Members of the Malaysian Laboratory Haematology Society (MMLHS) Scientific Meeting & Morphology Workshop 2025 held on 26th-28th August 2025 at Kuala Terengganu, Terengganu.

SPEAKERS' ABSTRACTS

Microscopic Haematology: A Practical Guide on Red Cell Disorders

Farisyah Syakirah Mohd Zain
Haematology Unit, Department of Pathology, Hospital Seberang Jaya, Ministry of Health Malaysia, Penang, Malaysia

Peripheral blood smear examination remains an indispensable, rapid and cost-effective diagnostic tool in modern haematology, particularly in acute inpatient settings. When performed in laboratories equipped with a fully automated Full Blood Count (FBC) analyser, alongside competent smear preparation and staining facilities, it allows for timely morphological assessment that may unveil vital red cell abnormalities. These red cell changes, whether isolated or occurring alongside leukocyte or platelet anomalies, frequently serve as critical early indicators that prompt further targeted investigations, ultimately contributing to accurate diagnosis and optimal clinical management. Given its diagnostic value, it is imperative that all phases of laboratory workflow including preanalytical, analytical and postanalytical steps are executed with precision to ensure reliable interpretation and reporting. This session will specifically highlight red cell morphologies indicative of haemolysis, an urgent and often life-threatening condition demanding immediate recognition and prioritization by the haematology laboratory team. Prompt identification of haemolytic features such as schistocytes, spherocytes, polychromasia or red cell fragmentation can be pivotal in guiding emergent clinical decisions. Thus, the vigilance and competency of every member in the haematology unit are paramount. Early morphological clues from a peripheral smear not only support clinical correlation but can be life-saving when dealing with rapidly evolving haematological emergencies.

Application of Digital Imaging in Routine Haematology Laboratory

Nor Ayuni Khuzaimah
Haematology Unit, Pathology Department, Hospital Pulau Pinang, Ministry of Health, Penang, Malaysia

Since the early 1990s, automated digital microscopy systems and digital slide technology have increasingly become vital components of haematology laboratories. These advancements facilitate the electronic capture, storage, and analysis of blood cell images, thereby improving diagnostic accuracy and operational efficiency. Over time, these technologies have advanced to become more sophisticated, user-friendly, and well-integrated into routine laboratory workflows. Currently, digital imaging is widely utilised worldwide in haematology labs for automated differential counts, morphological evaluations, and telemedicine consultations. This session will share the experiences of the Haematology Unit at Hospital Pulau Pinang, including an assessment of digital microscopy performance, its reliability within our laboratory setting, and key insights gained. Additionally, the *Discussion* will cover the limitations and challenges faced during the implementation process.

Morphological Insights into Paediatric Inherited Anaemias and Bone Marrow Failure Syndromes

Noraesah Mahmud
Haematology Unit, Pathology Department, Hospital Kuala Lumpur; [3]Haematology Unit, Medical Department, Hospital Kuala Lumpur

Inherited anaemias and bone marrow failure syndromes (IBMFS) in children represent a broad and heterogeneous group of haematological disorders, often presenting with overlapping clinical and laboratory features. In settings where access to advanced molecular diagnostics is limited, morphological assessment plays a crucial role in the early identification and characterisation of these conditions. This session focuses on peripheral blood and bone marrow morphological features that serve as critical diagnostic clues in selected inherited anaemias and marrow patterns commonly observed in IBMFS. Case examples will include both commonly seen and rare morphological presentations. The importance of integrating morphological findings with clinical context, family history, and basic laboratory data in establishing a diagnosis will be highlighted. Early recognition of these cases not only facilitates timely and appropriate patient management and referrals but also supports genetic counselling and long-term care planning for affected families.

