66TH ANNUAL CONFERENCE OF INDIAN SOCIETY OF HAEMATOLOGY & BLOOD TRANSFUSION

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ISHBT INDIAN SOCIETY OF HEMATOLOGY & BLOOD TRANSFUSION

UTTAR PRADESH HEMATOLOGY GROUP UPHG

Haematocon 2025

Conference News Bulletin

DAY 3

LUCKNOW

6TH - 9TH NOVEMBER, 2025

INNOVATIONS IN HEMATOLOGY: SHAPING THE FUTURE OF DIAGNOSIS AND TREATMENT

HOLIDAY INN LUCKNOW AIRPORT, AN IHG HOTEL, UTTAR PRADESH, INDIA

NEW LEADERSHIP TO SHAPE ISHBT FUTURE

The AGM of ISHBT during Haematocon-2025 welcomed new president and gave away awards to the best performers

BRIGADIER Dr Tathagat Chatterjee took over the presidentship of Indian Society of Haematology and Blood Transfusion (ISHBT) from current chairperson of Prof Sarmila Chandra on the penultimate day of Haematocon-2025.

Addressing the annual general body meeting on Saturday, Dr Chatterjee, who has vast exposure attending international symposiums and experience in the field of hematology assured that he would strive to take ISHBT to a new height.

ISHBT Secretary Prof Tuphan Kanti Dolai informed the ISHBT AGM about various activities of the society and narrated as to how ISHBT has grown leaps and bounds in recent years. Prof Dolai said ISHBT's life membership has increased by 137 from 2202 to 2339.

The AGM discussed taking the quiz competitions to undergraduate level after its spectacular success post doctoral and post graduate level.

AGM thanked Prof SP Verma for smooth conduct of the Hematocon-2025 at Lucknow with registration in the annual event touching 1,500. Dr Ankit Khurana, organizing secretary of Haematocon-2026, briefed about progress of the event which is going to be held in Ahmedabad.

Prof RK Jena, secretary Indian College of Haematology, academic wing of ISHBT, briefed about ICH's activities. ICH Guidelines on Thalassemia was released on this occasion. ICH zonal council members were elected uncontested.

Dr. Sreejesh Sreedharanunni of PGIMER, was presented Dr KC Das memorial award for best published article in any national and international journal. Dr Puja Choudhary was given the Dr DN Das best paper award for the best original paper published in IJHBT and the best reviewer award went to Dr Dibyajyoti Sahoo, Dr Ankasha Garg and Dr Rashi Garg. Dr Mallesh Dhanush was given best research paper award while Dr Abatar Kishan Ganju was presented the member with valuable contributions to society membership.

The best faculty of masterclass was won by Dr Rakhee Kar. The best DM/DrNB award went to Dr Namrata Kaul of PGIMER and Dr Amiya Ranjan Nayak of Bagchi Sri Shankara Cancer Centre and Research Institute, Odisha. The best local chapter award was won by Varanasi Haematology Group.

The winner of Quiz for PG and PDT along with Dr JC Patel best paper award will be given away at the valedictory session on Sunday.

CALL FOR ACTION

Professionals, research, innovation and delivery is the way forward

INDIAN Society of Haematology and Blood Transfusion (ISHBT) has now completed more than than 50 years of its challenging journey as the sole national scientific society in hematology. The annual conference of the society provides a platform for all professionals in the field to come together and discuss recent advances in diagnosis and management of haematological disorders.

The past 25 years have witnessed tremendous advancement in the training and fellowship programmes including DM Clinical Hematology/Hematopathology as well as DrNB Doctorate programmes of NBE in Hematology. This has increased the specialist cadre in hematology and improved the prospects of this subject nationally catering to the professional needs of patients with haematological diseases. However, we need more of such specialist in our country and for this robust awareness programmes are the need of the hour.

BRIG (DR) TATHAGATA CHATTERJEE
President ISHBT (Nov 25–Nov 26)

Research in hematology has to be taken more seriously now. Collaborative projects with scientific institutes of excellence is the need of the hour. We must have MOUs with top institutes like IITs, NCCS(Pune), DIPAS(Delhi), IIS(Bangalore) in the fields of nanotechnology, cell sciences and molecular biology, proteomics and Artificial intelligence.

A strong committee must be formed to implement DM Hematopathology in NEET-SS exams so that appointments of Hematopathologist can be created in departments of Pathology in various medical colleges.

We must also go all out to increase the number of life members of our society to at least 5000 in the next two years. This is a realistic target and all of us must share this responsibility.

I take this opportunity to thank all the respected members of our society for giving me the privilege to be Executive body member in the past and now the President Elect of ISHBT this year. I assure you of my hard work and dedication that will bring laurels to the society.

Long live ISHBT. Jai Hind!

Metabolic targeting platelets key to combat thrombosis

THE interaction between metabolic pathways and cell signaling plays a crucial role in numerous biological processes, including innate and adaptive immunity, cancer development and inflammation. Although studies of platelet bioenergetics suggest metabolic flexibility, the specific contributions of metabolic regulation to platelet activation and thrombosis remain poorly understood.

Pyruvate kinase M2 (PKM2), a key regulator of aerobic glycolysis, supports lactate production and metabolic reprogramming in cells. Our research demonstrates that restricting the formation of PKM2 dimers using the small molecule ML265 suppresses lactate production and glucose uptake in activated human and mouse platelets. This inhibition also impairs platelet activation, aggregation, and thrombus formation under arterial shear conditions in vitro.

