnosed with plasma cell dyscrasias were selected. Data from haematological, biochemical and radiological investigations were collected. For evaluation of each case of Multiple Myeloma, revised International Myeloma Working Group criteria were applied.

Results:

30 patients were diagnosed during the study period. Multiple myeloma was the most frequent diagnosis (17/30, 56.67%). An overall male-to-female ratio was 2.33:1. Among all 30 patients, the most common clinical feature was renal impairment (66.67%), followed by anaemia and generalised weakness (56.67% each). Hypercalcaemia was present in 10/30 patients (33.33%). All 17 patients with multiple myeloma demonstrated an M-band on serum protein electrophoresis.

Conclusion:

This study demonstrates the heterogeneous clinicopathological spectrum of plasma cell dyscrasias encountered in a tertiary care setting, with Multiple Myeloma being the predominant diagnosis. Renal impairment, anaemia, generalised weakness and skeletal manifestations were frequent presenting features. An integrated approach incorporating clinical, haematological, biochemical, electrophoretic, radiological and pathological evaluation is essential for accurate diagnosis and classification of plasma cell dyscrasias.

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Frailty in Newly Diagnosed Multiple Myeloma: Prevalence, Determinants and Prognostic Impact in a Real-World Indian Cohort Using the Simplified IMWG Frailty Score.

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Institution: 1. Seth G.S. Medical College and K.E.M. Hospital, Mumbai
2. Tata Memorial Hospital, Mumbai

Introduction:

Frailty is increasingly recognized as a determinant of treatment tolerance and outcomes in multiple myeloma (MM). Its applicability in India warrants evaluation given younger age at diagnosis, lower life expectancy, and higher comorbidity burden than Western populations.

Objective:

To evaluate the prevalence, determinants, and prognostic impact of frailty using the simplified International Myeloma Working Group (sIMWG) frailty score in a real-world Indian cohort.

Method:

This single-centre observational study included consecutive newly diagnosed MM patients aged ≥60 years (extended from ≥65 years given India's younger presentation age and high comorbidity burden) from January 2022-December 2025. Patients with prior treatment or missing data were excluded. Frailty was assessed using the sIMWG frailty score (age, Charlson Comorbidity Index, ECOG performance status); scores 0-1 were non-frail and ≥2 frail. Baseline characteristics, treatment outcomes, and survival were analyzed.

Results:

A total of 125 patients with a median age of 66 (range: 60-85) were evaluated for frailty. The cohort included 86 (68.3%) male patients, with CRAB features in the form of hypercalcemia in 14 (11.2%), renal dysfunction in 42 (33.6%), anemia in 67 (53.6%) and bone lesions in 108 (86.4%) patients. Six (4.8%) and 80 (64%) patients had R-ISS stage I and II respectively, while 39 (31.2%) had R-ISS stage III. High-risk cytogenetics as per IMWG/IMS 2025 was seen in 18 (14.3%) patients.

As per the sIMWG frailty score, 78 (62.8%) were classified as frail. The factors responsible for frailty were predominantly poor PS (ECOG-PS ≥2) in 58 (46.2%) and a CCI score ≥2 in 40 (32%); mainly due to high comorbidities [diabetes mellitus (36.8%), ischemic heart disease (26.3%), and chronic kidney disease (18.4%)]. Of note, age 76-80 years (sIMWG score of 1) contributed to frailty in 7 (5.6%) patients, and age >80 years (sIMWG score of 2) was noted only in 2 (1.6%).

VRd was the most common regimen (62.4%), followed by VCd (26.4%); 5.9% received doublet therapy. Frail patients had more serious infections (37.3% vs 17.4%, p=0.020) and all four ICU admissions occurred in this group. Grade ≥3 hematologic toxicity occurred in 34.2% of frail patients. Among 88 evaluable patients, ≥VGPR rates were lower in frail patients (65.4% vs 75%, p=NS).

At a median follow-up of 25 months, the 18-month PFS was 84.6% vs 59% in non-frail and frail patients (^{2}=5.021, p=0.025). On univariate analysis, frailty, anemia, hypercalcemia, and eGFR predicted inferior PFS; frailty was the sole independent predictor of PFS (HR 1.985, 95% CI 1.020-3.862, p=0.044). Twenty-six deaths occurred, with 21 (80.7%) in frail patients (^{2}=4.721, p=0.03).

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Conclusion:

Nearly half of all frail patients were aged ≤65 years, suggesting Indian myeloma patients accumulate frailty through mechanisms beyond age. Frailty independently predicted inferior PFS and OS, emphasizing the need for routine frailty assessment and frailty-directed therapeutic approaches.

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O-22

Attrition rates in multiple myeloma: a report from Indian patients at a Tertiary Cancer Centre

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Background:

Because of its relapsing-remitting nature, patients with multiple myeloma [1,2] require extended therapy with chemotherapy, novel agents, autologous stem cell transplants (ASCT), monoclonal antibodies and/or immune therapies for optimal outcomes. Attrition during treatment can negatively impact long-term outcomes and factors influencing the same may be important clues to achieve optimal patient outcomes.

Objective:

This study aims to examine attrition rates and the factors influencing them.

Method:

We analysed 244 consecutive newly diagnosed patients of multiple myeloma from January 2017 to December 2017 registered at Tata Memorial Hospital, Mumbai. The patient factors such as age, comorbidities, clinical and laboratory evaluations were recorded along with the lines of therapy (LOTs) received, best response and use of ASCT. Attrition was defined as failure to receive a subsequent line of therapy despite progression of the disease or death from disease according to similar stringent definitions used in similar studies elsewhere. Factors influencing attrition were analyzed using the chi-square test for categorical variables, and either the t-test or logistic regression for continuous variables. An odds ratio greater than 1 and a p-value less than 0.05 were considered as statistically significant.

Results:

The median age of study cohort was 57.11 years, with 66.4% being male. CRAB (Hypercalcemia, renal dysfunction, anemia, bony lesion) features were noted in most patients with following frequencies: 42 (17.3%) patients with hypercalcemia, 70 (28.9%)-renal dysfunction, 15 (6.2%) of the patients received dialysis,139 (56.9%) patients had anaemia at presentation and 213 (87.2%) patients had bony lesions. R-ISS was available for 199 patients with 19 (9.5%), 128 (64.3%) and 45 (22.6%) of them having R-ISS I, II, and III respectively.

26.2% patients had high-risk cytogenetics (as per the IMWG consensus criteria 2016) at presentation. The most common 1st line therapy given was bortezomib, cyclophosphamide and dexamethasone combination (77.5% of patients). 8.2% of patients had stem cell transplants done & 22.63% patients had received maintenance post

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first-line therapy. The median progression free survival (PFS) and overall survival (OS) was 3.337 years and 5.043 (Range=4.143–5.943) years respectively.The median OS in the attrited group was 2.87 years (Range 2.37–3.37 years) while those with attrition to second line therapy showed even poorer median OS of 1.08 years (Range 0.67–1.49 years). The attrition rates for individual lines of therapy were 28.6% for the second LOT, 34.2% for the 3rd LOT, and 45.3% for the subsequent LOTs. The overall attrition rate at the end of follow up was 62.9% (141 patients). Among the factors analysed for attrition, on univariate analysis higher age, ECOG scores of 2 or more, renal dysfunction at presentation, ISS stage 3, and not receiving ASCT showed a significantly higher likelihood of attrition. On multivariate analysis (Overall regression model significant with $p<0.001$) only having an ECOG of 2 or higher was significantly associated with higher risk of attrition.

Conclusion:

Our study shows a high proportion of patients do not receive subsequent lines of therapy in our clinical setting highlighting the importance of first-line treatment including ASCT and maintenance. Most effective therapies should be used in earlier lines so that patients can benefit from all available treatment modalities.

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O-23

Comparison of differential count in leukopenic samples by the regular and low WBC mode on an automated hematology analyzer with the manual differential count

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Co-Authors: Dr. Shanaz Khodaiji, Dr. Vidisha Mahajan

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Introduction:

Accurate leukocyte differential counts are critical in clinical decision-making, in leukopenic patients where low WBC counts challenge automated hematology analyzers. Modern analyzers incorporate specialized low WBC (LW) modes to enhance accuracy of differential counts, but their performance requires validation against normal WBC (NW) modes and manual differentials (OB).

The aim of our study was to assess the performance of the LW and NW modes of an automated hematology analyzer with OB for WBC differential counts in leukopenic samples, across WBC count categories. The manual differentials were performed by 2 independent pathologists and the mean was taken as OB.

Method:

A total of 240 leukopenic samples were analyzed.

Of these, 214 (89.2%) samples, were from oncology and 26 (10.8%) from non-oncology patients. Samples were categorized into WBC count ranges: <200/μL (n=25), 200–499/μL (n=23), 500–999/μL (n=57), 1000–1499/μL (n=62), and 1500–2000/μL (n=72).

Results:

Correlation analysis between NW, LW, and OB was performed for neutrophils, lymphocytes, monocytes, eosinophils, and basophils across total WBC count categories using Spearman correlation coefficient Figure 1 demonstrates the relationship between total leukocyte count ranges and the accuracy of LW mode. Figure 2 demonstrates the relationship between total leukocyte count ranges and the accuracy of NW mode. Each curve represents a different leukocyte subset.

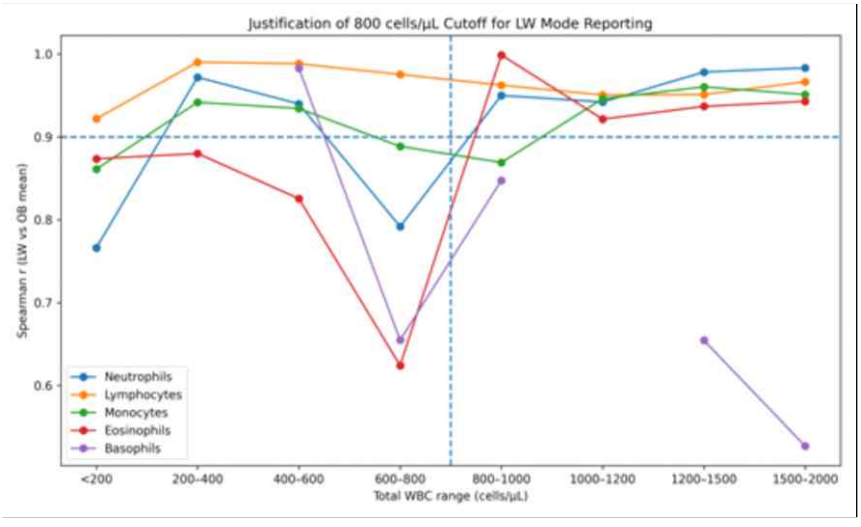

Figure 1:
Spearman correlation coefficient of LW vs OB

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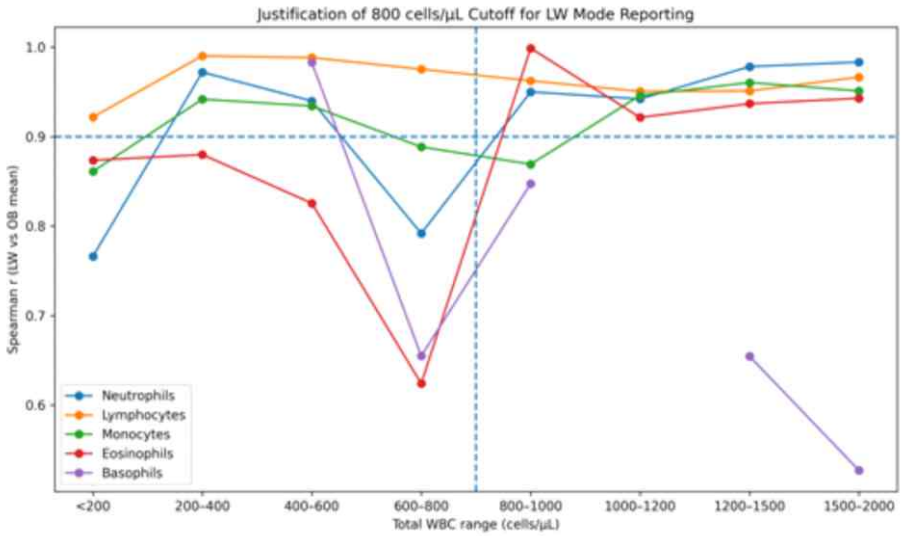

Figure 2:
Spearman correlation coefficient of NW vs OB

Interpretation:

Our study demonstrates that when total WBC counts reach 800 cells/μL, in LW, a convergence of correlation coefficients above 0.90 is observed for all major leukocyte subsets. Beyond this threshold, LW mode demonstrates stable and reliable agreement with the reference OB, and further increases in WBC do not yield substantial gains in accuracy.

In the LW mode, at total WBC counts below 600–800 cells/μL, substantial divergence in correlation coefficients is observed across leukocyte subsets, particularly for monocytes and basophils. This reflects increased analytical variability related to low cell event numbers and biological dispersion.

When a vertical reference line corresponding to the NW-derived reliability threshold (~1000 cells/μL) was applied, NW mode demonstrated acceptable agreement with the reference method only beyond this level.

Conclusion:

Based on the statistical analysis, 800 cells/μL represents the optimal operational cut-off above which LW mode differential counts may be reported without routine manual smear verification, provided no abnormal analyzer flags are present.

Compared with NW mode, LW mode demonstrates earlier stabilization and superior analytical performance in leukopenic samples, justifying its use in lower total leukocyte counts.

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Dry Tap Dilemma

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Background:

Dry tap during bone marrow aspiration presents a significant diagnostic challenge which often points to a significant underlying marrow pathology and is usually associated with marrow fibrosis, extensive neoplastic infiltration, necrosis, and marked hypercellularity, requiring bone marrow trephine biopsy for definitive diagnosis.

Objective:

To evaluate the clinicopathological spectrum of dry tap bone marrow aspirates and assess the diagnostic utility of bone marrow trephine biopsy in establishing the final diagnosis.

Method:

A retrospective observational study was conducted at a tertiary care teaching hospital. We found consecutive cases documented as dry tap during bone marrow aspiration from departmental records. For these cases, available information on clinical features, demographics, peripheral blood results, trephine biopsy morphology, immunohistochemistry, cytogenetic findings, and any additional special stains (where appropriate) were reviewed. Final diagnoses were categorized as hematological neoplasms, metastatic lesions, and non-neoplastic marrow disorders based on the WHO Classification of Haematolymphoid Tumours.