Role of Morphology in the Diagnosis of Acute Leukaemia Based on WHO classification

Rosline Hassan
Department of Haematology, School of Medical Sciences, Universiti Sains Malaysia, Kubang Kerian, Kelantan

Flow-cytometry, cytogenetic, and molecular tests are being increasingly used in making the diagnosis of Acute Leukaemia (AL). However, morphological analysis of blood cells is still a crucial diagnostic aid. The information provided by the morphological analysis of the blood cells is relevant upfront to support the diagnosis of the patients with AL. WHO-HAEM5 classification, published in 2022, takes a genetic-centric approach and recognises the importance of genetic alterations. It provides a refined framework for characterising haematological malignancies. It illustrates the diagnostic utility of morphology, flow cytometry, immunohistochemistry and next-generation sequencing in resource-rich settings. However, its implementation in low and middle-income countries remains challenging due to limited access to advanced diagnostic tools. Nevertheless, morphology is still the fundamental requirement in the application of the WHO-HAEM5 classification. Quantitative measurement of blast cell counts either in peripheral blood or bone marrow and characterising the morphology of blast cells, will discriminate myeloblast or lymphoblast. Blast cell counts of >5% in bone marrow or > 2% in blood should proceed with genetic testing to qualify the category of AML-defining genetic lesion. For ALL, diagnosis is established by the presence of at least 20% lymphoblast in the bone marrow or peripheral blood. Role of performing morphology examination is required upfront in making diagnosis and classification of AL. Currently, the morphology examination is performed using light microscopic examination. New haematology analyser is currently under development using neural networks based on the morphology features of the blood cells.

Utilisation of Molecular Testing in Ph Negative Myeloproliferative Neoplasm (MPN), Myelodysplastic Neoplasm (MDS) and MDS/MPN overlap syndromes

Siti Shahrum Muhamed Said
Department of Pathology, Hospital Tunku Azizah, Ministry of Health Malaysia, Kuala Lumpur.

MPN, MDS and MDS/MPN overlap syndromes represent a biologically diverse group of myeloid malignancies with significant genetic and clinical heterogeneity. The 5th edition of the WHO Classification of Haematolymphoid Tumours (WHO-HAEM5) underscores the integration of molecular, morphological and clinical findings for accurate diagnosis, risk stratification and treatment decision. In Ph-negative MPNs such as polycythaemia vera (PV), essential thrombocythemia (ET), and primary myelofibrosis (PMF), driver mutations in *JAK2, CALR,* or *MPL* are present in most cases. However, 10–15% of patients, particularly those with PMF or ET, may be triple-negative, lacking these canonical mutations. In such instances, expanded molecular profiling is imperative. Somatic mutations in non-driver genes such as *ASXL1, EZH2, SRSF2,* and *U2AF1* are frequently observed and serve as indicators of clonality, assist in distinguishing from reactive conditions and carry independent prognostic significance. WHO-HAEM5 also redefines MDS by introducing categories with defining molecular alterations, including MDS with biallelic *TP53* inactivation and MDS with *SF3B1* mutation. MDS/MPN overlap syndromes display both proliferative and dysplastic features and frequently harbour mutations in genes related to splicing, epigenetic regulation, and RAS pathway signalling. Molecular testing including next-generation sequencing (NGS) has become central to the diagnostic workup of myeloid neoplasms, enabling the detection of driver and co-occurring mutations that refine classification, risk stratification, and influence treatment. More recent prognostic models such as MIPSS70v2, MIPSS-PV and the IPSS-M have incorporated somatic mutation profiles alongside cytogenetic and clinical data to better predict outcomes and guide therapeutic interventions. In conclusion, molecular testing is now indispensable in the era of precision medicine, offering critical insights across the continuum of MPN, MDS, and MDS/MPN disorders.