Mechanistically, limiting PKM2 dimerization leads to downregulation of PI3K-Akt/GSK3 signaling in both human and murine platelets. In proof-of-concept experiments, mice with platelet-specific PKM2 deficiency showed reduced agonist-induced platelet activation, aggregation, and PI3K-Akt/GSK3 signaling, and were less prone to arterial thrombosis in FeCl3-induced carotid and laser-induced mesenteric artery injury models.

Notably, this occurred without affecting normal hemostasis. Similarly, wild-type mice treated with ML265 showed resistance to arterial thrombosis without changes in tail bleeding times. In a venous thrombosis model (IVC stenosis), both PKM2-deficient mice and ML265-treated wild-type mice had a significantly lower thrombus burden 48 hours post-surgery compared to controls.

This reduction correlated with lower levels of citrullinated histone H3 (CitH3), a marker of neutrophil extracellular traps (NETs), and improved IVC wall contractility. Mechanistic studies revealed that thrombin-stimulated platelets from either PKM2-deficient mice or ML265-treated wild-type mice had reduced SNAP23 phosphorylation and PF4 release, indicating impaired α-granule exocytosis.

The releasates from these platelets also had diminished capacity to induce NETosis. Furthermore, human whole blood treated with ML265 and perfused over a tissue factor-coated surface at venous shear rates showed significantly reduced platelet-leukocyte aggregate formation. Consistent with murine results, ML265-treated human platelets stimulated with thrombin exhibited reduced PF4 release and generated releasates that were less effective at triggering NETosis.

These findings highlight a central role for PKM2 in regulating platelet metabolism, signaling, and function in both arterial and venous thrombosis, identifying it as a promising therapeutic target for antithrombotic interventions without affecting normal hemostasis.

PROF ANIL K CHAUHAN
Division of Hematology/ Oncology, University of Iowa, US

New findings highlight a central role for PKM2 in regulating platelet metabolism, signaling, and function in both arterial and venous thrombosis

Mechanistic basis of anemia

PROF MOHANDAS NARLA
New York Blood Center

ANEMIA is a major global health problem affecting nearly 2 billion individuals. Significant research efforts have focused and continue to be focused on defining the mechanistic basis of anemia. The major factors that contribute to anemia are extravascular hemolysis due to decreased red cell lifespan, intravascular hemolysis due to complement activation and decreased red cell production due to ineffective erythropoiesis in the bone marrow. These distinct mechanisms individually or in combination are responsible for anemia in various human disorders.

Anemia due to decreased red cell lifespan is a major contributor to anemia in red cell membrane disorders, enzymopathies such as G6PD- and PK-deficiencies and hemoglobinopathies including sickle cell disease. Complement mediated-intravascular hemolysis is a major feature of anemia in PNH while ineffective erythropoiesis is a significant contributor to anemia in thalassemia, Diamond-Blackfan anemia and myelodysplasia.

In my presentation I dealt on the laboratory diagnosis including genetic analysis and treatment options for inherited red cell membrane disorders including hereditary spherocytosis, hereditary elliptocytosis, Southeast-Asian ovalocytosis, hereditary xerocytosis and hereditary hydrocytosis.

CONFERENCE GLIMPSES

How I manage newly diagnosed multiple myeloma in 2025?

THE treatment of multiple myeloma (MM) has significantly changed over the past decade with the introduction of several new therapies and the development of highly effective and novel combinations that have led to deep and durable responses in patients with newly diagnosed myeloma.

While autologous stem cell transplantation (ASCT) has remained an integral component of myeloma therapy strategy, there has been increasing convergence in terms of the initial regimens for treatment of MM. An accurate diagnosis and risk stratification remains the cornerstone of myeloma therapy, especially the accurate classification and distinguishing active myeloma from precursor phases of the disease such as monoclonal gammopathy of undetermined significance and smoldering multiple myeloma.

DR SHAJI KUMAR
Professor of Medicine and Consultant at the Mayo Clinic, US

The primary goals of initial therapy of MM remains consistent and is aimed at rapid control of the disease, achievement of deep responses, reversal of disease related complications, limit the impact of initial therapy on the ability to collect stem cell transplant and achieve all these goals with minimal toxicity using a treatment that is best adapted for the individual patients.

The treatment selection for MM is primarily driven by two important factors-the risk characteristics or biological aggressiveness of the disease that often dictates how intense a treatment that we hope to deliver for the patient and the patient frailty that determines how intense a therapy the patient can tolerate. Phase three trials over the past year few years have consistently demonstrated improved outcomes with the use of multi-drug combinations that incorporates A proteasome inhibitor, immunomodulatory drug and an anti CD38 monoclonal antibody. These regimens have demonstrated improved outcomes in both transplant eligible as well as those considered ineligible for transplant if the patients have functional status that allows them to receive a 4-drug combination with its increased toxicity.

The current approach to treatment of myeloma involves assessment of the disease risk primarily using genomic markers as with the newly proposed IMS MWG risk stratification and also considering other risk factors that have been described in this disease along with the formal assessment of the frailty status.

Based on this the patients should be started on a 4-drug combination or in case there is concern about tolerability start with a 3 or 2 drug combination and gradually escalate as tolerated.

J B CHATTERJEA ORATION

Sickle Cell Disease: The past, present and the future

SICKLE Cell Disease (SCD) arises due to a mutation in the *HBB* gene (CAG → GTG), resulting in a single amino acid substitution in the β-globin chain at the 6th position (β6Glu →Val). This simple genetic "misspelling" leads to profound clinical variability, making SCD one of the greatest enigmas of medical science. Distinct haplotypes like Senegal, Benin, Bantu and Arab-Indian exhibit remarkable differences in pathophysiology and phenotype. The Arab-Indian haplotype is predominantly observed in India.