Results:

A total of 45 dry tap bone marrow aspirates were evaluated, with 29 males (64.4%) and 16 females (35.6%). Eight cases (17.8%) were pediatric, including seven cases of B-lymphoblastic leukemia/lymphoma and one case of mixed-phenotype acute leukemia. Hematological neoplasms constituted 26 cases (57.8%), with mature B-cell neoplasms being the predominant group (14/45, 31.1%), followed by acute leukemias (9/45, 20.0%) and myeloproliferative neoplasms (2/45, 4.4%). Mature B-cell neoplasms included plasma cell neoplasms, hairy cell leukemia, Burkitt lymphoma, lymphoplasmacytic lymphoma/Waldenström macroglobulinemia, B-cell non-Hodgkin lymphoma, immunoblastic/plasmablastic lymphoma, and classical Hodgkin lymphoma. The remaining cases comprised metastatic marrow involvement, myelodysplastic syndrome, hypocellular marrow, marrow necrosis, megaloblastic erythropoiesis, reactive marrow changes, and other non-neoplastic conditions. Trephine biopsy established the definitive diagnosis in aspirates that were non-contributory due to dry tap, while immunohistochemistry was indispensable for lineage assignment and subclassification of malignancies.

Conclusion:

Dry tap is an important clinicopathological finding that should lead to thorough evaluation for underlying marrow pathology. In the present study, more than half of the cases represented hematological neoplasms, with mature B-cell neoplasms and acute leukemias accounting for the majority. Pediatric dry tap cases were exclusively associated with acute leukemia. Bone marrow trephine biopsy, complemented by immunohistochemistry and ancillary investigations, remains indispensable for accurate diagnosis, disease classification, and guiding timely therapeutic decision-making.

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O-25

Repeat 3+7 versus FLAG re-induction in acute myeloid leukaemia with residual disease after standard 3+7 induction: a real-world single-centre analysis

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Background:

Approximately half of patients with acute myeloid leukaemia (AML) have residual marrow disease on the day 14/28 assessment after standard 3+7 induction. The optimal re-induction strategy is undefined: FLAG is widely preferred on the assumption of greater cytoreductive efficacy, but head-to-head data against a repeat 3+7 course are scarce, particularly from resource-constrained settings.

Aims:

To compare response, measurable residual disease (MRD) negativity, toxicity, induction mortality and overall survival (OS) between repeat 3+7 and FLAG re-induction in patients with residual disease after 3+7 induction.

Methods:

We retrospectively analysed 133 consecutive patients with newly diagnosed AML treated with 3+7 (daunorubicin–cytarabine) induction. Residual disease was defined as ≥5% blasts on the day 14/28 marrow. Groups were compared using Fisher's exact and Mann–Whitney U tests; OS was estimated by Kaplan–Meier with log-rank testing. No correction for multiplicity was applied and p values are descriptive.

Results:

Median age was 30 years (IQR 20–39) and 57.1% were male; ELN 2022 risk was favourable in 42.9%, intermediate in 33.1% and adverse in 23.3%. A day 14/28 marrow was evaluable in 127 patients, of whom 64 (50.4%) had residual disease; 55 (85.9%) received re-induction — FLAG 24, repeat 3+7 14, HiDAC 8, HiDAC–venetoclax 4 and HMA–venetoclax 5.

Comparing repeat 3+7 (n=14) with FLAG (n=24), the groups were balanced for ELN risk (p=0.843), marrow blasts (45.0% vs 70.5%, p=0.126) and performance status; baseline LDH was lower in the 3+7 group (340 vs 455 U/L, p=0.018). Composite overall response was 8/14 (57.1%) versus 14/24 (58.3%) (p=1.000) and complete remission 42.9% versus 50.0% (p=0.745). MRD negativity among evaluable patients was 2/9 (22.2%) versus 8/18 (44.4%) (p=0.406). Febrile neutropenia and documented infection occurred in 100% of both groups; composite critical-care requirement was 64.3% versus 45.8% (p=0.328) and induction mortality 28.6% versus 25.0% (p=1.000). Median OS for the whole cohort was 17.0 months (95% CI 14.9–NE) and did not differ between re-induction arms (7.4 vs 17.0 months; log-rank p=0.625); day 14/28 residual disease showed a trend toward inferior survival (p=0.079).

Conclusion:

In this young, real-world AML cohort, residual disease after 3+7 induction was common, and FLAG re-induction conferred no measurable advantage over a repeat 3+7 course in response, MRD negativity, induction mortality or survival, at the cost of a similarly high infective and critical-care burden. These hypothesis-generating findings, limited by small numbers and retrospective design, suggest that a repeat 3+7 course remains a defensible re-induction option where cost and drug availability constrain practice.

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O-26

The Cartwheel Chronicles

Presenting Author: Dr. Advait Karmarkar

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Institution: Department of Pathology, Dr. D. Y. Patil Medical College, Hospital & Research Centre, Pimpri, Pune, Maharashtra, India

Background:

Plasma cell disorders are a group of heterogeneous disorders both benign and neoplastic, that share common morphologic characteristics. Increase in plasma cells are not unique to plasma cell neoplasms, they can also be seen in various hematological disorders. In addition, the morphology of plasma cells can change following therapy and in relapsed disease, it may be difficult to distinguish true neoplastic changes from secondary plasmacytosis due to a different disease process. This case series aims to illustrate histomorphological presentations of plasma cell disorders commonly seen in routine practice and to make a distinction between plasma cell disorders and other hematological disorders with increased plasma cells, using extensive clinicopathological correlation.

Objective:

To review a series of cases with increased plasma cell population in bone marrow and to study cytomorphology correlation to differentiate between reactive and neoplastic causes of increased plasma cells.

Method:

A retrospective analysis of 26 cases presenting with increased plasma cell populations was conducted in a tertiary care centre. This multi-parametric approach, combining clinical history, cytopathology, laboratory parameters, immunophenotyping and cytogenetic analysis was used to make final diagnoses.

Results:

There was significant overlap in the diagnosis of plasma cell disorders (benign and neoplastic), as shown in the series. Plasma cells by themselves, or plasma cell morphology alone, were not enough to make a definite diagnosis. In order to differentiate accurately, clinical features, disease history, the presence of monoclonal proteins and the results of multi-parametric diagnostic testing were used to exclude non-neoplastic and secondary plasma cell elevations.

Conclusion:

Differentiating neoplastic from reactive plasma cell proliferations requires a high index of suspicion and an integrated clinicopathological approach. Diagnostic accuracy relies on correlating cytomorphological features with laboratory and clinical data to avoid misdiagnosis, ensure proper disease classification, and informed targeted therapeutic management.

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Marrow Beyond Malignancy: : A Window into Systemic Disease

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Background:

Bone marrow is a dynamic hematopoietic and immunological organ, its specialized microenvironment continuously adapts in response to systemic physiological and pathological stimuli. Even though bone marrow examination is traditionally associated with the evaluation of haematological malignancies, a diverse range of systemic non-neoplastic disorders also show characteristic morphological alterations within the marrow. Despite accounting for a substantial proportion of routine bone marrow examinations, these entities lack a standardized morphology-based interpretative framework. Rather than representing isolated disease-specific findings, many of these alterations reflect recurring biological responses to systemic insults and some may closely mimic haematological malignancies, posing significant diagnostic challenges.

Objective:

To characterize the recurring marrow response patterns observed in systemic non-neoplastic disorders, correlate these morphological alterations with their underlying biological processes, and evaluate their diagnostic significance through a morphology-based framework.

Method:

A retrospective observational study was conducted on 420 consecutive bone marrow aspirates and trephine biopsies received at a tertiary care center. Cases diagnosed as normocellular marrow without significant pathological abnormalities, haematological malignancies and metastatic involvement were excluded from the study. The remaining 130 non-neoplastic bone marrow examinations constituted the study cohort. Clinical presentation, haematological parameters, peripheral blood smear findings, ancillary investigations, bone marrow morphology, and final diagnoses were comprehensively reviewed and correlated. The observed morphological alterations were interpreted according to the predominant marrow response pattern and were subsequently subclassified based on the underlying disease process. Their clinicopathological characteristics and diagnostic implications were analysed.

Results:

Among the 420 bone marrow examinations reviewed, 130 (31.0%) represented non-neoplastic disorders. The study cohort consists of 65 males and 65 females (male: female ratio 1:1) with a mean age of 31.1 years. Despite the heterogeneity of the underlying diseases, the marrow findings consistently converged into three principal response patterns: hematopoietic responses- reflecting altered cellular production and maturation; immunoinflammatory responses- depicted by macrophage-mediated alterations and reactive immune activation; and infiltrative responses - resulting from replacement of the marrow by infectious or storage-related processes. Among these three patterns, immune-inflammatory pattern predominated (83/130, 63.8%), followed by hematopoietic adaptation (39/130, 30.0%), whereas infiltrative responses comprised the smallest subset (8/130, 6.2%). These comprehensive patterns encompassed the diverse spectrum of systemic disorders encountered in our study and provided a biologically coherent approach to morphological interpretation. Few cases were seen exhibiting overlap with suspicious haematological malignancies albeit being established as benign, emphasizing

the importance of integration of morphology with clinical findings and ancillary investigations to ensure diagnostic accuracy.

Conclusion:

Bone marrow examination acts as a window into a variety of systemic disorders through a finite and reproducible pattern of responses, reflecting their biological process. Interpreting non-neoplastic marrow morphology into these major response patterns offers a practical framework for evaluation, demonstrating that bone marrow examination extends beyond the diagnosis of haematological malignancies. However, marrow morphology represents a temporal snapshot of the evolving disease process. One should always be cautious in cases of unexplained or persistent dysplastic alterations and, where appropriate, supplement it with ancillary investigations and longitudinal follow-up to exclude emerging clonal hematopoietic disorders.

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O-29

Co-inheritance of α-Globin Gene Multiplication in β-Globin Disorders:" A Single-Centre Molecular Diagnostic Experience

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Background:

Beta-globin gene disorders display wide phenotypic heterogeneity, and this variability is frequently modulated by co-inherited alpha-globin gene defects. While alpha-globin gene deletions are known to ameliorate the severity of beta-thalassemia by reducing the alpha/beta chain imbalance, co-inheritance of alpha-globin gene multiplication has the opposite effect – it increases the relative excess of alpha chains and can worsen the clinical and haematological severity of an otherwise mild heterozygous beta-thalassemia or other beta-chain variant carrier state. Recognition of this modifier is important for accurate diagnosis and genetic counselling.

Objective:

To characterize the spectrum of β-globin abnormalities associated with co-inherited α-globin gene multiplication in a single-centre cohort and to highlight its relevance as a genetic disease modifier in β-globin disorders.

Method:

This retrospective observational study included 45 patients with co-inherited α-globin gene multiplication and β-globin abnormalities identified at a tertiary molecular diagnostic laboratory. Hematological parameters, HPLC findings and clinical details were reviewed. β-globin variants were characterized by Sanger sequencing, while α-globin copy number abnormalities were detected by Multiplex Ligation-dependent Probe Amplification (MLPA).

Result:

Among the 45 patients included, the median age at presentation was 28.5 years (range: 2–73 years), and 53.3% were female. The cohort had a mean haemoglobin concentration of 7.36 g/dL. ααα anti-3.7 multiplication emerged as the predominant α-globin abnormality (88.9%), while IVS-I-5 (G>C) was the most frequently associated β-globin mutation (55.6%). Most patients possessed five α-globin gene copies (53.3%), followed by six (33.3%) and seven (13.3%) copies.

Conclusion:

α-Globin gene multiplication is an important genetic modifier of β-globin disorders and should be considered in patients with clinical or hematological findings that are disproportionate to the primary β-globin mutation. Comprehensive molecular evaluation of both α- and β-globin genes facilitates accurate diagnosis, prognostication and appropriate genetic counselling.

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Clinical Profile, Prognostic Stratification and Treatment Outcomes of Patients with Chronic Lymphocytic Leukemia: A Single-Center Real-World Experience

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Background:

Chronic lymphocytic leukemia (CLL) is a biologically heterogeneous mature B-cell malignancy with variable clinical presentation and outcomes. Cytogenetic and molecular abnormalities, particularly TP53 aberration, IGHV mutation status, and recurrent cytogenetic abnormalities, provide important prognostic information.[1,2] We evaluated the clinical profile, prognostic characteristics, treatment patterns, and outcomes of patients with CLL in a single-center real-world cohort.

Method:

This retrospective observational study was conducted at a tertiary-care center from January 2023 to June 2026. Consecutive patients diagnosed with CLL during the study period were included. Demographic, clinical, hematological, immunophenotypic, cytogenetic/molecular, treatment, and outcome data were collected. Disease stage was assessed using Rai/Binet systems. TP53 aberration, IGHV mutational status, and cytogenetic abnormalities were evaluated where available. Treatment indications were based on contemporary iwCLL/ESMO recommendations. Treatment response, complications, disease progression, and survival outcomes were assessed.[1,3]

Result:

During the 6 year study period from January 2020 to June 2026, a total of 76 patients with CLL were included. The mean age was 64.8 ± 9.7 years, with 53 (69.7%) males. The mean hemoglobin, total leukocyte count, absolute lymphocyte count, and platelet count were 10.2 ± 2.1 g/dL, 68.4 ± 54.7 $\times 10^9$/L, 57.8 ± 48.9 $\times 10^9$/L, and 142.6 ± 67.4 $\times 10^9$/L, respectively. Lymphadenopathy (51, 67.1%) and splenomegaly (38, 50.0%) were the commonest clinical findings. Advanced Rai/Binet stage (Rai III–IV/Binet C) was present in 29 (38.2%) patients. Among patients with available cytogenetic testing, del(13q), trisomy 12, del(11q), and del(17p) were detected in 25 (45.5%), 13 (23.6%), 9 (16.4%), and 6 (10.9%), respectively. TP53 aberration was identified in 9 (13.0%) patients, while 33 (47.1%) had IGHV-unmutated disease.

Treatment was initiated in 43 (56.6%) patients, most commonly for progressive marrow failure, symptomatic/progressive lymphadenopathy or splenomegaly, and constitutional symptoms. Targeted therapy was the predominant treatment approach. An overall response was achieved in 39/43 (90.7%) treated patients. During a mean follow-up of 25.8 ± 11.4 months, 12 (15.8%) patients experienced disease progression and 8 (10.5%) died.

Conclusion:

This single-center real-world cohort demonstrates significant clinical and biological heterogeneity in CLL. Advanced disease stage and adverse molecular/cytogenetic features were associated with poorer outcomes. Integration of molecular and cytogenetic assessment may facilitate individualized risk-adapted treatment and improve therapeutic decision-making in routine clinical practice.

Keywords:

CLL; TP53; IGHV; cytogenetics; prognostic factors; treatment outcomes.