Morphology Review: Differentiating Between Benign and Malignant

Mimi Azreen Abdullah
Haematology unit, Pathology Department, Hospital Raja Perempuan Zainab II 15586 Kota Bharu Kelantan

Peripheral blood smear review remains a vital diagnostic tool, complementing automated full blood count (FBC) analysis in the detection of haematologic abnormalities. Accurate interpretation of peripheral blood smears remains a cornerstone in the diagnosis and monitoring of haematological disorders. Thus, it is important to recognise and distinguish reactive (benign) changes from malignant haematologic features. Starting with the interpretation of FBC analysers flags and scattergrams, followed by criteria for initiating blood smear reviews, it is important to know the key morphological features that differentiate reactive cells from malignant counterparts, to differentiate them from features seen in malignant processes like leukaemia and lymphoma. Applying this knowledge in practical scenarios is crucial for timely and accurate morphological evaluation, ultimately helping in patient management.

Myeloproliferative Neoplasms (MPN) – Predicting progression to acute leukaemia

Wendy N. Erber
School of Biomedical Sciences, The University of Western Australia, Australia

Myeloproliferative neoplasms are acquired clonal haemopoietic stem cell disorders which result in increased production of blood cells. Patients are at risk of transforming to secondary acute myeloid leukaemia, with this being highest in those living with myelofibrosis. The process of leukaemia transformation progresses through phases marked by an increase in the number of "blast" cells from the "accelerated" to "blast" phase when the 20% threshold is reached. This progression is unpredictable, and clinicians are reliant on patient's symptoms and signs, blood count alterations and the emergence of blast cells. Current tests lack the requisite sensitivity, specificity or practicality to identify these changes or use them for predictive purposes in routine practice. CD34-positive cells can be assessed by immuno-histochemical staining of bone marrow biopsies, to identify increased numbers of paratrabecular cells and interstitial clusters. Flow cytometry has been used to measure circulating CD34-positive cells. When the number increases to $>15x10^6$/L, it is indicative leukaemic progression. Cytogenetic alterations accumulate with disease acceleration and new somatic mutations (e.g. *ASXL1*, *IDH1/2*, *RAS*, *RUNX1*, *TP53*) are acquired at this progressive stage leading to transformation to overt leukaemia. Despite this, prediction remains challenging. New modalities are required, and some will be discussed.

The Monocytosis Dilemma - Differentiating Benign from Malignant

Wendy N. Erber
School of Biomedical Sciences, The University of Western Australia, Australia

Monocytes are mononuclear of the mononuclear phagocyte system that originate from bone marrow progenitor cells. Their main functions are phagocytosis, cytokine production and antigen presentation. Monocytes have roles in responding to infections and inflammatory conditions. The reference counts are 0.2-0.8 $x10^9$/L (adults) and 0.7-1.5 $x10^9$/L (children), constituting 2-8% of circulating leucocytes. Monocytes are defined by morphological criteria: size, cytoplasm amount and colour, granules, nuclear shape, nucleolus and chromatin condensation. These characteristics distinguish monoblasts, promonocytes, immature monocytes and monocytes. Cellular antigens (especially CD14, CD64, CD68) vary in their expression during the differentiation stages and can be assessed by flow cytometry and immunohistochemistry. Reactive monocytosis can be transient when secondary to acute viral infections, or persistent with chronic infections (e.g. tuberculosis) or inflammatory conditions (e.g. autoimmune diseases), solid tumours and non-myeloid haematological neoplasms, smoking and medications. Clonal monocytosis is an unregulated and uncontrolled expansion and proliferation of the monocytic mitotic compartment in the marrow, thereby increasing the absolute monocyte count and leading to morphological alterations. Malignancies involving monocytes include juvenile and chronic myelomonocytic leukaemia, acute myeloid leukaemia's, chronic myeloid leukaemia and histiocytic sarcomas. The distinction between benign and malignant can be challenging and may also require assessment of genes for acquired mutations.

Quality Assurance in Haematology: Basic Requirement for Implementation

Ehram Jamian
Haematology Unit, Pathology Department, Hospital Sultanah Aminah (HSA), Ministry of Health Malaysia, Johor Bahru, Johor Darul Takzim,

The challenge of implementing Quality Assurance in Haemotology Laboratories is very high. Therefore, understanding the implementation process is very important. The relationship between the concepts of ISO 15189: 2022 in principle, the concept of 3M1D1E and the basic concepts of 6.2.2 Competence requirements, 6.2.3 Authorisation and 6.2.5 Personnel records need to be refined.