PROF RK JENA
Secretary, ICH & EHG
Past-President ISHBT

SCD: THE PAST

The first case of SCD in India was reported in the Nilgiri Hills of Tamil Nadu by Lehman and Cutbush (1952), followed by reports from Odisha (Dr. Bijaya Nanda, 1967) and Gujarat (Dr. Yazdi Italia, 1978). Despite being identified over 70 years ago, SCD has long suffered from neglect. Hydroxyurea (HU), approved in 1998, remains the only reliable, cost-effective disease-modifying therapy available.

Several Indian clinicians and scientists have made pioneering contributions to SCD research, including Dr BC Kar, Dr D Patel and Dr Pradeep Patra (Burla, Odisha); Dr PK Patra (Chhattisgarh); Dr Yazdi Italia (Gujarat)and Dr Dipika Mohanty, Dr Roshan Kolah, Dr Manisha Madkaikar, Dr Heena Tabassum (ICMR)

The National Health Mission (NHM) launched Sickle Cell Control Programmes in Maharashtra (2006), Gujarat (2006), and Odisha (2010).

SCD: THE PRESENT

A major milestone was the 3rd Global Congress on Sickle Cell Disease held in Bhubaneswar (21–24 February 2017). This event brought together experts from the Sickle Global Network, ICMR, DBT, NGOs, and various Ministries of the Government of India.

The advocacy team of ISHBT including Dr TK Dolai, Dr HP Pati, Dr Maitreyee Bhattacharyya, Dr Manoranjan Mahapatra including myself played a pivotal role in sensitizing policymakers, including Shri Arjun Munda, Hon'ble Minister for Tribal Affairs, about the magnitude of SCD in India and the need for a National Control Programme.

During Haematocon 2022 (Kolkata), the ICH–ICMR Guidelines on the Management of SCD were released. Discussions among ministers, ISHBT, and ASH representatives (Dr Narla Mohan Das and Dr Alexis Thompson, President Elect and President of ASH) reinforced the urgency of a national mission.

This culminated in a historic moment on 1st July 2023, when the Hon'ble Prime Minister of India Shri Narendra Modi launched the National Sickle Cell Anaemia Elimination Mission (NSCAEM), with clear objectives, strategic pillars, and dedicated budgetary support. Under this mission, ICMR was directed to prioritize SCD research, including the development of novel therapies. The program covers 17 high-burden states and includes capacity-building workshops in collaboration with ISHBT and NHM.

Odisha's model has emerged as a national benchmark, adopting innovative, community-level technologies such as Microchip Electrophoresis (Gazelle) and Dried Blood Spot–HPLC under a public–private partnership model. This approach ensures cost-effectiveness, sustainability, and accuracy in detecting all hemoglobinopathies (Sickle, Thalassemia, HbE) in a single test. As a result, Odisha reported the highest prevalence in India (Homozygous 1.98%, Trait 7.8%, total ≈10% of the population).

Community-based research by Dr Bontha V Babu (ICMR, Delhi) has also highlighted the significant social stigma associated with SCD and led to the development of an ICMR Stigma Scale and practical recommendations for stigma reduction.

SCD: THE FUTURE

Several new therapies are under development like Sailin-HbS (AYUSH product), Oral Decitabine and Etavopivat. A Phase I/II Gene Therapy trial is currently underway in India.

Unmet needs in SCD management include strengthened healthcare infrastructure and manpower, Model Day Care Centres providing holistic one-window care, Stigma reduction strategies, development of new, accessible drugs and expansion of PND and BMT facilities

The ICH–ICMR Roadmap for SCD in India provides a strategic framework emphasizing identification, accessibility, efficiency, quality, safety, equity, and empowerment.

CONFERENCE GLIMPSES

DR. MALTI SATHE ORATION LECTURE

Challenges in management of aplastic anemia

APLASTIC anemia (AA) is a rare but potentially fatal condition with bone marrow failure characterised by pancytopenia and a hypocellular marrow. Though uncommon globally, its prevalence is significantly higher in Asia, including India. The exact reason for the increased prevalence of AA in India is not known. High incidence of AA in India has been linked to lower socio-economic status. At All India Institute of Medical Sciences (AIIMS), Delhi we usually see around 300-500 new cases of AA annually.

DR MANORANJAN MAHAPATRA
Professor and Head Department of Hematology at AIIMS, New Delhi

Once considered uniformly fatal, its prognosis has dramatically improved over the past two decades owing to refined diagnostic modalities, advances in immunosuppressive therapy (IST), wider applicability of hematopoietic stem cell transplantation (HSCT). The management of AA is undergoing a paradigm shift, moving toward individualized therapy based upon genetics, molecular risk markers, and donor availability.

The challenges in Management of AA in developing countries including India are plenty, which include - long interval between disease onset to diagnosis and from diagnosis to treatment, infections present in majority of patients at the time of diagnosis, lack of standard of care (only 20-30 percent of patients receive HSCT or ATG), cost of therapy and socioeconomic issues and lack of patient support mechanisms.

Although the supportive care facilities have significantly improved over the years, the above mentioned challenges are the major barriers for successful outcome of aplastic anemic patients in developing countries. The definitive management of AA include Allogenic hematopoietic stem cell transplant (HSCT) and immunosuppressive therapy (IST) including anti-thymocyte globulin with Cyclosporine-A. The integration of Eltrombopag, an oral TPO-RA, into first-line IST is a recent development in management of AA.