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O-31

Innovative Bridge in Ph Positivity: Efficacy of Combination Therapy with Inotuzumab Ozogamicin and Ponatinib in Pediatric Philadelphia-Positive and Ph-like Acute Lymphoblastic Leukemia

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Background:

Philadelphia chromosome-positive (Ph+) and Ph-like acute lymphoblastic leukemia (ALL) represent high-risk pediatric ALL subtypes with a significant risk of relapse despite the incorporation of tyrosine kinase inhibitors (TKIs). We evaluated the efficacy and safety of inotuzumab ozogamicin (INO) combined with ponatinib as bridging therapy to HSCT in this high-risk population.

Methods:

We retrospectively analyzed children (≤18 years) who received INO with ponatinib between April 2018 and April 2026. INO was administered as a single cycle with a total dose of 1.8 mg/m^2 on Days 1, 8, and 15, with concurrent ponatinib for 21 days. Clinical characteristics, response, transplant outcomes, toxicities, and survival were analysed.

Results:

Twenty-five children received INO during the study period. The median age was 10 years (range, 1.8–19 years), and 76% were male. Twenty-three children had B-cell ALL, while one each had mixed phenotype acute leukemia and chronic myeloid leukemia in blast crisis. INO was administered for relapsed disease in 19 patients (76%), predominantly early relapse (16/19, 84%), refractory disease in four (16%), and as upfront therapy in two (8%).

Among the 23 children treated for relapsed/refractory disease, 20 (87%) achieved MRD-negative complete remission following INO and ponatinib. Of the three non-responders, one achieved MRD negativity with subsequent blinatumomab and proceeded to HSCT, while two defaulted treatments.

Overall, 21 children underwent HSCT. Haploidentical transplantation was the most common donor type (13/21, 62%), followed by matched sibling (5/21,24%) and matched unrelated donor transplantation (3/21, 14%). Peripheral blood stem cells were used in all cases. Median neutrophil engraftment occurred on Day +14 (range, +12 to +20). Acute and chronic graft-versus-host disease occurred in 57% and 24% of patients, respectively. Cytomegalovirus reactivation occurred in two patients and adenovirus infection in one.

All transplanted patients developed Grade 1 sinusoidal obstruction syndrome (SOS), which resolved with supportive care alone. Prophylactic N-acetylcysteine was administered throughout conditioning and transplantation, and no patient required defibrotide. Importantly, there was no transplant-related mortality. At a median follow-up of 12 months, 17 of 21 transplanted children (81%) remained alive. Overall survival for the entire cohort was 76% (19/25).

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Conclusion:

INO combined with ponatinib provided an effective bridge to HSCT in children with Ph+ and Ph-like ALL, achieving high rates of MRD-negative remission and encouraging post-transplant survival. Despite universal occurrence of mild SOS, prophylactic N-acetylcysteine was associated with a favorable toxicity profile, with all cases resolving conservatively and no regimen-related mortality. Longer follow-up is required to determine the durability of remission and long-term survival benefits.

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O-32

Clinical, Laboratory Profile and Early Outcomes of Pediatric Hyperleukocytosis in Acute Leukemia: Retrospective comparative analytic Study from Central India

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Background:

Hyperleukocytosis (HL) in pediatric acute leukemia is a severe oncological emergency associated with profound rheological and metabolic complications.

Objective:

To compare the clinical characteristics, baseline laboratory profiles, early complications, and survival outcomes of children presenting with and without HL.

Method:

This retrospective comparative cohort study evaluated children (aged 1 month to 14 years) newly diagnosed with acute leukemia (ALL, AML, or JMML) at a tertiary care center in Central India between January 2024 and December 2025. Patients were stratified into HL cases (n = 30; baseline total leukocyte count of at least 100,000 cells/mm^3 for ALL or 50,000 cells/mm^3 for AML/JMML) and non-HL controls (n = 35).

Results:

The median total leukocyte count was 230,570 cells/mm^3 in the HL group vs 21,430 cells/mm^3 in controls. HL patients demonstrated highly proliferative lineages (T-cell ALL: 36.7%; AML: 23.3%; JMML: 10.0%) compared to controls (B-cell ALL: 71.4%). Within 7 days of diagnosis, HL cases experienced significantly higher rates of systemic sepsis (50.0% vs 8.6%, $p < 0.001$), tumor lysis syndrome (43.3% vs 8.6%, $p = 0.001$), respiratory complications (36.7% vs 2.9%, $p < 0.001$), and acute kidney injury (26.7% vs 5.7%, $p = 0.018$). Intensive care support was disproportionately required in the HL cohort (ICU stay: 30% vs 2.9%, $p = 0.004$; mechanical ventilation: 20.0% vs 2.9%, $p = 0.041$). Early mortality (under 7 days) was restricted solely to the HL group (13.3% vs 0%, $p = 0.041$). Six-month overall mortality was also significantly higher in HL cases (36.7% vs 14.3%, $p = 0.038$).

Conclusion:

Pediatric Hyperleukocytosis is a major clinical risk factor associated with high early morbidity, elevated intensive care needs, and substantial early and intermediate-term mortality in Central India. Implementing preemptive sepsis bundles, aggressive hydration, and rapid cytoreduction are essential to improve survival in resource-limited oncology setups.

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Caught in the Overlap: Immune Mediated Thrombotic Thrombocytopenic Purpura in Systemic Lupus Erythematosus–Sjögren's Syndrome

Presenting Author: Dr. Amruta B Bagul

Co-Authors: Amol Dube[1], Ishan Verma[2], Archit Pandharipande[3], Naila Khan[3], Vinay Rautiya[4], Sanjana Nalmas[5]

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Background:

Thrombotic thrombocytopenic purpura (TTP) is a rare with an incidence of approximately 3 cases per million per year, life-threatening thrombotic microangiopathy characterized by microangiopathic hemolytic anemia (MAHA), severe thrombocytopenia, and end-organ ischemia due to severe ADAMTS13 deficiency. Its coexistence with systemic lupus erythematosus (SLE) has been reported approximately 0.5-2% cases and association with SLE–Sjögren's overlap syndrome is exceedingly rare. Early recognition is critical because delayed treatment is associated with significant morbidity and mortality.

Case report:

A 41-year-old female with a known 10-year history of SLE, on long term steroids presented with loose stools, generalized weakness, palpitations, weight loss, oral ulcers, and alopecia. Examination revealed pallor and tachycardia (150/min). Investigations showed severe anemia (Hb 5.4 g/dL), thrombocytopenia (18,000/μL), acute kidney injury, and 3.3% schistocytes on peripheral smear. Despite only mildly elevated LDH (388 U/L), low retic count (0.5%) and Normal bilirubin, MAHA was suspected. Bone marrow examination demonstrated trilineage hematopoiesis. A PLASMIC score of 6 indicated high risk for TTP. ADAMTS13 activity was <4% with positive ADAMTS13 autoantibodies, confirming acquired immune-mediated TTP. Autoimmune evaluation revealed ANA positivity (1:320), dsDNA positivity, anti-Ro52 positivity, dry eyes on Schirmer's test, and Graves' disease with elevated TRAb levels, suggesting SLE–Sjögren's overlap with autoimmune thyroid disease. During plasma exchange (PLEX), she developed acute ischemic strokes and atrial fibrillation. Initial response to five sessions of PLEX was followed by recurrent thrombocytopenia, prompting escalation with rituximab. Following four doses of rituximab, ADAMTS13 activity improved to 54%, platelet counts recovered to 198,000/μL, renal function normalized, and neurological deficits improved.

Discussion:

This case highlights secondary immune-mediated TTP occurring in the setting of SLE Sjögren's overlap syndrome with Graves' disease. Diagnostic recognition was challenging because classical biochemical evidence of hemolysis was not prominent despite the presence of schistocytes. Prompt initiation of PLEX, corticosteroids, and rituximab resulted in remission despite severe neurological complications and early refractory disease.

Conclusion:

TTP should be suspected in autoimmune disease patients presenting with thrombocytopenia and schistocytes even when biochemical markers of hemolysis are not markedly elevated. Early recognition, Peripheral blood smear, ADAMTS13-directed evaluation, prompt plasma exchange, and timely immunosuppressive therapy are crucial for achieving favorable outcomes.

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Timing matters: Predictors of outcome in severe aplastic anemia

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Background:

Previously reported Indian multicentric data shows a 61.6% overall survival in haploidentical transplants and 64% in Fanconi anemia. We analyzed our cohort for predictors of outcome in both inherited and acquired aplastic anemia.

Objective:

To evaluate predictors of transplant outcomes in children with inherited and acquired severe aplastic anemia undergoing hematopoietic stem cell transplantation.

Method:

Children with Inherited Bone Marrow Failure Syndrome (IBMFS) or acquired Severe Aplastic Anemia (SAA) undergoing HSCT were retrospectively analyzed. Survival outcomes were estimated using the Kaplan–Meier method and compared using the log-rank test.

Results:

A total of 30 transplants were performed in 29 patients: 14 with IBMFS and 16 with acquired severe aplastic anemia (ASAA). Peripheral blood stem cells were used in all cases with reduced-intensity conditioning and serotherapy. 11 patients each underwent matched related and matched unrelated donor HSCT, while 7 underwent haploidentical transplant. Among haploidentical transplants, one was T-replete and six were T-deplete.

In IBMFS cohort, median age at diagnosis was 2 years (range 0–11) and at transplant was 5 years (range 2–12). Two children were diagnosed incidentally at the time of malignancy evaluation. In ASAA cohort, median age at diagnosis and transplant was 8.5 years (ranges 3–15 and 4–10).

Overall survival for entire cohort, IBMFS group and ASAA was 86%, 100% and 75% whereas GVHD-free, relapse-free survival was 82.7%, 92.3% and 75%. HCT-CI scoring demonstrated significantly superior survival in low/intermediate-risk patients compared to high-risk(95.8% vs 40%, $p = 0.01$).All IBMFS patients were free of active infection at time of HSCT with 62.5% (10 out of 16) of ASAA undergoing transplant with ongoing infections. All deaths occurred in patients transplanted with ongoing infection (aspergillus, primary graft failure, Rickettsial ARDS)

Conclusion:

Children with inherited bone marrow failure syndromes can be safely followed until transfusion dependence, with excellent transplant outcomes. The presence of active infection at time of transplantation is the most significant predictor of poor outcome in acquired severe aplastic anemia.

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O-35

Emergency Splenectomy in Immune Thrombocytopenia Presenting with Intracranial Hemorrhage

Presenting Author: Mithun Abraham Prakash

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Background:

Intracranial hemorrhage (ICH) is the most severe bleeding complication of immune thrombocytopenia (ITP) and is associated with high mortality. Standard medical therapies, including corticosteroids, intravenous immunoglobulin (IVIG), and platelet transfusions, may be inadequate in patients with rapidly progressive neurological deterioration. The role of emergency splenectomy as a salvage strategy in this setting is unclear.

Method:

We performed a retrospective analysis of patients with ITP who presented with ICH and underwent emergency splenectomy at a tertiary care center in India between January 2008 and July 2025. Clinical characteristics, radiological findings, perioperative details, hematological responses, and long-term outcomes were analyzed. Overall survival (OS) and event-free survival (EFS) were estimated using the Kaplan–Meier method.

Results:

Twenty-seven patients (25 adults, 2 children) underwent emergency splenectomy for ITP-associated ICH. The median age was 37 years (range 7–57), and 77.8% were female. Fifteen patients (55.5%) had chronic ITP. Median platelet count at presentation was 7×10^9/L (range: 3-22 $\times 10^9$/L). Intraparenchymal hemorrhage was the most common radiological pattern (55.5%), with midline shift present in 44.4%. Only four patients (14.8%) received IVIG prior to surgery. Splenectomy resulted in hematological remission in 18 of 24 evaluable patients (75%), with a median time to platelet recovery ($>100 \times 10^9$/L) of 3 days (range: 1-30 days). Overall mortality attributable to ICH was 14.8%. At a median follow-up of 34 months, the 3-year OS and EFS were 77.8% and 61.8%, respectively.

Conclusion:

Emergency splenectomy can be a life-saving intervention in selected patients with ITP presenting with ICH, particularly when rapid neurological deterioration limits the effectiveness of medical therapy. In resource-limited settings, splenectomy remains a cost-effective option that offers rapid platelet recovery during the acute episode as well as durable remissions in a substantial proportion of patients.

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Thalidomide in pediatric thalassemia with hypersplenism and transfusion burden: a single center experience

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Background:

Hypersplenism contributes significantly to increasing transfusion requirements and disease-related morbidity in children with thalassemia. Therapeutic options are limited when splenectomy is undesirable because of young age or anticipated long-term complications. Thalidomide, through its immunomodulatory effects and ability to augment fetal hemoglobin production, has emerged as a potential therapeutic option; however, pediatric data remain limited.

Objectives:

To evaluate the efficacy and safety of thalidomide in children with thalassemia complicated by hypersplenism and persistent transfusion burden.

Method:

This retrospective observational study included 12 children with thalassemia treated with thalidomide at a tertiary pediatric hematology center. The cohort comprised 7 children with HbE/ -thalassemia, 3 with -thalassemia major, and 2 with -thalassemia intermedia. Nine patients had transfusion-dependent thalassemia (TDT), while three had non-transfusion-dependent thalassemia (NTDT), including children with autoimmune hemolytic anemia and alloimmunization. The median age was 9 years (range, 6–15 years), with a male to female ratio of 5:7. Thalidomide was administered at a fixed dose of 50 mg once daily together with aspirin 75 mg twice weekly. Treatment duration ranged from 6 to 18 months. Clinical response, spleen size, hemoglobin concentration, transfusion requirement, and adverse events were assessed.

Results:

Seven of the 12 children demonstrated a meaningful clinical response. Mean hemoglobin increased by approximately 1.5–2.0 g/dL. Median spleen size decreased from 8–10 cm below the left costal margin before treatment to 3–4 cm during follow-up. Transfusion requirements decreased substantially, with the interval between transfusions increasing by approximately 1.5–2 weeks, and two children became completely transfusion independent. Thalidomide was generally well tolerated. Two patients developed mild gastrointestinal upset that responded to conservative management. Two patients discontinued treatment because of financial constraints rather than drug-related toxicity. No peripheral neuropathy, thromboembolic events, or other serious adverse events were observed during the study period.

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Conclusion:

In our single-center experience, thalidomide was associated with improvement in hemoglobin, reduction in spleen size, and decreased transfusion burden in selected children with thalassemia complicated by hypersplenism. The treatment was generally well tolerated during mid-term follow-up. Although limited by the small sample size and retrospective design, these findings suggest that thalidomide may represent a useful therapeutic option for carefully selected pediatric patients in resource-constrained settings and support the need for prospective multicenter studies to define its long-term efficacy and safety.