Quality Assurance in Haematology: Ensuring the Validity of Examination Results

Ehram Jamian
Haematology Unit, Pathology Department, Hospital Sultanah Aminah (HSA), Ministry of Health Malaysia, Johor Bahru, Johor Darul Takzim,

International standard ISO 15189 requires the laboratory to use test methods that are able to meet quality requirements and that are fit for the purpose of patient management. Implementation of monitoring the validity of *Results* is very important. The aim of this presentation is to aid the lab user for understanding the process of acceptance testing, method verification, monitoring of Internal Quality Control criteria at regular intervals, in a timeframe that allows a meaningful indication of current performance, measurement uncertainty and periodicity of comparison of examination, it include qualitative and quantitative test in Haematology. The process of understanding the implementation of these quality activities will be discussed practically.

Quality Assurance in Flow Cytometry

Ehram Jamian
Haematology Unit, Pathology Department, Hospital Sultanah Aminah (HSA), Ministry of Health Malaysia, Johor Bahru, Johor Darul Takzim,

Diagnostic flow cytometry has been established in laboratory diagnostics as an indispensable, deeply specialised method. Because

of the special needs of the analysis in vital cells, the *Introduction* and maintenance of quality management systems stays a challenge. Meanwhile, experience has been collected for quality controls and validation in diagnostic flow cytometry. This will be summarised in this presentation. The focus will be on the relevant rules and regulations for diagnostic laboratories and the associated requirements for MS ISO 15189: 2022. Factors influencing flow cytometry analyses, strategies for the standardisation and harmonisation in flow cytometry, and possibilities for verification and validation in the flow cytometry laboratory are given

Ensuring Quality in Haematological Qualitative Test

Mimi Azura Aziz
Haematology Laboratory, Pathology Department, Hospital Tunku Azizah, Kuala Lumpur

Key elements to ensure quality medical laboratory testing includes robust quality management system (LQM, PK, AK, TPM), highly trained and competent staff, strict adherence to standard operating procedures (SOPS), well-maintained equipment's and reagents, quality control measures including IQC & EQA, accurate record-keeping and document control as well as continuous monitoring and improvement processes which should be guided should by international standards- ISO 15189. To provide focus in this lecture, emphases are made on how to meet the Processes Requirements stated in Section 7 of MS ISO15189: 2022 with examples for qualitative haematological tests.

Diagnostic Challenges in Laboratory Testing for Lupus Anticoagulant

Suzana Zainol
Haematology Laboratory, Pathology Department, Hospital Tunku Azizah, Kuala Lumpur

Lupus anticoagulant (LA) testing is a key component in diagnosing antiphospholipid syndrome (APS), but obtaining accurate *Results* is challenging due to various factors. Testing should be limited to patients with clear clinical indications to avoid incidental findings and unnecessary interventions. Pre-analytical variables significantly influence test accuracy. These include the type of anticoagulant used, timing of specimen collection, and specimen handling. Testing during acute thrombosis, inflammation, or pregnancy may yield false *Results* due to transient physiological changes, while anticoagulant therapy may interfere with assay performance. Analytical challenges involve assay selection, reagent sensitivity, and specificity. Due to the heterogeneity of lupus anticoagulant, reliable detection requires two phospholipid-dependent assays based on different principles, following a standardised three-steps procedure (screen-mix-confirm). Interpretation requires determination of adequate cut-off values to determine positivity in all three steps. Nevertheless, circulating inhibitors, factor deficiencies, and anticoagulant use can still result in false positives or negatives, necessitating cautious interpretation. To ensure accurate diagnosis, the post-analytical phase of LA testing requires standardised reporting, clear interpretative comments, and awareness of assay limitations. It is best performed together with Anticardiolipin and Anti-β2-Glycoprotein 1 antibody testing to provide a comprehensive evaluation of antiphospholipid antibodies. *Results* should be interpreted in a clinical context with knowledge of ongoing treatment to avoid misdiagnosis and guide management. Strong communication between clinicians and laboratories further enhances interpretation and clinical decision-making. Ultimately, optimising LA testing demands a comprehensive approach that includes justified clinical indications, strict adherence to pre-analytical requirements, and standardised diagnostic procedures to minimize pitfalls and achieve reliable *Results*.