Aplastic anemia management is undergoing transformative evolution. In both adult and pediatric populations, the management of aplastic anemia requires a combination of accurate diagnosis, timely definitive therapy, and meticulous supportive care. With refining of immunosuppressive therapy (IST), growing experience with haplo-HSCT, and refined diagnostics enabling accurate classification, aplastic anemia has become a largely curable disease, if diagnosed and treated early.

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Platelet refractoriness: diagnosis and treatment

PLATELET refractoriness is a significant clinical challenge commonly encountered in hematology and transfusion medicine. It refers to a poor post-transfusion platelet count increment following at least two consecutive platelet transfusions. Identifying the cause of refractoriness and instituting appropriate corrective measures are crucial for ensuring effective patient care.

The diagnosis of platelet refractoriness is primarily based on the Corrected Count Increment (CCI), which assesses the rise in platelet count after transfusion relative to the number of platelets administered. A one-hour CCI of less than 7,500 to 10,000 generally suggests an immune-mediated cause, while a 24-hour CCI of less than 5,000 indicates non-immune factors. Alternatively, the Percent Platelet Recovery (PPR) can also be used, with values below 20 percent confirming refractoriness.

Non-immune causes account for nearly 60 to 80 percent of all cases and are often transient or reversible. These include sepsis, fever, bleeding, disseminated intravascular coagulation (DIC), splenomegaly, medications such as amphotericin B, vancomycin, or heparin, bone marrow suppression, chemotherapy or radiotherapy, graft-versus-host disease, and poor platelet storage or ABO incompatibility. Immune causes, seen in about 20 to 40 percent of cases, are due to alloantibodies directed against HLA class I antigens, human platelet antigens (HPA), ABO antigens, or rarely, drug-dependent antibodies.

DR SUDHA SETHY
Head, Dept of Clinical Hematology, SCB Medical College, Cuttack, Odisha

The diagnosis of platelet refractoriness is primarily based on the Corrected Count Increment (CCI), which assesses the rise in platelet count

A stepwise diagnostic approach is recommended. First, inadequate platelet increments should be confirmed by measuring post-transfusion counts at one and 24 hours. Next, non-immune factors must be ruled out by assessing for infection, fever, splenomegaly, bleeding, DIC, or concurrent medications. If these are excluded, immune causes should be investigated using HLA antibody screening, HPA antibody testing, and platelet crossmatching to identify compatible donors.

Management focuses on addressing the underlying cause. Treating infections, controlling bleeding, stopping causative drugs, and optimizing transfusion timing are key in non-immune cases. For immune-mediated refractoriness, the best options are HLA-matched or crossmatch-compatible platelets. HPA-matched and ABO-identical platelets may also be used when available. In severe or refractory cases, therapies like intravenous immunoglobulin, immunosuppressive treatment, or continuous platelet infusion can be considered. Preventive strategies such as minimizing unnecessary transfusions, using leukoreduced platelet products, and preferring single-donor apheresis platelets help reduce alloimmunization and recurrence.

As platelet refractoriness is a multifactorial condition four steps are essential (a) Confirm inadequate CCI (b) Exclude non-immune causes (c) Identify immune causes and provide matched platelets, and (4) Use preventive measures like leukoreduction to reduce recurrence.

CONFERENCE GLIMPSES

Neutropenic fever: Key role of nurses in tackling complication

NEUTROPENIC fever is a critical condition in patients with low neutrophil counts, often seen in haematology patients undergoing chemotherapy or those with haematological malignancies. Understanding its implications is essential for providing effective care and to reduce morbidity and mortality.

The field of neutropenic fever is becoming more bumpy for every hematologists and hemato-oncologists due to MDR organisms, atypical infections esp viral and fungal with novel immunotherapies and alternative modes of HSCT and also due to co-existing non infectious inflammatory conditions like CRS/ICANS etc.

DR AVINASH KUMAR SINGH
PHMRI and Patna Hematology Clinic, Patna, Bihar

Nurses play a vital role in identifying, managing, and preventing complications arising from neutropenic fever. Neutropenic fever is defined as the occurrence of a fever with a body temperature of 38.3°C or higher in a patient whose absolute neutrophil count is less than 500 cells per cubic millimetre. It is a medical emergency commonly observed in patients receiving chemotherapy, radiation therapy, or undergoing bone marrow or stem cell transplantation. The condition results from the body's decreased ability to fight infections due to low neutrophil levels.

The most frequent causes of neutropenic fever include chemotherapy- or radiation-induced myelosuppression, cytopenia following hematopoietic stem cell transplantation or other cellular therapies, and bone marrow infiltration by malignant cells. It may also occur in patients with bone marrow failure syndromes or certain autoimmune disorders that suppress marrow function. Risk factors such as recent chemotherapy or radiation therapy, prolonged neutropenia (especially with counts below 100 cells/mm³), and the use of central venous catheters significantly increase vulnerability to infection.

In assessing patients, nurses must monitor vital signs frequently and observe for early indicators of infection. A thorough physical examination is essential to detect potential infection sites, including the skin, respiratory tract, and urinary system. Management involves initiating empirical broad-spectrum antibiotics within the first hour of fever onset, as any delay can lead to rapid deterioration. Nurses should also help evaluate the need for growth factors such as granulocyte-colony stimulating factor (G-CSF) to promote neutrophil recovery.

Education and collaboration are equally important.