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O-37

Clinico-etiological and management profile of Acquired Hemophilia A: A single centre experience from India

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Background:

Acquired hemophilia A (AHA) is a rare but potentially life-threatening autoimmune bleeding disorder caused by autoantibodies directed against factor VIII, typically presenting as sudden, unprovoked bleeding in individuals without a personal or family history. Reported mortality ranges from 15–42%, largely attributable to diagnostic delay and bleeding complications. While large registries such as EACH2 and the UK national surveillance study have characterized its epidemiology, etiological spectrum, and treatment outcomes in Western populations, data from India remain scarce, limited to a handful of small single-centre series.

Objective:

To describe the clinical presentation, etiological associations, laboratory profile, treatment approach, and duration of response in patients with AHA managed at a single tertiary care centre in India.

Method:

We retrospectively analysed 13 patients diagnosed with AHA at our centre. Data recorded included demographics, presenting bleeding manifestation, relapse history, acute hemostatic management, immunosuppressive therapy (IST), etiological association, and laboratory parameters (APTT, FVIII activity, and Bethesda inhibitor titre). Duration of response was calculated from initiation of IST to documented relapse or last follow-up.

Results:

The cohort comprised 6 males and 7 females, aged 48–86 years (mean 67.3 ± 12.9 years). The most common presentation was soft-tissue bleeding- ecchymoses or intramuscular hematoma seen in 12/13 patients, with hematuria in 3/13 (isolated or combined). All patients had a markedly prolonged APTT (range 45.8s to >400s) with failure to correct on incubation with normal plasma, and severely reduced FVIII activity (<7% in all). Bethesda inhibitor titres ranged from 3.94 to 1110.4 BU; a high titre (>5 BU) was seen in 10/13 (77%), and a low titre in 3/13 (23%). Etiological evaluation identified idiopathic disease in 6/13 (46%), an underlying autoimmune association in 5/13 (38%)- pemphigus vulgaris, undifferentiated connective tissue disease, rheumatoid arthritis, and isolated ANA positivity, a co-existing malignancy (transitional cell urothelial carcinoma) in 1/13 (8%), and an atopic association (bronchial asthma) in 1/13 (8%).

For acute bleed control, FFP was used in 11/13 patients, recombinant activated factor VII in 4/13, and activated prothrombin complex concentrate in 1/13. All patients received corticosteroids as first-line IST; rituximab was added in 7/13 (54%), cyclophosphamide in 3/13, and cyclosporine and azathioprine in 1 patient each. Relapse occurred in 5/13 patients (38%), including one patient with 3 documented relapses. Duration of response was documented in 10/13 patients, ranging from 3 to 30 months (median ~1 year) before relapse or last follow-up. Notably, patients with a high Bethesda titre had a substantially higher relapse rate than those with a low titre (50%

vs. 0%), and a lower FVIII activity at diagnosis was also associated with relapse, suggesting that inhibitor strength and residual factor activity may have prognostic value in this cohort.

Conclusion:

This single-centre Indian series demonstrates a clinico-etiological spectrum of AHA broadly comparable to Western registries, with idiopathic disease predominating (46%, closely mirroring the EACH2 registry) followed by autoimmune associations. Bethesda titre and FVIII activity at diagnosis can have a potential role in the prognosis. In our experience, rituximab-based regimens resulted in more durable remissions than other immunosuppressive approaches, supporting its role as a preferred agent in AHA management.

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O-38

Clinical, microbiological profile and outcomes of febrile neutropenia in acute leukemia patients undergoing induction therapy in a tertiary care centre from North India

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Background:

Febrile neutropenia is a near-universal complication of induction chemotherapy in acute leukaemia and is the leading driver of treatment-related morbidity and mortality. The mechanism is twofold: cytotoxic-induced myelosuppression eliminates the neutrophil response, while mucosal barrier disruption allows translocation of endogenous flora. In Indian tertiary care settings this risk is compounded by Gram-negative predominance and a high burden of antimicrobial resistance, including extended-spectrum beta-lactamase (ESBL)-producing Enterobacteriaceae and carbapenem-resistant organisms.

Objective:

To determine the incidence of febrile neutropenia during induction chemotherapy; to identify the clinical focus and bacterial pathogen spectrum with antibiotic susceptibility profiles; to evaluate age and risk stratification as risk factors; and to assess the impact of infection on chemotherapy delivery and end-of-induction outcome.

Method:

A prospective observational study was conducted at the Department of Clinical Haematology, MGMCH, Jaipur. Sixty-eight consecutive patients with newly diagnosed acute leukaemia (ALL 51, AML 12, APML 5) undergoing induction chemotherapy were enrolled for one year. At the first febrile episode (temperature $\geq$ 38.3 °C or $\geq$ 38.0 °C sustained for $\geq$ 1 h with absolute neutrophil count $<$ 500/μL), peripheral blood, PICC-line, and urine cultures were sent along with site-directed investigations. Baseline demographics, haematological parameters, risk stratification, comorbidities, and time from symptom onset to chemotherapy initiation were recorded on a structured proforma. Endpoints included culture positivity, organism spectrum, antibiotic escalation, antifungal prophylaxis, chemotherapy dose modification, remission status, MRD, and induction mortality. Categorical associations were assessed with Fisher's exact test; continuous variables with the Mann-Whitney U test; multivariable logistic regression was used for independent predictors of escalation and chemotherapy modification ($p < 0.05$ significance threshold).

Results:

The cohort comprised 42 males and 26 females; median age 13 years (IQR 5–32). Febrile neutropenia occurred in 61/68 patients (89.7%; 95% CI 80.2–94.9%). Among these 61, at least one bacterial culture was positive in 8 patients (13.1%; 95% CI 6.8–23.8%). Nine isolates were identified: 7 (77.8%) Gram-negative — Escherichia coli (n=3), Aeromonas species (n=2, including one A. hydrophila), Acinetobacter baumannii (n=1), Pseudomonas species (n=1) — and 2 Gram-positive (coagulase-negative Staphylococcus, Staphylococcus haemolyticus; both AML). Antibiotic escalation was required in 21/61 (34.4%).Escalation was significantly associated with blood culture positivity (OR 10.82, p=0.029), documented clinical focus (OR 5.94, p=0.003), respiratory involvement (OR 24.00, p<0.001), and oxygen requirement (p=0.0008). Multivariable analysis identified culture positivity (adjusted OR 6.08, p=0.043) and fever at diagnosis (adjusted OR 3.97, p=0.036) as independent predictors;

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age and risk stratification were not significant. Chemotherapy dose modification occurred in 13/68 (19.1%) and was independently predicted by oxygen requirement (adjusted OR 16.15, p=0.008). Complete remission was achieved in 64/68 (94.1%); MRD was negative in 54/68(79.4%). Induction mortality was 2.9% (2/68); both deaths were culture-negative patients with severe sepsis progressing to multi-organ failure.

Conclusion:

Febrile neutropenia during acute leukaemia induction is near-universal (89.7%) at our centre. The pathogen spectrum is Gram-negative dominant (77.8%), consistent with Indian tertiary care benchmarks. Both fatal cases were culture-negative, confirming that microbiological negativity does not exclude life-threatening sepsis; early empirical broad-spectrum escalation remains essential. Antibiotic escalation is a meaningful clinical severity marker, strongly linked to respiratory support requirement and chemotherapy dose compromise. A complete remission rate of 94.1% with induction mortality of 2.9% compares favourably with published Indian data. Institution-specific surveillance is essential to keep empirical antibiotic policy aligned with the evolving local resistance landscape.

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Poster Papers

P-1

HLH-Spectrum Hyperinflammation During Induction Therapy for Acute Monocytic Leukemia: A Stereotyped Syndrome of Persistent Fever, Conjugated Hyperbilirubinemia, Platelet Transfusion Refractoriness and Dramatic Corticosteroid Responsiveness

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Background:

Secondary hemophagocytic lymphohistiocytosis (HLH) is an uncommon but potentially fatal hyperinflammatory syndrome complicating acute myeloid leukemia (AML). Recognition during induction chemotherapy is particularly difficult because persistent fever, worsening cytopenias and hepatic dysfunction frequently mimic neutropenic sepsis, drug toxicity or refractory leukemia. Monocytic AML is increasingly recognized to possess a cytokine-rich inflammatory milieu predisposing to macrophage activation.

Objective:

To describe two patients with acute monocytic leukemia who developed a remarkably similar HLH-spectrum hyperinflammatory syndrome during induction therapy and to highlight the clinical clues leading to timely diagnosis and successful treatment.

Method:

Clinical characteristics, laboratory parameters, microbiological investigations, bone marrow morphology, treatment and outcomes of two AML-M4 patients developing persistent inflammatory syndromes during induction chemotherapy were retrospectively reviewed.

Results:

A 30-year-old woman with FLT3-ITD/NPM1-mutated AML treated with azacitidine-venetoclax and a 42-year-old man with bZIP CEBPA-mutated AML treated with standard 3+7 induction developed an almost identical clinical syndrome characterized by persistent fever despite appropriate antimicrobial therapy, severe platelet transfusion refractoriness, progressive predominantly conjugated hyperbilirubinemia (peak bilirubin 14 mg/dL and 8 mg/dL), elevated ferritin (6692 ng/mL and 1992 ng/mL), relatively preserved fibrinogen (208 mg/dL and 278 mg/dL) and normal triglyceride levels. Extensive infectious evaluation was unrevealing. Day-15 bone marrow examination demonstrated clearance of leukemic blasts with marked histiocytic proliferation; one patient showed overt hemophagocytosis while the other demonstrated prominent histiocytic activation with plasmacytosis. Dexamethasone initiated after marrow assessment resulted in complete defervescence within 24–48 hours, rapid platelet recovery without further transfusion support, normalization of bilirubin and prompt clinical recovery in both patients. One patient subsequently underwent matched sibling donor hematopoietic stem cell transplantation and remains flow cytometric and molecular MRD negative, while the second continues consolidation therapy in morphological remission.

Conclusion:

Persistent fever, conjugated hyperbilirubinemia and platelet transfusion refractoriness during induction therapy for AML with monocytic differentiation may represent an under-recognized HLH-spectrum hyperinflammatory

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syndrome even in the absence of classical HLH-2004 criteria. Mid-induction bone marrow examination may provide a crucial diagnostic clue by demonstrating clearance of leukemia with macrophage activation. Early recognition and timely corticosteroid therapy can rapidly reverse this inflammatory syndrome and facilitate successful continuation of curative AML-directed treatment.

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P-2

A phase II study to evaluate the role of Pomalidomide in maintaining treatment-free remission post TKI discontinuation in patients with chronic myeloid leukemia-chronic phase

Presenting Author: Dr Bikash Sourav[1]

Co-Authors: Manju Sengar[1], Alok Shetty[1], Lingaraj Nayak[1], Bhausaheb Bagal[1], Thomas Eipe[1], Prashant Tembhare[1], Avinash Bonda[1], Neha Sharma[1], Santhosh Devadas[2], Dhanlaxmi Shetty[1], P.G.Subramanian[1], Anant Gokarn[1], Sachin Punatar[1], Navin Khattry[1], Smruti Mokal[1], Hasmukh Jain[1*]

Institution: 1. Tata Memorial Centre, Affiliated to Homi Bhabha National Institute, Mumbai
2. Ramaiah Institute of Oncosciences, Ramaiah Memorial Hospital, Bangalore

Background:

Tyrosine kinase inhibitors (TKIs) have significantly improved outcomes in patients with chronic-phase chronic myeloid leukemia (CML-CP). Treatment-free remission (TFR) is the new goal for CML patients who achieve a durable deep molecular response (DMR). Immune-mediated control, particularly through T-cell and NK cell activity, appears to contribute to maintaining TFR and may be enhanced by pomalidomide. We hypothesize that a short course of pomalidomide in could improve relapse-free survival following TKI discontinuation.

Method:

Adults (≥18 years) with CML-CP who had received TKI therapy for at least 3 years, maintained MR4 for a minimum of 2 years, and had no warning or failure responses beyond 6 months of TKI initiation were eligible. During the intervention phase, imatinib was reduced to 200mg daily for one month and stopped while pomalidomide was initiated. Pomalidomide 2mg was administered on alternate days for 21 days/ 28-day cycle for six months. The primary endpoint was 12-month molecular relapse-free survival (mRFS).

Results:

This multi-center Phase II study (CTRI/2020/10/028549), was initiated in May 2022. Among the first 82/110 evaluable patients who completed follow-up, the median age was 40.5 years (range, 20–64), and 72% were male. The 12-month mRFS was 43.7% (95%CI, 32.6–54.3). Forty-six patients (56%) lost MMR, all were restarted on imatinib and regained MMR; 44 subsequently achieved MR4.0. The median time to regain MMR and MR4.0 was 3.5 months (95%CI, 2.9–4.1) and 5.6 months (95%CI, 4.2–7.0), respectively. Three patients discontinued treatment due to grade 3 myalgia, grade 3 skin rash, and grade 4 myocardial infarction. No thrombotic events or deaths occurred. QOL assessment showed significant improvements ($P<0.05$) in nausea/vomiting and global health status following TKI discontinuation.

Conclusion:

Pomalidomide was safe and did not result in higher rates of TKI withdrawal syndrome. Final analysis will establish its role in maintaining treatment-free remission.

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A Storm in the Blood: Unravelling the Cause of Hyperleukocytosis

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Background:

Leucocytosis in an elderly patient with multisystem complaints can present a diagnostic challenge, as the initial symptoms often point towards metabolic and cardiac disease rather than an underlying primary haematological malignancy. We report a case in which leucocytosis, initially investigated as a suspected cardiac event, was diagnosed as acute myeloid leukaemia (AML) with a distinctive immunophenotypic and molecular profile.

Case Description:

A 68-year-old female with a 2-month history of diabetes mellitus presented with chest pain, profuse sweating, backache, and epigastric abdominal pain. Contrast-enhanced CT scan of the abdomen revealed mild hepatomegaly with fatty liver, findings suggestive of acute calculous cholecystitis, a prominent common bile duct, minimal ascites, and bilateral minimal pleural effusion. Initial laboratory investigations demonstrated mildly elevated troponin and CPK-MB. Complete blood counts showed anemia (Hb 8 g/dL), leucocytosis (WBC 184.1 $\times 10^3/\mu L$) with thrombocytopenia (platelets 22 $\times 10^3/\mu L$). Peripheral smear findings were suggestive of 95% blasts, which were moderate to large in size, with round to oval nuclei, coarse chromatin, occasional nucleoli, and scant amount of cytoplasm. Few blasts showed presence of Auer rods.