Pre-Analytical Errors in Coagulation Testing: Identification and Prevention

Ahmad Zakwan Mustafa
Department of Haematology, School of Medical Sciences, Universiti Sains Malaysia, Kelantan, Malaysia

Pre-analytical errors represent a significant source of variability and inaccuracy in coagulation testing, impacting both clinical outcomes and laboratory efficiency. This presentation explores the prevalence, types, and consequences of such errors, particularly within the context of the Haematology Laboratory at Hospital Pakar USM. Key areas addressed include improper patient preparation, sample collection inconsistencies, and suboptimal handling and transport protocols. Through real-world data and case examples, the session highlights how common errors—such as clotted samples, incorrect blood-to-anticoagulant ratios, and delayed processing—can lead to misdiagnosis, inappropriate treatment, and financial waste. Emphasis is placed on practical strategies for minimizing errors, including standardised protocols, adherence to CLSI guidelines, and robust quality control measures. The presentation concludes with a call for enhanced staff training, technological support, and institutional commitment to best practices in coagulation testing. This initiative aims to improve diagnostic accuracy, patient safety, and cost-effectiveness across healthcare settings.

Non-Hodgkin Lymphoma Diagnosis by Trephine Biopsy - Limitations and Pitfalls

Ahmad Toha Samsudin
Premier Integrated Labs, Gleneagles Kota Kinabalu, Sabah

The circumstances of bone marrow trephine aim to establish the diagnosis of cytopenia, staging of lymphoma, monitoring treatment response and rule out another disease. BMT is also being subjected in cases with unexplained organomegaly or mass lesion

inaccessible for biopsy. The findings of primary haemopoietic disease such as leukaemia, MPN, MDS or secondary metastatic deposit could explain the pathology behind those cytopenia or cytosis. However, the bone marrow with lymphoid infiltrate demands different twist in determining their significance. Lymphoid aggregates are seen in a minority of bone marrow biopsy specimens, and when present, their neoplastic nature is often apparent by morphologic evaluation. However, the distinction between benign and malignant aggregates can be a diagnostic challenge when there are multiple aggregates with no documented history of lymphoma. Florid lymphoid cell infiltrates are sometimes seen in association with benign conditions such as aging, autoimmune diseases, inflammatory conditions, and infectious disorders may mistakenly concluded as lymphoproliferative disorder. Conversely, subtle lymphoid cells frequently overlooked as non-specific infiltrate. Therefore, appreciating the topographical distribution and pattern of aggregates are equally important as the study of morphological details which in turn help to optimise the ancillary tests such as immunohistochemistry and molecular analysis. These two issues of differentiating benign and neoplastic lymphoid infiltrate in the marrow as well as pitfall in the pattern of bone marrow infiltration in lymphoproliferative disease will be discussed, emphasising on topography, pattern of infiltrate, and cytologic appearance before recommending the appropriate immunohistochemistry panels and/or molecular tests.