ISHBT-EHA finding new ways to tackle hemoglobinopathies

HEMATOLOGISTS of repute from India and Europe captivated an audience of hematology scholars with their in-depth research presentations on hemoglobinopathies during a joint session on thalassemia, organized by the European Hematology Association and the Indian Society of Hematology and Blood Transfusion (ISHBT) during Haematocon-2025 in Lucknow on Saturday.

Deliberating on 'Diagnosis of Hemoglobinopathies – The Indian Perspective', Tuphan Kanti Dolai, secretary ISHBT, said hemoglobinopathies constitute one of the most common inherited disorders in India, posing a major public health challenge due to their high prevalence and genetic diversity.

India is home to nearly 10 distinct types of hemoglobinopathies, with varying regional and ethnic distribution patterns, he said. Prof Dolai said thalassemias and hemoglobinopathies differ in their genetic defects and clinical manifestations while thalassemias are caused by a quantitative defect, hemoglobinopathies result from qualitative defects. The prevalence of hemoglobinopathies in India is significant with varying degree, Beta-Thalassemia (2.9–4.6%), Sickle Cell Disease (5–40%), HbE diseases (3–50%), Hemoglobin D (2%), Alpha-Thalassemia (3–18%)

Each year, approximately 12,500 children are born with thalassemia, and the estimated affected population exceeds 1.5 lakh. The projected thalassemia population in India could reach 2.75 lakh by 2026, with one child born every hour with the disorder. Accurate diagnosis forms the cornerstone of prevention and management. Various methods for detecting hemoglobinopathies include Hemoglobin electrophoresis, High-Performance Liquid Chromatography, Capillary Zone Electrophoresis, Molecular methods and point-of-care testing.

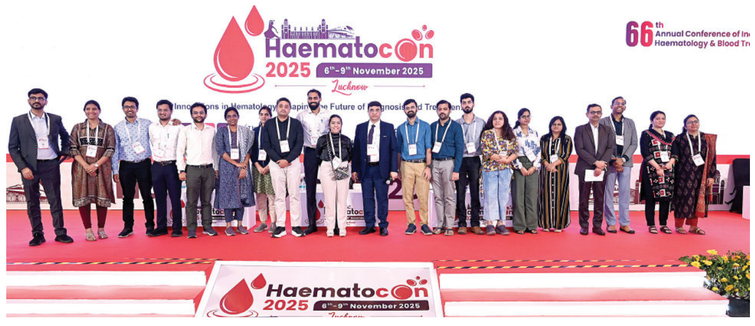
Presenting an 'Overview of Pathophysiology and Management of Beta Thalassemia,' Dr. Miguel R. Abboud from the American University of Beirut, Lebanon, highlighted the evolving therapeutic landscape for the disorder.

Dr. Abboud noted that blood transfusions and iron chelation continue to remain the cornerstone of thalassemia management. Bone marrow transplantation, he said, plays a major role in treating beta thalassemia and is now being expanded to include haploidentical donors, widening the donor pool.

Dr. Abboud also emphasized the promise of gene therapy and gene-editing technologies, which have shown highly successful outcomes, though accessibility remains a challenge in many regions. The introduction of novel agents such as Mitapivat and Luspatercept has significantly improved hemoglobin levels in non–transfusion-dependent thalassemia (NTDT) and reduced transfusion requirements in transfusion-dependent thalassemia (TDT). He added that several other promising agents are currently in active clinical trials, signaling a hopeful future for patients with beta thalassemia.

Similarly, Erfan Nur, Consultant hematologist and transplant physician of Amsterdam UMC, Sanquin Research, made his presentation titled 'Allogenic HCT in hemoglobinopathies, Whom, when and how to transplant' said Hematopoietic cell transplantation (HCT) is guided by the severity of the disease, with myeloablative conditioning regimens remaining the standard approach for children, yielding good event-free and overall survival rates.

Serotherapy, an important component of rejection prophylaxis, is widely accepted, using agents such as antithymocyte globulin (ATG) or alemtuzumab. However, outcomes differ with age - adolescents and adults experience higher mortality following myeloablative regimens, said Dr. Nur. ISHBT President elect Brigadier Dr Tathagat Chatterjee and prominent hematologist Dr Deepak Mishra moderated the session.



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Laboratory approach to hemolytic anemia

THE laboratory approach to hemolytic anemia begins with confirming that hemolysis, or premature destruction of red blood cells, is truly occurring. This confirmation is based on two broad types of evidence: signs of increased red cell destruction and the bone marrow's compensatory response. Laboratory findings such as elevated serum lactate dehydrogenase (LDH) and increased unconjugated bilirubin suggest heightened red cell breakdown. A marked decrease in serum haptoglobin is a highly specific indicator, as haptoglobin binds to free hemoglobin released during cell destruction. In cases of intravascular hemolysis, urine may show the presence of hemoglobin or hemosiderin, reflecting ongoing or chronic red cell destruction.

PROF SHAILAJA SHUKLA
Lady Hardinge Medical College, New Delhi

The bone marrow responds to hemolysis by increasing red cell production, which is seen as a raised reticulocyte count or a high reticulocyte production index. A peripheral blood smear provides further evidence, often showing polychromasia, which represents larger, bluish, immature red cells known as reticulocytes. Once hemolysis is established, the next step is to identify its underlying cause through targeted investigations. The complete blood count and peripheral smear are essential initial tools, as red cell morphology offers crucial diagnostic clues. The presence of spherocytes suggests autoimmune hemolytic anemia or hereditary spherocytosis, while fragmented red cells, or schistocytes, indicate microangiopathic hemolytic anemia such as thrombotic thrombocytopenic purpura, hemolytic uremic syndrome, or disseminated intravascular coagulation. Bite cells and Heinz bodies point toward oxidative injury, as seen in glucose-6-phosphate dehydrogenase (G6PD) deficiency, and sickle cells are diagnostic of sickle cell disease. Target cells, although non-specific, may be seen in hemoglobinopathies like thalassemia.