Immunophenotyping on peripheral blood showed a blast population (91% of gated events) which were positive for cytoplasmic MPO, CD13, CD33, CD11b, CD16, CD38, CD123, CD58, and CD48, with negative CD34, HLA-DR, and CD117, an immunophenotype usually implicated with Acute promyelocytic leukemia, however due to non-congruence of morphology and immunophenotyping expression being CD117 negative, it was diagnosed as Acute Myeloid Leukemia (AML).

Molecular next-generation sequencing (NGS) excluded fusion transcripts (including PML-RARA) but identified pathogenic frameshift mutations in NPM1 (W288Cfs*12, VAF 41.9%) and DNMT3A (F827Lfs*4, VAF 47.1%), as well as a TET2 frameshift variant (C1271Wfs*29, VAF 46.2%), constituting a molecular triad characteristic of NPM1-mutated AML.

Discussion:

This case demonstrates that AML in elderly, may first come to attention through vague, unrelated complaints of chest pain, biliary-type abdominal discomfort, and general malaise rather than through any overt haematological symptom, with leucocytosis being an incidental finding on routine blood work. The concurrent NPM1, DNMT3A, and TET2 mutations are significant, while DNMT3A and TET2 are classically regarded as early mutations associated with clonal haematopoiesis and myelodysplastic change; their co-occurrence with NPM1 raises the possibility that this patient harboured an undiagnosed MDS-like clonal process and subsequently progressed to overt AML over time. This supports that AML may have a mutational profile that evolved through secondary leukemic transformation from a previously unrecognised pre-leukemic or dysplastic clone, rather than arising as a true de novo leukemia in elderly patients.

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Conclusion:

This case emphasises the need to integrate morphological assessment, immunophenotyping, and comprehensive molecular profiling in the evaluation of unexplained leucocytosis. The simultaneous presence of mutations of DNMT3A, TET2 and NPM1 is consistent with the multistep process of leukemogenesis, where DNMT3A and TET2 are early mutations involved in the development of clonal haematopoiesis, and NPM1 is a late transformation event in the evolution of AML. Although antecedent MDS was not documented in this patient, the observed mutational pattern suggests the presence of an occult pre-leukemic clone. This study highlights the importance of next-generation sequencing (NGS) for disease clonal evolution, prognosis and therapeutic decisions.

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Daratumumab Based Quadruplet Therapy in Newly Diagnosed Multiple Myeloma: Outcomes from a Tertiary Care Centre in India

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Institution: 1. Seth GS Medical College & KEM Hospital, Mumbai,
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Background:

Daratumumab based quadruplet regimens have emerged as the new standard of care in first-line treatment of NDMM patients. Landmark trials have established the superiority of the quadruplet regimen with greater depth of response in comparison to the triplet combination. However, data from the Indian setting where higher rates of infections are seen remains limited.

Objective:

This study reports efficacy and safety profile of daratumumab based quadruplet therapy used as frontline treatment of Indian NDMM patients.

Method:

Electronic medical records of a single tertiary centre were reviewed. All patients receiving at least one complete cycle of daratumumab based quadruplet induction regimens between April 2024 and May 2026 were included. Demographic details, baseline characteristics, RISS, high risk cytogenetics as per IMS-IMWG CGS 2025, type of induction regimen, duration of treatment, depth of response, MRD status and adverse effects as per CTCAE v5.0 criteria were recorded.

Results:

A total of 32 patients were included. Median age was 55 years (range: 27-85) of which 21(62.5%) were male. At presentation, 24 patients had anemia (75%), 10 had renal dysfunction (31.3%), 4 had hypercalcemia (12.5%) and 27 (84.4%) had lytic bone lesions. Sixteen patients were RISS III (50%), 15 were RISS II (46.9%) and 1 was RISS I (3.1%). High risk cytogenetics as defined by IMS-IMWG CGS 2025 was present in 12 (37.5%).

The predominant quadruplet regimen was Dara-VRD (23,71.9%) followed by Dara-VTD (3, 9.4%) and Dara-VCD (4, 12.5%). Daratumumab was administered intravenously in 27 patients (84.4%) while 5 patients received subcutaneously (15.6%). Median cycles of therapy was 5 (range: 1-15). Induction therapy is ongoing in 21 patients (65.6%). Post Induction, 5 underwent ASCT (15.6%) and 6 went on maintenance therapy (18.8%). Out of 32, response was evaluable in 26 patients; 15 had CR (57.7%) and 8 had VGPR (30.7%) as per the IMWG response criteria. ORR (≥PR) was 96.1%. Of 23 patients achieving ≥ VGPR, 19 (82.6%) achieved ≥ VGPR at the end of 4 cycles indicating early deep response. At the end of 8 cycles, 12 patients underwent MRD assessment (37.5%). Seven (58.3%) were MRD positive and 5 (41.7%) were MRD negative.

No treatment discontinuation was observed. Infusion reaction to daratumumab was seen in 3 patients (9.4%). Grade ≥3 haematologic toxicities included neutropenia in 13 (40.6%), anaemia in 7 (21.9%) and

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thrombocytopenia in 3 (9.4%). Infections occurred in 15 patients (46.9%); 6 (18.8%) required hospitalisation. At a median follow up of 6.5 months PFS and OS data remains immature.

Conclusion:

Daratumumab based quadruplet regimens in treatment of NDMM patients displays efficacy in our setting with the majority achieving ≥ VGPR while also demonstrating early deep response and MRD negativity. Toxicity was manageable with neutropenia and infections as predominant adverse events and no treatment discontinuation. Longer follow up is needed for PFS and OS outcomes.

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P-5

Acute leukemia during pregnancy: a single-center retrospective cohort study of maternal & fetal outcomes in India

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Background:

Acute leukemia during pregnancy is a rare life-threatening condition that poses significant clinical and ethical challenges. The treatment involves cytotoxic chemotherapy which is inherently fetotoxic. Most cases require balancing maternal survival against fetal safety and decisions regarding chemotherapy initiation and delivery planning must be individualized. Evidence guiding management is limited, particularly from low- and middle-income countries (LMICs). We evaluated clinical characteristics, treatment approaches, and maternal and fetal outcomes of pregnant patients with acute leukemia treated at a tertiary referral center.

Method:

We conducted a retrospective single-center cohort study including consecutive patients diagnosed with acute leukemia during pregnancy between January 2017 and December 2025. Clinical records were reviewed for demographic characteristics, leukemia subtype, pregnancy management, treatment received, and maternal and fetal outcomes.

Results:

Twenty pregnant patients with acute leukemia were identified, including 7 (35%) with acute myeloid leukemia (AML) and 13 (65%) with acute lymphoblastic leukemia (ALL). The median (IQR) age was 27 (23–29.5) years and the median gestational age at diagnosis was 25 (10.5–34.5) weeks; 30%, 40%, and 30% of patients were diagnosed during the first, second, and third trimesters, respectively. Seven patients underwent therapeutic termination of pregnancy, whereas 13 continued their pregnancies. Two pregnancies resulted in intrauterine fetal demise. Among the remaining 11 pregnancies, three resulted in preterm cesarean deliveries and eight in term vaginal deliveries. Ten live-born infants remained healthy on follow-up, with no congenital malformations or treatment-related teratogenic effects observed. Six AML patients received standard 7+3 induction chemotherapy and one received azacitidine-venetoclax, while all ALL patients received modified Berlin–Frankfurt–Münster (BFM)-based induction chemotherapy. Sixteen of twenty patients (80%) achieved complete remission, while four patients experienced induction mortality. After a median follow-up of 2 years, three patients were lost to follow-up; among the remaining patients, one died during follow-up and two experienced disease relapse.

Conclusion:

Gestational age at diagnosis was the principal determinant of therapeutic strategy and fetal outcome. Favorable maternal remission rates and encouraging fetal outcomes were achieved through individualized multidisciplinary management, with no congenital anomalies observed among live-born infants. These findings support the timely administration of standard leukemia-directed therapy whenever feasible while tailoring obstetric management according to gestational age and maternal condition.

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P-6

Short term outcomes of Flu/Cy/Mel + TBI Conditioning in Haplo-Identical Hematopoietic Stem Cell Transplantation

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Background:

Haploidentical hematopoietic stem cell transplantation (haplo-HSCT) has emerged as an established alternative donor transplant option for patients with high-risk hematological malignancies lacking a matched related or unrelated donor. The Flu/Cy/Mel-TBI conditioning regimen is increasingly being utilized to achieve optimal disease control and engraftment; however, published data regarding its early clinical outcomes remain limited. We evaluated the short-term outcomes of patients undergoing haploidentical HSCT using this conditioning regimen at our center.

Objective:

To evaluate engraftment, transplant-related outcomes, and one-year overall survival following haploidentical HSCT using Flu/Cy/Mel-TBI conditioning.

Method:

A retrospective observational study was conducted at a single tertiary care center including thirteen consecutive patients who underwent haploidentical HSCT using Flu/Cy/Mel-TBI conditioning between November 2024 and May 2026. Primary endpoints included neutrophil engraftment, platelet engraftment, and one-year overall survival. Secondary endpoints included acute and chronic GVHD, infectious complications, veno-occlusive disease, transplant-associated thrombotic microangiopathy, graft failure, disease relapse, and transplant-related mortality.

Results:

Thirteen patients (median age, 46 years; range, 17–69 years) underwent haploidentical HSCT. Twelve patients were evaluable for engraftment. Median neutrophil and platelet engraftment occurred on day 15 (range, 10–19) and day 17 (range, 12–27), respectively. One patient developed acute GVHD and chronic GVHD occurred in one patient (7.7%). TA-TMA occurred in two patients (15.4%), while VOD/SOS occurred in two patients (15.4%); one patient experienced both complications. Infectious complications included febrile neutropenia, pneumonia, CMV reactivation, and herpesvirus infection. No disease relapse was observed during one-year follow-up. One-year overall survival was 69.2% (9/13; 95% CI, 38.6%–90.9%). Four patients died, including two from pulmonary complications, one from TA-TMA, and one from infectious complications associated with immune reconstitution failure

Conclusion:

Flu/Cy/Mel-TBI conditioning demonstrated reliable hematopoietic engraftment with a low incidence of acute and chronic GVHD following haploidentical HSCT. No disease relapse was observed during the first year after transplantation. Infectious complications remained the leading cause of transplant-related mortality. In the study cohort, one-year overall survival of 69.2% (95% CI, 38.6%–90.9%) was encouraging. Larger prospective studies with longer follow-up are warranted to validate these findings.

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P-7

Multimodal Salvage Therapy for Severe Graft-versus-Host Disease: A Single-Centre Experience

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Background:

Severe graft versus host disease (GVHD) can profoundly alter the clinical trajectory following allogenic hematopoietic stem cell transplantation (allo-HSCT), with substantial implications on morbidity and mortality. Grade III–IV acute GVHD occurs in approximately 15% of allo-HSCT recipients, while moderate-to-severe chronic GVHD occurs in 10–20%. Reported incidence in Indian allo-HSCT cohorts is heterogeneous, reflecting differences in underlying disease, donor characteristics, conditioning regimen, graft source and GVHD prophylaxis. Higher-grade acute GVHD at presentation is associated with an increased risk of corticosteroid-refractory disease and adverse outcomes, particularly with lower gastrointestinal involvement. Moreover, in a multicenter cohort of steroid-refractory/dependent acute GVHD, mortality approached 80% among patients with grade III–IV disease and 86% among those with lower-GI involvement. These observations highlight the importance of prompt therapeutic escalation in severe GVHD. However, the optimal timing for introducing second-line treatment remains unclear, underscoring the need to explore individualized, risk-adapted approaches to multimodal management.

Objective:

To describe the clinical spectrum of severe GVHD following allo-HSCT and the real-world use, sequencing, timing, response and outcomes of multimodal salvage therapy.

Method:

A retrospective, descriptive, single centre study of 13 patients with severe GVHD managed between February 2025 and July 2026.

Acute GVHD was graded using Mount Sinai Acute GVHD International Consortium (MAGIC) criteria, while chronic GVHD was staged according to the 2014 National Institutes of Health (NIH) consensus criteria. The same criteria were used for response assessment.

Clinical and transplant characteristics, GVHD onset and severity (grade/stage), histopathological findings, treatment strategies and timing of salvage therapy were evaluated. Treatment response was assessed at Day 28 and Day 56, wherever evaluable in patients with acute GVHD, and at 12 weeks in patients with chronic GVHD. Infectious complications and mortality were also assessed.

Observations:

13 patients with severe GVHD requiring escalation beyond corticosteroids were evaluated. 8 patients had acute GVHD, with 3 patients developing GVHD following donor lymphocyte infusion, while 5 had chronic GVHD. Lower gastrointestinal involvement was the predominant clinical manifestation, occurring in 9 out of 13 patients (69.2%). Ruxolitinib was incorporated early in the treatment course, with a median interval of 4 days from initiation of corticosteroids to ruxolitinib. Treatment was individualized according to GVHD phenotype, organ involvement and clinical response. Extracorporeal photopheresis was used in 7 patients (53.8%), fecal microbiota transplantation was performed in 2 patients, and mesenchymal stromal cell therapy in 2 patients. Clinicopathological discordance between histological and clinical severity was noted in some patients. Infectious complications were frequent, with CMV reactivation being the most common, occurring in 7 patients (53.8%). Three mortalities (one from acute and two from chronic category) were encountered. Overall response rate at day 28, for acute severe GVHD, was noted to be 87.5%. At 12 weeks, 2 out of 5 patients with chronic GVHD had measurable response.

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Conclusion:

GVHD requiring escalation beyond corticosteroids remains a major therapeutic challenge. There is an unmet need for early identification of patients unlikely to demonstrate adequate steroid response who may benefit from prompt escalation to second line agents. In patients with severe or rapidly progressive disease, clinical judgement should guide timely therapeutic decisions rather than waiting for unequivocal steroid failure or histopathological confirmation. Histopathology should complement, rather than delay, clinical decision-making, particularly when clinicopathological discordance is encountered. Waiting for unequivocal steroid failure may lose a therapeutic opportunity in GVHD. Prospective studies are needed to identify predictors of steroid non-response and establish the optimal sequencing of multimodal interventions.

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P-8

The vitamin that made the difference: A case of Thiamine-responsive megaloblastic anemia

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Background:

Thiamine-responsive megaloblastic anaemia (TRMA) is a rare autosomal recessive disorder. The classical triad are megaloblastic anaemia, diabetes mellitus, and sensorineural hearing loss. The SLC19A2 gene mutation leads to defective thiamine transport, and this results in impaired cellular metabolism, giving rise to heterogenous clinical presentations. sequencing.