Method Verification on Qualitative Haematological Tests

Noor Hidayat Hussain
Department of Pathology, Hospital Sultan Idris Shah (HSIS) Serdang, Jalan Puchong, 43000 Kajang, Selangor, Malaysia

Qualitative haematological tests are used to detect the presence or absence of a substance or to evaluate cellular characteristics, such as morphology. These tests do not provide numerical *Results* but instead offer descriptive or qualitative interpretations such as "positive," "negative," "reactive," "non-reactive," "normal," or "abnormal." Qualitative analysis plays a crucial role in clinical laboratory testing, aiding in the screening, diagnosis, and confirmation of various diseases. Given the diversity and complexity of some of these tests, it is essential to implement structured verification strategies to ensure their accuracy and reliability. Regarding quality requirement, ISO 15189:2022 emphasises the need for method verification before using any examination methods. This verification ensures that the performance characteristics or specifications outlined by the manufacturer or method are met. It is essential that all validated methods undergo verification before they are implemented in practice. To ensure proper method verification, laboratories should develop a protocol based on established guidelines, such as the CLSI guideline EP12-A2 (User Protocol for Evaluation of Qualitative Performance, Approved Guideline-Second Edition). The guidance is provided for both precision and method-comparison studies. The protocol includes steps like a repeatability study, followed by a reproducibility study. Furthermore, a comparison study is carried out to evaluate the candidate method against an established comparative method. This study involves two types of data analysis: comparing known clinical diagnostics or unknown clinical diagnostics. This structured approach guarantees that qualitative methods provide accurate and reliable *Results* for clinical decision-making.

Tackling Panic Values in Laboratory Test Results

Mohd Nazri Hassan
Department of Haematology, School of Medical Sciences, Universiti Sains Malaysia, Kubang Kerian, Malaysia, Hospital Pakar Universiti Sains Malaysia, Kubang Kerian, Malaysia

Panic values also referred to as critical values, are laboratory *Results* that significantly deviate from the normal range and may indicate potentially life-threatening conditions. These values are pivotal in clinical decision-making and require immediate medical attention. The Malaysia standard MS ISO 15189:2022 emphasises the importance of reporting critical *Results* as part of effective panic value management. Panic values encompass both numerical data and descriptive reports across various laboratory disciplines, including haematology, clinical biochemistry, anatomic pathology, transfusion medicine, and medical microbiology. Thus, all the clinical laboratories play a vital role in identifying, verifying, and rapidly communicating these *Results* to ensure patient safety and support timely clinical decision-making. The key component to manage panic values effectively, laboratories should establish the procedure of handling panic value with a clear panic value list, integration of Laboratory Information Systems (LIS) to alert the clinician, clear communication protocols, escalation procedures, and thorough documentation. Proper documentation of all steps taken is equally important to ensure accountability and traceability. Additionally, emphasis should be placed on staff training, interdepartmental coordination, and promoting a culture of safety and accountability. These strategies collectively enhance patient safety by ensuring that critical laboratory findings lead to timely and appropriate clinical responses.

Enhancing Diagnostic Accuracy and Quality Practices in Haematology Through Interactive Education Quiz

Razan Hayati Zulkeflee
Department of Haematology, School of Medical Sciences, Universiti Sains Malaysia, Kubang Kerian, Kelantan

Diagnostic accuracy and quality assurance are fundamental to effective practice in laboratory haematology. This interactive quiz session is developed to reinforce essential knowledge, sharpen diagnostic proficiency, and promote understanding of quality standards among laboratory technologists, medical trainees, and related professionals. Designed as a dynamic, game-based learning experience using the Kahoot platform, this session aims to provide real-time feedback, encourage healthy competition, and support continuous learning through engaging and enjoyable methods. The quiz will explore seven essential focus areas: (1)

Quality Assurance in Haematology, (2) Morphological Review—highlighting the differentiation between benign and malignant features, (3) Flow Cytometry Quality Control, (4) Quality in Haematological Qualitative Testing, (5) Pre-Analytical Factors in Coagulation Testing, (6) Method Verification for Qualitative Tests, and (7) Management of Panic Values in clinical settings. This learning tool is intentionally crafted to be mobile-accessible, fast-paced, and enriched with current guideline references and case-based explanations. By incorporating competitive elements and visual learning aids, the session aims to make complex quality and diagnostic concepts more accessible and memorable. The broader goal of this educational initiative is to improve professional competency, ensure readiness in managing high-risk laboratory *Results*, and foster a culture of diagnostic precision and accountability.