The direct antiglobulin test (Coombs test) is a pivotal investigation that distinguishes immune-mediated hemolysis from non-immune causes. A positive test indicates antibodies or complement coating the red cells, as seen in autoimmune hemolytic anemia or transfusion reactions. A negative result points to intrinsic defects such as membrane or enzyme abnormalities, or mechanical destruction. Subsequent investigations are guided by these findings; tests like osmotic fragility or EMA binding for membranopathies, G6PD assays for enzymopathies, hemoglobin electrophoresis for hemoglobinopathies, and ADAMTS13 or coagulation studies for microangiopathic processes. For suspected paroxysmal nocturnal hemoglobinuria, flow cytometry for CD55 and CD59 is confirmatory.

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How should we approach bone marrow metastasis

BONE marrow is a blood rich soft connective tissue in the cancellous space of bone and cavity of long bone marrow and is an essential source of hematopoietic cell production. The rich blood supply of marrow provides favorable conditions for tumor cell proliferation and growth. It is one of common organs to be involved by tumors that metastasize via blood stream.

Term 'Metastasis' was first recorded in Greek writing in 1580 AD,means change of place, order, nature of cells (migration or transition). Metastasis refers to ability to leave a primary tumor through circulation toward the distant tissue and form a secondary tumor the tumor cells interact with bone marrow microenvironment to survive and grow, this microenvironment is called "Metastatic niche: An environment rich in growth factors, cytokines. chemokines and signaling molecule for survival and growth of tumor cell is provided by metastatic niche. This is called Paget "Seed and Soil" theory and states that tumor metastasis entails a series of interactions between the tumor cells and stromal cells. Disrupting these reactions can serve as therapeutic intervention for bone metastasis.

In adult tumor metastasizing to bone marrow are CA breast, Lung, prostate, kidney, thyroid. In children neuroblastoma, rhabdomyosarcoma, Ewing's o sarcoma, Wilm's tumor and germ cell are tumors metastasizing in marrow. Once marrow metastasis is detected, the survival rates drastically declines.

DR SAVITRI SINGH
King George Medical University, Lucknow

> The vast growth in research on metastasis in the past decade has yielded an unprecedented wealth of information on the intrinsic and extrinsic tumor mechanism

Clinical presentation in suspected marrow metastasis is unexplained anemia, hemocytopenia, fever, bone pain, abnormality on imaging studies, pathological fracture, hypercalcemia and also may be any combination of these features. For diagnosis clinical, radiological investigation, CBC, PS, biochemical parameter,bilateral bone marrow aspirate/ bone marrow biopsy with IHC, and ancillary test such as cytogenetic, molecular tests are mandatory to pick up early deposit in marrow. The diagnosis of metastatic involvement of bone marrow has profound effect on prognosis and treatment. Accurate, diagnose of tumor metastasis requires integration of clinical, imaging, laboratory findings, results of IHC cytogenetics and molecular studies.

The vast growth in research on metastasis in the past decade has yielded an unprecedented wealth of information on the intrinsic and extrinsic tumor mechanism determining the metastatic behavior. However, integrating and applying new knowledge-oriented development of metastatic-oriented anticancer drugs are required to thwart the development of metastatic disease at any stage of |development.

CONFERENCE GLIMPSES

Understanding ICANS: A major challenge

IMMUNE Effector Cell–Associated Neurotoxicity Syndrome (ICANS) is a common complication of Chimeric Antigen Receptor T-cell (CAR-T cell) therapy and, less frequently, of bispecific antibody therapy. It is a neuropsychiatric syndrome that typically occurs due to cytokine-mediated inflammation following cytokine release syndrome (CRS), although it may also occur independently of CRS.

PROF UDAY POPAT
Department of Stem Cell Transplantation, The University of Texas

ICANS most often develops between days three and 10 after cell infusion. The reported incidence ranges from 20% to 70%, depending on the CAR-T product used, with severe cases occurring in 20–30% of patients. It is more frequent with CAR-T constructs utilizing CD28 as a costimulatory domain and is particularly associated with Axicabtageneciloleucel and Brexucabtageneautoleucel. Risk factors include higher CAR-T cell doses or peak expansion, greater disease burden, and preexisting neurological comorbidities.

The exact pathophysiology of ICANS remains incompletely understood. It is thought to result from endothelial activation and blood–brain barrier disruption due to elevated cytokine levels. This increased endothelial permeability leads to cerebral edema and associated neurotoxicity.

Clinical manifestations typically include encephalopathy with altered mental status and varying levels of consciousness, up to coma. Other features may include hallucinations (visual or auditory), seizures, aphasia, agraphia, tremor, and headache.

Laboratory findings often reveal cytopenias, elevated inflammatory markers, and high circulating levels of proinflammatory cytokines. Neuroimaging may demonstrate cerebral edema, and EEG frequently shows diffuse slowing consistent with encephalopathy, occasionally with seizure activity.

Management involves corticosteroids and supportive care, including seizure prophylaxis and measures to reduce cerebral edema. With appropriate treatment, symptoms typically resolve within 7–10 days of onset.