Case Presentation:

We report a 15-year-old boy, born to consanguineous parents, who presented with a progressive multisystem disorder. His earliest manifestation was anaemia in infancy, requiring first blood transfusion at eight months old, with haemoglobin analysis of HbE– thalassaemia. Although initially transfusion-independent, he became transfusion-dependent by ten years of age. Two years earlier, he was diagnosed with type 1 diabetes mellitus. His speech was markedly affected by bilateral severe sensorineural hearing loss. Persistent thrombocytopaenia added to clinical complexity, treated as chronic immune thrombocytopaenic purpura, with no response to immunoglobulin therapy. The constellation of puzzling manifestations raised suspicion of an underlying syndromic diagnosis. Whole exome sequencing confirmed TRMA, unifying his seemingly unrelated manifestations under a single diagnosis. Initiation of thiamine therapy resulted in tremendous improvement within weeks, with sustained haemoglobin levels and normalized platelet count. This case illustrates diagnostic fragmentation, initially managed as separate conditions. The coexistence of these features, particularly in the setting of consanguinity, should prompt consideration of a genetic illness.

Conclusion:

TRMA is a treatable genetic disorder. Early recognition is crucial as thiamine supplementation, which is cost-effective, can significantly improve haematological parameters, modify disease trajectory and prevent progression to systemic complications.

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P-9

Etiology of Pyrexia of Unknown Origin (PUO) with pancytopenia hidden in bone marrow

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Background:

Pyrexia of unknown origin (PUO) with pancytopenia is an important clinical combination that suggests infection(severe sepsis), hematological malignancy , autoimmune disorders, etc. However, diagnosing Tuberculosis as a cause of pancytopenia is rare .

Here we present a case of 26/F admitted in tertiary care hospital with prolonged fever on and off since one month with bleeding manifestation in form of haematuria without any constitutional symptoms. On routine investigations found out to have severe pancytopenia with CBC of 6.6/2100/4000 . Pt was treated with broad spectrum higher antibiotics and was also transfused with blood and blood products. TPO agonist trial was given however pt didn't respond to any of above treatment. So in view of persistent cytopenia, we performed bone marrow biopsy.

Objective:

Diagnosing etiology of PUO with PANCYTOPENIA not responding to routine treatment.

Method:

Bone marrow examination was done to find out cause of PUO with PANCYTOPENIA

Results:

Bone marrow biopsy showed hypercellular marrow with adequate trilineage hematopoiesis; focal non necrotising granulomatous inflammation. Histological findings favour mycobacterial etiology. Pt was started on AKT. Fever and cytopenias responded to treatment within 2 weeks. CBC- 9.5/4500/28000

Conclusion:

It's important to keep disseminated TB as a differential in a case of PUO with cytopenias. Bone marrow studies play a pivotal role in diagnosis . Early initiation of AKT shows prompt response.

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Beyond the ovary: an uncommon presentation of paediatrics diffuse large B-cell lymphoma

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Background:

Primary extra nodal diffuse large B cell lymphoma (DLBCL) is an exceptionally uncustomary presentation of Non Hodgkin lymphoma, having commonly arising within lymph nodes. Its rarity, coupled with non-specific clinical manifestations and overlapping radiological appearances, often results in diagnostic uncertainty and delayed recognition. These tumours frequently masquerade as more common ovarian or renal pathologies, making accurate determination of the organ of origin and timely diagnosis particularly challenging.

Case Description:

A 6-year-old Syrian girl presented with breathlessness, lethargy, and constipation, proceeded by a three-year history of progressive weight loss, poor appetite, migratory polyarthralgia, and intermittent vasculitic rash. She is the second born of a pair of identical twins and her elder twin has established Systemic Lupus Erythematosus (SLE), diagnosed a year prior to this. The family history is notable for multigenerational consanguinity and a gastrointestinal malignancy in her paternal great-grandmother. Child was in respiratory distress with features of massive pleural effusion with a large abdominal mass detected on palpation. Initial contrast enhanced CT demonstrated bilateral adnexal masses with omental caking and abdominal lymphadenopathy, raising the suspicion of ovarian malignancy or Burkitt lymphoma. Bilateral kidneys were swollen however had no suspicious mass arising from it. Despite an elevated tumour marker CEA125, her immunophenotyping from peritoneal and pleural fluid were inconclusive. Tissue biopsy was initially precluded by haemodynamic instability. Following clinical deterioration and subsequent referral, diagnosis of possible Familial Mediteranean Syndrome was considered, of which she showed transient marked clinical response.

An Magnetic resonance imaging (MRI) later demonstrated extensive retroperitoneal and extra nodal disease without identifiable adnexal masses, suggesting a renal rather than ovarian origin.

Histopathological examination of the renal biopsy confirmed diffuse large B-cell lymphoma (DLBCL) of the germinal centre B-cell (GCB) subtype. Despite chemotherapy, the disease progressed rapidly and patient ultimately succumbed to disease related complications.

Conclusion:

This case highlights the importance of maintaining a high index of suspicion for lymphoma in children presenting with atypical abdominal masses, particularly when clinical or radiological findings are not entirely consistent with more common solid neoplasms. Large abdominal masses may obscure their true organ of origin, making adnexal and renal lesions difficult to be distinguished radiologically. Awareness of this diagnostic pitfall and systematic assessment of characteristic imaging features are key. Prompt tissue diagnosis enables timely initiation of appropriate therapy and may improve clinical outcomes.

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Thymoma-Associated PRCA Revealing a KIF23-Related CDA

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Background:

Congenital dyserythropoietic anemia (CDA) is a rare inherited disorder characterized by ineffective erythropoiesis and distinctive erythroid dysplasia. Although it typically presents in childhood or early adulthood, late-onset presentation can pose a significant diagnostic challenge. We report a diagnostically challenging case of a 46-year-old woman who presented with severe transfusion-dependent normocytic normochromic anemia and profound reticulocytopenia. Bone marrow examination demonstrated selective absence of erythroid precursors, leading to an initial diagnosis of thymoma-associated pure red cell aplasia (PRCA). She underwent thymectomy but remained transfusion dependent. Her subsequent course was complicated by pulmonary tuberculosis, while immunological evaluation revealed hypogammaglobulinemia with marked B-cell lymphopenia, fulfilling the diagnostic criteria for Good syndrome. Persistent anemia despite appropriate management prompted further evaluation for an underlying inherited erythroid disorder.

Objective:

To highlight the role of whole exome sequencing in the evaluation of unexplained transfusion-dependent anemia and its contribution to resolving a complex diagnostic dilemma.

Method:

Comprehensive clinical, hematological, immunological, radiological, and bone marrow evaluation was performed. Following an atypical clinical course despite thymectomy, whole exome sequencing was undertaken to identify an underlying genetic cause.

Results:

Whole exome sequencing identified a heterozygous KIF23 c.2252A>G (p.Asn751Ser) variant, classified as a Variant of Uncertain Significance (ACMG PM2). KIF23 variants are associated with congenital dyserythropoietic anemia type IIIA, an autosomal dominant disorder. Correlation of the genetic findings with the patient's clinical presentation, persistent transfusion dependence, exclusion of secondary causes, and overall clinicopathological features supported the diagnosis of late-onset CDA type IIIA.

Conclusion:

This case highlights the importance of reconsidering the diagnosis in patients with persistent reticulocytopenic, transfusion-dependent anemia who fail to respond as expected to treatment for presumed PRCA. Whole exome sequencing, interpreted alongside clinical and pathological findings, can uncover rare inherited erythroid disorders even in adulthood. The coexistence of thymoma and Good syndrome created a significant diagnostic pitfall, emphasizing the need for a multidisciplinary approach and timely molecular evaluation in atypical cases.

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Unmasking genetic complexity in Beta thalassaemia trait

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Background:

We present a case series of three young female patients with symptomatic microcytic hypochromic hemolytic anemia, whose clinical severity was disproportionate to the expected phenotype of β-thalassemia trait.

Case 1:

A 30-year-old female presented with progressive exertional dyspnea, easy fatigability, and generalized weakness. Investigations revealed severe microcytic hypochromic anemia (Hb 7.1 g/dL) with reticulocytosis, elevated LDH, indirect hyperbilirubinemia, and peripheral smear showing microcytosis, hypochromia, anisopoikilocytosis, and target cells. Iron profile, vitamin B12, folate, red cell enzyme studies, and autoimmune hemolytic workup were normal/negative. HPLC was suggestive of **β-thalassemia trait**, while molecular analysis identified a **heterozygous β-globin gene mutation (IVS-I-5, G>C) with co-inherited α-globin gene triplication.**

Case 2:

A 32-year-old female was referred for hematology evaluation prior to splenectomy for splenomegaly. She had chronic microcytic hypochromic hemolytic anemia with reticulocytosis, elevated LDH, indirect hyperbilirubinemia, and target cells on peripheral smear. Iron studies, vitamin B12, folate, red cell enzyme studies, and autoimmune hemolytic workup were unremarkable. HPLC demonstrated **β-thalassemia trait**, while molecular testing revealed a **homozygous α-globin gene mutation at codon 130(G —>C) consistent with Hb Sun Prairie.**

Case 3:

A 19-year-old female presented with progressive dyspnea and easy fatigability. Evaluation showed severe microcytic hypochromic hemolytic anemia with reticulocytosis, elevated LDH, indirect hyperbilirubinemia, and peripheral smear showing microcytosis, hypochromia, anisopoikilocytosis, and target cells. Iron profile, vitamin B12, folate, red cell enzyme studies, and autoimmune hemolytic workup were normal/negative. HPLC was consistent with **β-thalassemia trait**, while genetic analysis identified a **homozygous α-globin gene mutation at codon 130 (G>C), consistent with Hb Sun Prairie.**

Method:

HPLC was performed for hemoglobinopathy screening. Molecular genetic testing was undertaken to identify disease-causing α- and β-globin gene variants responsible for the severe clinical phenotype.

Results:

All three patients were young females presenting with symptomatic severe microcytic hypochromic hemolytic anemia. Laboratory evaluation showed low hemoglobin, reticulocytosis, elevated LDH, indirect hyperbilirubinemia, and peripheral smear showing microcytosis, hypochromia, anisopoikilocytosis, and target

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cells. Iron studies, vitamin B12, folate levels, red cell enzyme studies, and autoimmune hemolytic workup were normal/negative in all cases. HPLC demonstrated β-thalassemia trait in all patients.

Molecular genetic testing identified heterozygous β-globin gene mutation [IVS-I-5 (G>C)] with co-inherited α-globin gene triplication in one patient, while the other two patients harbored a homozygous α-globin gene mutation at codon 130 (G>C), consistent with Hb Sun Prairie. These genetic modifiers explained the unexpectedly severe hemolytic phenotype.

Conclusion:

Clinicians should remain vigilant when evaluating patients with -thalassemia trait who present with unexpectedly severe microcytic hemolytic anemia. Early use of HPLC followed by molecular genetic testing is crucial to detect coexisting globin gene abnormalities, allowing accurate diagnosis, appropriate management, improved prognostication, and effective genetic counseling.

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Dual Autoimmune Crisis: Active SLE with concurrent severe Aplastic Anaemia successfully treated with Horse Anti-Thymocyte Globulin

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Background:

Cytopenias occur in up to 80% of patients with systemic lupus erythematosus (SLE), but true aplastic anaemia is not among them. When a patient with established lupus develops a hypocellular marrow, the clinician faces a question of attribution: is this a rare haematological manifestation of the lupus, or a second and separate disease running alongside it? The distinction is therapeutically decisive, since the former would be expected to respond to intensified lupus-directed treatment while the latter requires T-cell directed immunosuppression. We report an adolescent in whom the answer proved to be the latter — two concurrently active autoimmune diseases, each requiring its own therapeutic logic.

Objective:

To illustrate the diagnostic reasoning by which severe aplastic anaemia was established as an entity distinct from the cytopenias of the patient's underlying lupus, and to report the response to horse anti-thymocyte globulin based immunosuppression in an adolescent carrying both diagnoses concurrently.

Method:

Single-patient case description with longitudinal clinical, laboratory and histopathological correlation. Evaluation included serial complete blood counts, two sequential bone marrow aspirate and trephine biopsies, PNH clone assay by multi-parameter flow cytometry,next-generation sequencing panel, faecal elastase-1, viral and tropical infection screening, and serial SLE serology. Attribution was tested not only by exclusion but by the observed response — or failure of response — to lupus-directed therapy. Treatment comprised h-ATG with cyclosporine A and eltrombopag, with follow-up to five months post-infusion.

Results:

A 17-year-old girl diagnosed with seronegative SLE at 12 years, with biopsy-proven ISN/RPS Class V membranous lupus nephritis and neuropsychiatric involvement, developed pancytopenia in mid-2025 (haemoglobin 6.1 g/dL, TLC 0.19 $\times$ 10^9/L, ANC 0.02 $\times$ 10^9/L, platelets 5 $\times$ 10^9/L) that persisted despite withdrawal of tacrolimus, mycophenolate and dapsone. Sequential marrow biopsies showed cellularity falling from $\sim$15% to $<$10%. PNH clone was absent ($<$1%); the NGS panel identified only two heterozygous variants of uncertain significance (SBDS p.Gly24Arg, RPA1 p.Ser517=) with an incidental pathogenic G6PD Mediterranean variant, and a normal faecal elastase-1 (386 μg/gm) excluded the exocrine pancreatic phenotype of Shwachman–Diamond syndrome. Anti-dsDNA rose from negative to 850 IU/mL concurrently with the marrow failure, but the marrow did not behave as a lupus manifestation: pulse methylprednisolone produced only a transient rise in counts, four weekly doses of rituximab 375 mg/m2 failed to consolidate haematopoiesis, and cellularity continued to fall while her renal and mucocutaneous disease remained quiescent. Horse-ATG 40 mg/m2 over four days was administered with cyclosporine and eltrombopag; headache, infusion hypersensitivity, febrile neutropenia and transaminitis occurred and were managed without dose compromise. At five months the ANC had normalised (2.46 $\times$ 10^9/L), platelets had risen to 63 $\times$ 10^9/L and haemoglobin to 9.0

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g/dL, with reticulocytosis of 3% documented, and the transfusion interval had lengthened from one to two weekly to approximately monthly.