SCD: Urgent unmet need for spinal fractures

SICKLE cell disease (SCD) is associated with spinal pathologies including vertebral fractures and infections. The pathophysiology of spinal fractures is speculated to be multifactorial, including ischemic insult due to microvascular infarction; compensatory medullary expansion and cortical thinning due to anemia; and low bone density due to malnutrition.

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These conditions may occur starting in childhood and cause substantial morbidity throughout life. New-onset fractures cause acute pain, which may frequently be masked by multifocal pain experienced during recurrent vaso-occlusive crises. It is common to have multiple spinal fractures, which can contribute to chronic pain through a combination of bone pain, spinal deformity and central sensitization.

Despite the known elevated risk, spinal fractures remain under-diagnosed among individuals living with SCD. Additionally, when the diagnosis of spinal fractures is delayed, the treatment window is missed for interventions such as vertebral cement augmentation that could reduce pain.

There is an urgent unmet need to investigate the pathophysiology and epidemiology of spinal involvement in SCD. This effort should start with developing safe and cost-effective imaging modalities such as low-dose x-rays for early detection of spinal involvement. Evidence should be collected from large cohorts to develop clinical guidelines for screening and diagnosis.

In practice, clinicians must have a high degree of suspicion for spinal pathologies starting in early life. Multidisciplinary care is critical; spinal specialists should be involved in the care of SCD patients. Bone health should also be emphasized through nutrition optimization and bone density screening, guided by primary care physicians, endocrinologists and nutrition specialists.

Primary amyloidosis: treatment and challenges

AL amyloidosis is a complex, multisystem disorder caused by misfolded immunoglobulin light chains produced by clonal plasma cells. These misfolded proteins aggregate into amyloid fibrils, depositing in organs and leading to dysfunction. Accurate diagnosis, typing, and risk stratification are essential for effective management.

The disease originates from a plasma cell clone producing unstable light chains. These undergo misfolding and aggregation into amyloid fibrils. Other contributors include serum amyloid A, SAP, GAGs, and transthyretin. Importantly, not all amyloidosis is AL or ATTR; hereditary variants and ALECT2 must be considered.

Correct amyloid typing is critical for diagnosis. For example, 29% of patients with ATTR amyloidosis may have incidental MGUS, leading to misclassification. ALECT2, the third most common acquired amyloidosis, predominantly affects renal function and is prevalent in Indian, Mexican, and Middle Eastern populations. Diagnostic tools include imaging (CMR, PET/CT, SAP scans), biomarkers (NT-proBNP, FLC), and novel assays like AmyLite™, which detects λ amyloid FLC with high specificity.

Outcomes in AL amyloidosis depend on organ involvement, clonal characteristics, and treatment response. Key prognostic indicators include renal staging (based on eGFR and proteinuria), cardiac markers (GLS, NT-proBNP, CMR-derived ECV), bone marrow plasma cell burden and cytogenetics (e.g., t(11;14), 1q gain)and functional status (SBP, 6-minute walk test, frailty)

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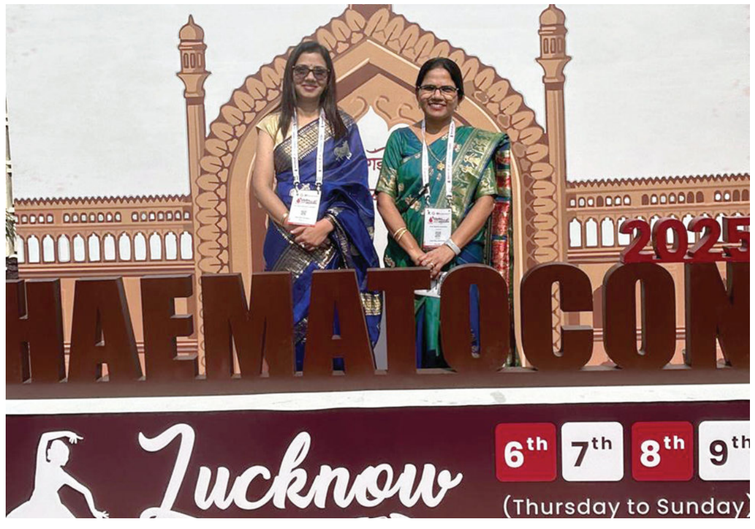
> AL amyloidosis requires a multidisciplinary approach that integrates precise diagnosis, risk-adapted therapy, and vigilant supportive care

The frontline regimen is daratumumab combined with bortezomib, cyclophosphamide, and dexamethasone (D-VCd), as per the ANDROMEDA trial. For patients with advanced cardiac disease (Stage IIIb/c), daratumumab monotherapy or dose-adjusted regimens are preferred. ASCT retains a role in selected high-risk patients, especially those with early relapses or adverse cytogenetics.

Relapse management depends on prior response, cytogenetics, and organ progression. Options include retreatment with daratumumab-based regimens (DVD, DRD, DPD), venetoclax for t(11;14) translocation, IMiD and PI-based combinations (RD, IRD, KD), ASCT for eligible patients.

Novel agents under investigation include bispecific antibodies (Teclistamab, Elrantamab), CAR-T therapies (NXC-201, FKC-288), anti-fibril monoclonal antibodies (CAEL-101, NEOD001), Light chain stabilizers.

These aim to eliminate plasma cell clones, remove soluble aggregates, and clear amyloid deposits. Achieving hematologic CR or near CR is the primary goal. FLC mass spectrometry (FLC-MS) negativity correlates with improved survival. MRD negativity is emerging as a new benchmark. AL amyloidosis requires a multidisciplinary approach that integrates precise diagnosis, risk-adapted therapy, and vigilant supportive care. D-VCd remains the cornerstone of frontline treatment.