Conclusion:

In a patient with established SLE, a hypocellular marrow should not be attributed to the lupus on serological coincidence alone; the response — or failure to respond — to lupus-directed therapy is itself a diagnostic test. Recognising acquired aplastic anaemia as a second, independent autoimmune entity permitted early escalation to T-cell directed therapy rather than further B-cell depletion. The h-ATG, cyclosporine and eltrombopag triplet produced a sustained partial haematological response with clinically meaningful reduction in transfusion dependence, despite this patient carrying a combination of diagnoses excluded from the trials from which the regimen derives.

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P-14

When the Counts Recover but the Virus Does Not: EBV-Driven Secondary Hemophagocytic Lymphohistiocytosis Evolving to Chronic Active EBV Disease in an Immunocompetent Young Adult

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Background:

Epstein–Barr virus establishes lifelong latency in more than 90% of adults and is, for almost all of them, an unremarkable event. In a minority the virus escapes cytotoxic control and drives a hyperinflammatory secondary haemophagocytic lymphohistiocytosis (HLH); in fewer still, viraemia persists and the illness becomes chronic active EBV (CAEBV) disease — a systemic proliferation of EBV-infected lymphoid cells with symptoms enduring beyond three months. The clinician treating EBV-HLH therefore faces a question of endpoint: when the fever settles and the counts recover, has the disease been treated, or only its inflammatory surface? The distinction is therapeutically decisive. Etoposide-based HLH-94/HLH-2004 chemo-immunotherapy ablates the hyperactivated effector compartment and reliably produces early remission but does not eradicate the EBV-infected clone; an underlying lymphoproliferation may declare itself only after apparent clinical improvement, and standard texts therefore counsel that patients be followed beyond symptom resolution until virological suppression is documented, a process that may take up to twelve months. Allogeneic haematopoietic stem cell transplantation remains the only curative modality in both T/NK- and B-cell disease, with reported survival of approximately 50–65% and consistently better outcomes when performed early and before the accrual of pre-transplant complications. We report a young man in whom the inflammatory and virological trajectories diverged completely.

Objective:

To document the dissociation between haematological and virological response in EBV-driven secondary HLH; to report progression to chronic active EBV disease in a previously healthy South Asian adult, a population in which the entity is sparsely described, CAEBV being reported predominantly from East Asia and from Central and South America; and to argue that failure of the viral load to fall despite sustained B-cell depletion is itself lineage-informative, implying a viral reservoir beyond the CD20-positive compartment and mandating EBER in situ hybridisation with lineage-specific immunophenotyping at diagnosis rather than in retrospect.

Method:

Single-patient case description with longitudinal clinical, laboratory and virological correlation. Evaluation included serial complete blood counts, sequential bone marrow aspiration and trephine biopsy, cerebrospinal fluid cytology, acute leukaemia and PNH panels by multiparametric flow cytometry, an inherited-HLH gene panel, comprehensive tropical, viral and autoimmune screening, and serial quantitative whole-blood EBV DNA PCR. HLH was established against the eight HLH-2004 criteria and severity graded by the HScore (Fardet et al.); CAEBV was assessed against the three essential criteria of the WHO Classification of Haematolymphoid Tumours, 5th edition. Response was adjudicated not by defervescence and count recovery alone but against the viral load trajectory, refractoriness being taken as failure of at least partial response by two to three weeks of protocol therapy. Treatment comprised intravenous immunoglobulin, dexamethasone and rituximab, followed by a body-surface-area-based HLH-2004 regimen and salvage bortezomib with ganciclovir and cyclosporine, with follow-up through relapse to refractoriness.

Results:

A 28-year-old previously healthy man presented in February 2026 with a two-month history of intermittent fever, generalised myalgia and fatigue, with hepatosplenomegaly on examination. He was pancytopenic (haemoglobin

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8.9 g/dL, TLC 0.59 × 10^9/L, ANC 0.16 × 10^9/L, platelets 15 × 10^9/L) with ferritin 4230 ng/mL, fibrinogen 169 mg/dL, triglycerides 134 mg/dL and whole-blood EBV DNA 21,717 copies/mL; VCA IgM was negative with EBV IgG positive, indicating reactivation rather than primary infection. Malaria, scrub typhus, leptospirosis, HIV, HBV, HCV, rK-39, blood and urine cultures and echocardiography were negative, as were ANA, ANCA, paraprotein studies and the acute leukaemia and PNH panels; cerebrospinal fluid showed no malignant cells and an inherited-HLH gene panel was despatched, satisfying the requirement to exclude coexisting immunodeficiency, malignancy and autoimmune disease. Five of eight HLH-2004 criteria were met — fever, splenomegaly, pancytopenia, hyperferritinaemia and marrow haemophagocytosis — with the triglyceride and fibrinogen criterion unmet and soluble CD25 and NK-cell cytotoxicity unavailable; the HScore was 219, rising to 242 on reassessment. Intravenous immunoglobulin, dexamethasone and rituximab produced defervescence, cessation of per-rectal bleeding and count recovery, but the disease proved refractory and he re-presented within three days of discharge with fever, petechiae, febrile neutropenia and vasopressor-dependent septic shock. A body-surface-area-based HLH-2004 regimen was initiated (BSA 1.435 m^2) with dexamethasone 10 mg/m^2/day tapered over eight weeks, etoposide 150 mg/m^2 twice weekly for four doses followed by weekly for four doses, and rituximab 375 mg/m^2 weekly. Counts again improved — haemoglobin 9.0 g/dL and TLC 1.44 × 10^9/L by day 20 — while the virus moved in the opposite direction, EBV DNA rising from 21,717 to approximately 148,000 and subsequently to approximately 590,000 copies/mL, an approximately 27-fold increase over baseline sustained through eight weekly doses of anti-CD20 therapy. Sustained platelet and red cell transfusion support was required throughout. Repeat marrow examination showed a hypocellular marrow with persistent haemophagocytosis; tissue biopsy for EBER in situ hybridisation and lineage assignment was precluded by refractory cytopenias and sepsis, leaving two of the three WHO-HAEM5 essential criteria for systemic CAEBV formally satisfied and the third — demonstration of EBV within T or NK cells — unassessable. On the basis of infectious mononucleosis-like illness beyond three months with sustained high-level viraemia and progressive organ dysfunction, CAEBV was diagnosed and salvage therapy begun with bortezomib 1.3 mg/m^2 subcutaneously on days 1, 4, 8 and 11, intravenous ganciclovir twice daily for 14 days and oral cyclosporine 100 mg twice daily. Adult-onset disease and thrombocytopenia, both recognised adverse prognostic factors, were present. The course was further complicated by multidrug-resistant nosocomial infection in the setting of protracted neutropenia, culminating in acute neuro-respiratory deterioration.

Conclusion:

In EBV-driven secondary HLH, defervescence and count recovery must not be accepted as evidence that the disease is controlled; the viral load is the endpoint, and serial quantitative EBV DNA is what separates self-limiting infection from progressive lymphoproliferation. Here the two trajectories diverged completely — counts improved on two separate occasions while EBV DNA rose by more than an order of magnitude — and had response been adjudicated clinically, refractoriness would have been recognised only at the point of catastrophic decompensation. The rise persisted through eight weekly doses of rituximab, which is itself informative: anti-CD20 therapy clears only the CD20-positive reservoir, and its failure argues that the infected compartment lay beyond it — in T or NK cells, or in the lymphoid progenitor pool now implicated in CAEBV pathogenesis. This case therefore does not support the B-cell phenotype described in Western series, and underscores that lineage cannot be inferred from the absence of a demonstrable T- or NK-cell lymphoproliferation; it must be established by EBER in situ hybridisation with lineage-specific immunophenotyping, obtained early, while cytopenias and sepsis still permit tissue sampling. Recognising the haematological–virological dissociation early is what creates the window for allogeneic haematopoietic stem cell transplantation, which remains the only curative modality and whose outcomes are best when it is not deferred until protocol completion.

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P-15

Individualizing the Venetoclax Window: Balancing Toxicity and Response in Acute Myeloid Leukemia: A Single-Centre Real-World Experience

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Background:

Venetoclax-based regimens have transformed treatment outcomes in acute myeloid leukemia (AML), particularly among patients unfit for intensive chemotherapy. However, pivotal trials employed fixed 28-day venetoclax dosing schedules, whereas real-world practice frequently requires individualized dose and duration modifications because of cytopenias, infections, and comorbid illness. Data describing how such individualized scheduling affects toxicity, infectious risk, and depth of response outside the trial setting remain limited.

Method:

We retrospectively analyzed 24 consecutive patients with AML who received venetoclax-containing therapy at a single centre. Data regarding baseline clinical characteristics, venetoclax dose and duration, antimicrobial prophylaxis, hematological toxicity, infectious complications, treatment modifications, measurable residual disease (MRD) response and survival were collected. As several patients had significant cytopenias before initiation of venetoclax, cytopenias were interpreted in the context of baseline blood counts and were not uniformly attributed to venetoclax.

Results:

The median age was 57.5 years (range, 8–84 years). Seventeen patients (70.8%) received venetoclax in the frontline setting, six (25.0%) for relapsed disease and one (4.2%) as a bridge to transplantation. Most patients received an azacitidine-based venetoclax regimen, with a small number receiving other venetoclax-containing backbones.

The mean documented venetoclax exposure was approximately 17.5 days per cycle, with exposure ranging from 5 to 28 days. Venetoclax duration was frequently individualized, with cycle-specific shortening and temporary interruptions documented in several patients. Most patients were initiated on the standard 100 mg venetoclax dose; a smaller subset (n=4, 16.7%) were started at a reduced dose of 50 mg, generally reflecting the presence of severe baseline cytopenias or a higher perceived risk of infectious complications.

Grade 3/4 neutropenia, thrombocytopenia and anemia during treatment were documented in 66.7%, 66.7% and 70.8% of patients, respectively. Given the high frequency of baseline cytopenias, these findings were interpreted as cytopenias occurring during therapy rather than uniformly as venetoclax-induced toxicity. Febrile neutropenia occurred in 50.0%, documented infections in 54.2%, and invasive fungal infections in 12.5% of patients.

MRD negativity was documented in 13 of 16 patients with an interpretable MRD assessment (81.3%). Among patients for whom the cycle of MRD conversion was documented, MRD negativity was achieved after a mean of approximately 3.8 cycles. When response was examined by molecular subgroup among the 16 MRD-evaluable patients, MRD negativity was observed across all major mutation categories: NPM1-mutated (2/2), core-binding-factor-rearranged (inv(16)/CBFB-MYH11) AML (2/2), RUNX1-mutated (3/4), and FLT3-mutated (ITD/TKD) AML (2/4). The two FLT3-mutated patients who remained MRD-positive included one with a co-occurring RUNX1 mutation. Single evaluable patients with TP53 or IDH2 mutations also achieved MRD negativity. No molecular subgroup was clearly associated with a lower likelihood of MRD clearance in this cohort, though small subgroup sizes precluded formal statistical comparison.

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Conclusion:

In this real-world AML cohort, venetoclax dosing was frequently individualized, both at initiation and during treatment, with a mean duration of approximately 17.5 days per cycle. Cytopenias and infections were common, though attribution to venetoclax was limited by frequent baseline cytopenias. Toxicity-driven modification of venetoclax duration and, in a subset of patients with severe cytopenias or higher infectious risk, dose reduction to 50 mg at initiation, appear essential for safe treatment delivery and did not appear to compromise disease response in this cohort, with MRD negativity achieved in 81.3% of evaluable patients after a mean of 3.8 cycles, and MRD clearance observed consistently across NPM1-, RUNX1-, and core-binding-factor-mutated subgroups, though the FLT3-mutated subgroup had a numerically lower MRD-negativity rate (2/4). Larger, controlled studies are needed to confirm that individualized scheduling preserves efficacy across molecular subtypes.

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P-16

Correlative diagnosis of multiple myeloma using bone marrow morphology serum protein electrophoresis and CRAB criteria: A case report

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Background:

Multiple myeloma is a clonal plasma cell neoplasm characterized by bone marrow plasmacytosis, monoclonal immunoglobulin production, and myeloma defining events. Due to its variable clinical presentation, diagnosis relies on a combination of clinical, laboratory, and pathological findings.

Objective:

To highlight the importance of clinicopathological correlation in the diagnosis of multiple myeloma using bone marrow examination, serum protein electrophoresis (SPEP), and CRAB criteria.

Method:

A 77-year-old male with clinical suspicion of multiple myeloma underwent peripheral smear examination, bone marrow aspiration, and serum protein electrophoresis. The findings were correlated with hematological parameters and CRAB criteria to establish the diagnosis.

Results:

Peripheral blood smear examination revealed normocytic normochromic anemia (hemoglobin: 8.8 g/dL) with marked rouleaux formation. Bone marrow aspiration was adequate and hypercellular for the patient's age, demonstrating 20% plasma cells. Serum protein electrophoresis revealed a prominent monoclonal (M) band measuring 4.32 g/dL. The laboratory and radiological findings fulfilled the CRAB criteria, including hypercalcemia (serum calcium: 13.2 mg/dL), anemia (hemoglobin: 8.8 g/dL), renal impairment (serum creatinine: 1.98 mg/dL and uric acid: 13.2 mg/dL), and right hip arthropathy. Correlation of the peripheral smear findings, bone marrow morphology, serum protein electrophoresis, and CRAB criteria established the diagnosis of Multiple Myeloma (ICD-10: C90).

Conclusion:

This case emphasizes the pivotal role of bone marrow morphology in conjunction with serum protein electrophoresis and clinical assessment for the diagnosis of multiple myeloma. Early clinicopathological correlation facilitates prompt diagnosis, appropriate ancillary investigations, and timely initiation of definitive therapy.

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HLH- Macrophage Activation syndrome in Juvenile Idiopathic Arthritis

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Background:

Macrophage Activation Syndrome (MAS) is a potentially fatal hyperinflammatory complication seen in >50% of infectious conditions like CMV and EBV.

About 10% of patients with systemic juvenile idiopathic arthritis (sJIA) develop MAS, representing a form of secondary hemophagocytic lymphohistiocytosis (HLH). It is characterized by uncontrolled activation of macrophages and T lymphocytes, resulting in a cytokine storm, multiorgan dysfunction, and high mortality if not recognized early.

Diagnosis remains challenging despite revised criteria, as the clinical features of MAS frequently overlap with active systemic juvenile idiopathic arthritis (sJIA) and severe infections, leading to its underdiagnosis. While the HLH-2004 diagnostic criteria remain important in the evaluation of MAS, these overlapping manifestations led to the development of disease-specific classification criteria for MAS in sJIA in 2016.

Objective:

To emphasize the occurrence of macrophage activation syndrome, a secondary form of HLH, in systemic juvenile idiopathic arthritis and highlight the importance of early recognition for management using clinical and laboratory parameters.

Method:

A child with previously diagnosed systemic juvenile idiopathic arthritis presented with persistent, prolonged fever, worsening arthritis and splenomegaly for a year, despite standard anti-inflammatory therapy. Comprehensive laboratory investigations, including complete blood counts, serum ferritin, triglycerides, fibrinogen, liver function tests, inflammatory markers, coagulation profile, bone marrow aspiration and lymph node biopsy, were performed. Based on clinical and laboratory findings HLH was diagnosed and MAS was favoured, with the help of HLH-2004 criteria and 2016 PRINTO classification.

Results:

The patient demonstrated persistent, prolonged fever for a year, splenomegaly, anaemia, markedly elevated serum ferritin, hypertriglyceridemia, elevated liver enzymes, and increased lactate dehydrogenase levels.

Bone marrow examination revealed hemophagocytosis, confirming macrophage activation. The patient fulfilled the diagnostic criteria for MAS in sJIA.

Prompt initiation of high-dose intravenous corticosteroids followed by cyclosporine resulted in rapid clinical improvement, normalization of inflammatory markers, and recovery of haematological parameters. Early diagnosis prevented progression to multiorgan failure and significantly improved clinical outcome.

Conclusion:

Macrophage Activation Syndrome should be suspected in every patient with systemic juvenile idiopathic arthritis who develop persistent fever, hyperferritinemia, liver dysfunction, and/or coagulopathy for early treatment and reduction of morbidity and mortality.

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Extramedullary plasmacytoma - a rare case

Presenting Author: Dr. Dhvani Thakker

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Institution: 1. Professor in Pathology, D. Y. Patil School of Medicine, Navi Mumbai
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Background:

Plasmacytoma is a localized plasma cell neoplasm characterized by a monoclonal proliferation of plasma cells.

Extramedullary plasmacytomas (EMPs) are rare (3-4%) plasma cell neoplasms that arise outside the bone marrow, most commonly occurring in the head and neck region, while spinal and dural (<1%) involvements are exceptionally uncommon.

Objective:

To report an unusual clinical presentation of extramedullary plasmacytoma manifesting as a dural space-occupying lesion in the thoracic spine.

Method:

A 48-year-old male presented with chronic lower back pain with clinical suspicion of spondylodiscitis. Case analysis with CT-guided biopsy and Immunohistochemistry markers with CD138, Kappa light chain and Lambda light chain confirmed the diagnosis of plasma cell neoplasm.

Results:

A 48-year-old male presented with lower back pain persisting for 8 months. Neurological and physical evaluations revealed a dural space-occupying lesion located in the D5-D8 spinal segments, with no involvement of bowel and bladder function, and palpable distal pulses. A CT-guided biopsy of the dural space-occupying lesion and Immunohistochemistry markers demonstrated strong and diffuse positivity for CD138 and Kappa, while Lambda markers were negative, establishing the monoclonal nature of the plasma cell proliferation.

Conclusion:

Dural extramedullary plasmacytoma of the thoracic spine is a rare(<1%) clinical entity. This case highlights the importance of incorporating plasmacytoma into the differential diagnosis of spinal dural space-occupying lesions, where prompt CT-guided biopsy and comprehensive immunohistochemical panel analysis (CD138, Kappa, and Lambda) are vital for an accurate diagnosis and subsequent management.

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P-19

When itching precedes the diagnosis: classical Hodgkin Lymphoma presenting with prurigo nodularis

Presenting Author: Dr. Prianuz Dewraja

Co-Authors: Dr. Jina Bhattacharyya

Institution: Gauhati Medical College, Guwahati

Background:

Pruritus is a recognized systemic manifestation of Hodgkin lymphoma (HL), but severe pruritus presenting as prurigo nodularis is uncommon. Persistent pruritus may precede the diagnosis of HL and can result in significant secondary skin changes due to chronic scratching.

Objective:

To describe an unusual presentation of classical Hodgkin lymphoma (cHL) with severe generalized pruritus and secondary excoriated prurigo nodularis, with complete resolution following lymphoma-directed therapy.

Method:

A detailed clinical evaluation, skin biopsy, lymph-node biopsy, immunohistochemistry, and imaging were performed. The clinical response of the cutaneous manifestations to chemotherapy was subsequently assessed.

Results:

A 19-year-old male presented with severe generalized pruritus of one-year duration. The itching initially involved the palms and soles and progressively increased in intensity, resulting in repeated scratching, multiple excoriations, and post-inflammatory hyperpigmentation involving the lower limbs, trunk, neck, scalp, and upper limbs. There was no preceding primary rash. Four to five months after onset of pruritus, the patient developed progressively increasing cervical swelling. Biopsy of the cervical lymph node with immunohistochemistry confirmed classical Hodgkin lymphoma. Skin biopsy showed orthokeratosis, parakeratosis, hypergranulosis, epidermal necrosis with regeneration, acanthosis, papillary dermal fibrosis, vascular proliferation, and mild perivascular lymphocytic infiltrate, consistent with excoriated prurigo nodularis, with no evidence of malignancy. The patient was initiated on ABVD chemotherapy. Following two cycles, there was marked clinical improvement, with complete disappearance of pruritus and healing of the skin lesions.

Conclusion:

Severe pruritus may precede the diagnosis of cHL and, through chronic scratching, can manifest as prurigo nodularis. In patients with persistent unexplained pruritus, particularly when associated with lymphadenopathy, an underlying hematological malignancy should be considered. Complete resolution of pruritus and healing of the skin lesions following lymphoma-directed therapy further supports an association with the underlying cHL.

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When MDS Hides a Second Clone IgM - κ Monoclonal Gammopathy with Cold Agglutinin Disease: a Diagnostic Challenge

Presenting Author: Neerukonda Sriteja

Co-Authors: Mallikarjun Kalashetty, Ashish Dixit

Institution: Department of Clinical Haematology, Manipal Hospital, No. 98, HAL Old Airport Road, Kodihalli, Bengaluru, Karnataka, 560017, India

Background:

The coexistence of myelodysplastic syndrome (MDS) and lymphoproliferative disorders poses a diagnostic challenge, particularly when overlapping cytopenias obscure an underlying lymphoplasmacytic neoplasm. Cold autoimmune hemolytic anemia (AIHA) may provide an important diagnostic clue to an associated IgM-producing disorder.

Objective:

To highlight the diagnostic complexity of concurrent MDS, Waldenström macroglobulinemia (WM), and cold AIHA and emphasize the importance of comprehensive re-evaluation in patients with progressive or treatment-refractory cytopenias.

Method:

We describe a 76-year-old man with a prior diagnosis of MDS who developed progressive cytopenias and severe hemolytic anemia. Diagnostic evaluation included complete blood counts, hemolysis workup, cold agglutinin titers, bone marrow morphology and immunohistochemistry, flow cytometry, cytogenetic and molecular studies, serum protein electrophoresis, immunofixation, serum IgM and free light-chain assays, and PET-CT imaging.

Results:

The patient demonstrated severe anemia, thrombocytopenia, and intermittent neutropenia. Direct antiglobulin testing was C3d-positive and IgG-negative, with a cold agglutinin titer of 1:512, consistent with cold AIHA. Bone marrow examination showed hypercellularity, erythroid predominance, dysmegakaryopoiesis, and low blasts, with an ETV6 mutation (VAF ~7.95%). Further evaluation revealed IgM-kappa monoclonal gammopathy, an M-band of 4.3 g/dL, and markedly elevated serum IgM of 7930 mg/dL. Flow cytometry demonstrated clonal plasmacytic cells, while PET-CT showed diffuse skeletal marrow involvement, supporting WM with concurrent low-blast MDS. Therapeutic plasma exchange reduced IgM to 2100 mg/dL, followed by rituximab and bendamustine-based therapy.

Conclusion:

Progressive or refractory cytopenias in patients with presumed MDS warrant comprehensive diagnostic reassessment. The presence of cold AIHA and markedly elevated IgM should prompt evaluation for an underlying lymphoplasmacytic disorder. Recognition of concurrent WM and MDS enables timely plasma exchange and chemoimmunotherapy, potentially improving clinical outcomes.

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P-21

Unmasking a Rare Telomeropathy: Homozygous CTC1 Mutation Presenting as Transfusion: Dependent Pancytopenia and Multisystem Disease in a Young Adult: A case of Coats Plus Syndrome

Presenting Author: Neerukonda Sriteja

Co-Authors: Mallikarjun Kalashetty, Ashish Dixit

Institution: Department of Clinical Haematology, Manipal Hospital, No. 98, HAL Old Airport Road, Kodihalli, Bengaluru, Karnataka, 560017, India

Background:

Telomere biology disorders (TBDs) are inherited conditions caused by defective telomere maintenance, resulting in bone marrow failure and multisystem manifestations. Coats Plus syndrome, or cerebroretinal microangiopathy with calcifications and cysts (CRMCC), is a rare autosomal recessive TBD caused by biallelic CTC1 mutations and may clinically overlap with other inherited bone marrow failure syndromes.

Objective:

To highlight the diagnostic challenges of CTC1-related Coats Plus syndrome and emphasize the role of comprehensive genetic evaluation in young patients presenting with unexplained pancytopenia and multisystem features suggestive of telomeropathy.

Method:

We describe a 19-year-old male from Bihar, India, with recurrent fever, weakness, weight loss, severe transfusion-dependent anemia, mucocutaneous abnormalities, and splenomegaly. Evaluation included complete blood counts, transfusion history, chromosomal fragility testing, abdominal imaging, liver assessment, fundoscopic examination, and whole-exome sequencing.

Results:

The patient had received approximately 40 packed red blood cell transfusions and demonstrated pancytopenia, with hemoglobin 5.4 g/dL and platelet count 30,000/μL. Examination revealed hyperpigmentation, nail dystrophy, buccal pigmentation, and splenomegaly. Imaging showed chronic liver disease with portal hypertension, splenomegaly (17.4 cm), splenorenal collaterals, and F2 fibrosis. Fundoscopy revealed right peripapillary hemorrhage. Chromosomal fragility testing was negative. Whole-exome sequencing identified a homozygous CTC1 c.1451A>C (p.His484Pro) variant, confirming Coats Plus syndrome/CRMCC.

Conclusion:

Unexplained pancytopenia with mucocutaneous, hepatic, and retinal manifestations should prompt evaluation for an underlying telomere biology disorder. Early molecular diagnosis of CTC1-related Coats Plus syndrome facilitates multidisciplinary management, genetic counseling, and timely consideration of hematopoietic stem cell transplantation for marrow failure.

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Determining stability of complete blood count samples stored in the Hematology laboratory at recommended temperature

Presenting Author: Dr. Uma Ramanathan

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Institution: Hematology Section, Department of Laboratory Medicine, Lalita Girdhar Tower (S1),
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Background:

Reliable stability of stored EDTA blood samples is essential for reflex testing, minimizing repeat venipuncture, and optimizing laboratory workflow. Most published studies have evaluated only routine CBC parameters. This study assessed the stability of routine and advanced hematological parameters and peripheral smear morphology in EDTA samples stored for up to 96 hours under recommended conditions.

Objective:

To determine retention times for EDTA blood samples by evaluating the stability of complete blood count parameters, reticulocyte parameters, IPF, and peripheral smear morphology under recommended storage conditions.

Method:

A prospective descriptive study was conducted on 100 EDTA blood samples stored at 2–8°C. Samples were analyzed on the Sysmex XN-1000 at baseline, 12, 24, 48, 72, and 96 hours. CBC parameters and peripheral smear morphology were assessed up to 96 hours, while reticulocyte parameters and IPF were evaluated up to 72 hours. Stability was assessed using percentage stability (±10% deviation from baseline), median percentage bias, and Bland–Altman analysis.

Retention time was determined by integrating analytical performance with peripheral smear findings.

Results:

Routine erythrocyte parameters demonstrated the greatest stability, remaining acceptable for up to 96 hours. Total leukocyte count, platelet count, reticulocyte count, RET-He, and RBC. He remained stable for 72 hours. Monocytes remained stable for 48 hours, whereas eosinophils, basophils, MPV, IRF, and RPI remained stable for 24 hours. IPF showed the earliest deterioration, remaining stable for only 12 hours. Peripheral smear findings remained reliable for 48 hours, after which progressive leukocyte degeneration and red cell crenation were observed.

Conclusion:

This study demonstrates that the stability of EDTA blood samples is parameter-dependent, necessitating a shift from uniform to parameter-specific retention policies. Adoption of these evidence-based retention limits can maximize the diagnostic utility of stored EDTA samples, support efficient laboratory practice, and reduce unnecessary repeat blood collection.

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First Report of Molecular Characterization of Congenital Dyserythropoietic Anaemia Type I with a CDAN1 gene mutation diagnosed by Next Generation Sequencing panel

Presenting author: Arati Nandan Saptarshi

Co-authors: Rashmi Dongerdiye, Tejashree Anil More, Dr. Prabhakar S. Kedar

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Abstract:

Background:Congenital dyserythropoietic anaemias (CDAs) are a rare, heterogeneous group of disorders characterized by ineffective erythropoiesis. CDAI is very rare and caused due to mutations in the CDAN1 or c15orf41gene. There is no report onthe molecular characterization of CDA I in the Indian population. An 8-year-old transfusion-dependent patient was referred forhaemolytic anaemia workup. Common causes of congenital haemolytic anemia were ruled out.A bone marrow aspiration study showed erythroid hyperplasia with mild dyserythropoiesis. Thus, the patient was considered for molecular characterization using next-generation sequencing.

Objective:

Diagnosis of unexplained congenital anaemia using next-generation sequencing.

Method:

Next-generation sequencing panel was used for identification of the disease-causing variant; bioinformatics tools were used to predict pathogenicity of the variantand functional validation was performed by gene expression studies using quantitative real-time PCR.

Results:

Red cell membrane, enzyme, hemoglobinopathy, and autoimmune hemolytic anaemia studies were normal. Bone marrow aspiration study showed erythroid hyperplasia with mild dyserythropoiesis.The patient showed c. 2671C>T, p. Arg891Cys variant in the CDAN1 gene usingnext-generation sequencing.Bioinformatics tools like SIFT, PolyPhen 2.0 predicted the variant to be damaging and deleterious. The expressions of CDAN1 and c15orf41 genes studied using quantitativereal-time PCR were significantly reduced as compared to healthy controls.

Conclusion:

Next-generation sequencing confirmed the diagnosis of CDA I in a patientwho underwent excessive investigationsearlier. CDA, being clinically similar to other prevalent causes of anaemia, should be kept in mindespecially when the common causes have already beenruled out.Further, the use of in-silico tools and gene expression analysis validated the functional impact of this variant.

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