CONFERENCE GLIMPSES

When science meets sincerity: The secret to a successful meet

A great medical conference is not just an event, it's a living experience bringing science, collaboration, and camaraderie together. A successful conference rests on meticulous planning, strong partnerships, and a personal touch at every stage.

Vision and Early Planning: Work begins a year in advance. Define a theme that captures current scientific priorities. Key elements include budgeting expenses and income, driving financial support, designing the scientific program, publicity, and hospitality. For larger conferences, early team alignment ensures every committee works in synergy toward one vision.

RAJESH SHARMA
Director, MICE Ideas Pvt. Ltd. and Nucleus

Pre-conference Collaboration and Visibility

Pre-conference visibility for sponsors is vital. Industry partners today seek meaningful engagement through academic symposia, faculty collaborations, exhibition booths, product launches, and online sessions. Regular networking among organizers, sponsors, and faculty sustains momentum and clarity.

Academic Value and CME Accreditation: Publishing the full scientific program three months in advance helps delegates plan travel and schedules, ensuring higher participation. Apply for CME accreditation early and feature it prominently.

Partnering with a PCO: A capable PCO shoulders operational workload, allowing the committee to balance professional practice, family, and duties. The right partner ensures precision, continuity, and a stress-free experience throughout the year.

Onsite Experience : Onsite efficiency including smooth registration, punctual sessions, helpful volunteers, and clear signage defines participant satisfaction. Keep delegates engaged with live polling, case-based discussions, and interactive Q&As. Post-event gratitude, highlights, certificates, and feedback sustain relationships and improvement.

Gastronomy: Food reflects hospitality. Plan menus to suit attendee mix, regional tastes, and dietary sensitivities. Thoughtful gastronomy turns breaks into opportunities for networking and rejuvenation.

Post-Conference Connection: After the applause fades, relationships remain. Share highlights, recordings, and feedback promptly. Gratitude builds trust for future editions.

Extending the Conference Beyond Its Walls

A conference's true impact lies in public benefit. Share key outcomes: advances, health messages, and insights through simple, accessible media. It reflects transparency, social responsibility, and the humane purpose of medicine.

"Conferences succeed when science meets sincerity, and every participant feels seen, valued, and inspired."

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WALKATHON RAISES AWARENESS ON BLOOD DISORDERS AT HAEMATOCON-2025

LUCKNOW witnessed a spirited Walkathon on Saturday morning as part of Haematocon-2025, the annual conference of the Indian Society of Hematology and Blood Transfusion. The event aimed to raise public awareness about blood disorders, including thalassemia, haemophilia, sickle cell disease and blood cancers.

The Walkathon started from the Chancellor Club and saw enthusiastic participation from leading hematologists across India, international experts, students, and members of various patient groups. Among them were representatives from thalassemia and haemophilia patient associations, who walked alongside doctors and researchers to spread the message of hope and awareness.

The Walkathon was led by Haematocon-2025 Organising Secretary Prof SP Verma, Co-Organising Secretary Prof Rashmi Kushwaha in the presence of ISHBT President Sarmila Chandra, President (Elect) Brig Dr Tathagata Chatterjee, ISHBT Secretary Prof Tuphan Kanti Dolai, Prof Manoranjan Mohapatra, Dean ICHProf HP Pati, Secretary ICH Prof RK Jena and other delegates of the conference.

Participants carried placards and banners with messages promoting early diagnosis, blood donation, and access to advanced treatments. The walk also served to remind the public of the growing burden of blood-related diseases and the need for greater attention to hematological care in hospitals.

Speaking at the event, senior doctors said that improving awareness is key to early detection and better outcomes for patients. They stressed the importance of ensuring that modern treatments for blood disorders and cancers reach people in every part of the country, not just big cities.

The Walkathon also highlighted the commitment of the hematology community to strengthen healthcare infrastructure and make treatment more affordable and accessible. Organizers said the event symbolized unity between medical professionals, patients, and caregivers in their shared fight against blood diseases.

As the Walkathon concluded, participants expressed optimism that such initiatives will help build a stronger understanding of blood health among the public. The event captured the spirit of Haematocon-2025—to advance hematology through awareness, collaboration, and compassion.

The Lucknow Haematocon!

DR MINATI RATH, MD
Obstetrics & Gynecology

Here comes our 2025, 66th Haematocon!
Our pride, the Indian Society of Haematology and Blood Transfusion
Focus this year is on the buzzword 'Innovation'!
Which can together strengthen the very foundation
Welcome to Lucknow, the Nawab's city of our nation!

With dedicated hematologits' active participation
ISHBT underwent tremendous expansion
Including molecular, genetic and immunological dimension
Hematological disorders now treated with precision
Hematologists updating themselves through international collaboration
ICH, the academic wing, honouring members providing valuable contribution
Indian Journal of Haematology & Blood Transfusion nurturing scientific aspiration

Delegates to explore Lucknow's rich culture and tradition
A blend of Hindu, Islamic, Persian and European contribution
A mesmerising architecture, depicting elaborate decoration
Not to ignore, the 'etiquette' & 'shayari', so dear to its population
To relish the delicious galauti kebabs, biriyani & malai makhan
What to say about the famous Lucknowi chikan

And last but not the least...
As the very word 'Luck-now' says, Good luck to our
Lucknow Haematocon!