Innovating the Malaysia Thalassaemia Diagnosis Code Beyond Laboratory Perspectives

Hafizah Hashim
Jabatan Patologi, Hospital Sultanah Bahiyah, Kedah, Malaysia

Thalassemia remains one of the most common genetic disorders in Malaysia, prompting significant national response. The National Thalassemia Control and Prevention Programme was launched in 2004, followed by a student screening initiative in 2016 targeting 16-year-olds. This screening aims to detect carriers early, raise awareness, among young generations reduce new cases. After five years, *Results* revealed that about 5.9% of Malaysians are thalassemia carriers. Accurate diagnosis has been hindered by fragmented data and inconsistent reporting. To address this, the Malaysia Thalassemia Diagnosis Code (MTDC) was introduced in 2012 to standardise laboratory reports and streamline diagnostic processes. Techniques such as Capillary Zone Electrophoresis (CZE) and High-Performance Liquid Chromatography (HPLC) are used for initial screening, while molecular analysis—including ARMS PCR, GAP PCR, RDB hybridization, Sanger sequencing, MLPA, and NGS—is applied to confirm α and β thalassemia cases. MTDC generated 44 codes for haemoglobin analysis and 174 diagnosis codes for α and β globin gene mutations. The MTDC initiative developed a national database, mapping genotype prevalence, setting cut-off values for α-thalassemia, and distinguishing carriers from disease cases. It has improved genetic counselling, enabled family screening, and made complex diagnoses more accessible. Beyond laboratory benefits, MTDC has strengthened healthcare communication and service delivery. Notably, data from the Malaysia Thalassemia Registry show a declining trend in thalassemia births since 2014, marking progress toward long-term disease control.

ABSTRACT

ORAL PRESENTATION

MW-OR01: Morphology Spectrum of Polycythaemia Vera Patients with Different *Janus kinase 2* Mutations

Nur Alia Najiehah Suhaimi[1], Nur Hasya Athirah Hanizam[2], Nik Norliza Nik Hassan[2], Mohamad Ros Sidek[3], Rosline Hassan[1], Zefarina Zulkafli[1], Faezahtul Arbaeyah Hussain[4], Sumaiyah Adzahar[5], Razan Hayati Zulkeflee[1]*
[1]*Department of Haematology, School of Medical Sciences, Universiti Sains Malaysia, Kubang Kerian, Kelantan,* [2]*Biomedicine Programme, School of Health Sciences, Universiti Sains Malaysia,* [3]*Human Genome Centre, School of Medical Sciences, Universiti Sains Malaysia Health Campus, Kota Bharu, Kelantan,* [4]*Department of Anatomic Pathology, School of Medical Sciences, Universiti Sains Malaysia, Kubang Kerian, Kelantan,* [5]*Department of Pathology and Medical Laboratory, Faculty of Medicine, Universiti Sultan Zainal Abidin, Kuala Terengganu, Terengganu*

Introduction: Polycythaemia vera (PV) is a myeloproliferative neoplasm driven by *JAK2* gene mutations in exon 14 (V617F) or exon 12, leading to distinct morphological and clinical features. This study examines the morphological characteristics of PV patients with *JAK2* V617F and *JAK2* exon 12 mutations. *Material and Methods:* The diagnosis of 10 PV cases were based on the WHO 2022 criteria and *JAK2* molecular status were retrieved. Trephine biopsies stained using Haematoxylin and Eosin (H&E) were obtained from the Haematology Laboratory of Hospital Pakar Universiti USM. Immunohistochemical staining for Glycophorin A and CD61, were performed for all patients to evaluate megakaryocytes and visualised erythroid lineage. Marrow fibrosis was assessed using the WHO three-tiered semi-quantitative grading system. Samples with a score 3 and above were further examined using Masson trichrome staining to identify new bone formation. *Results:* Bone marrow samples hyperplasia with panmyelosis, consistent with PV diagnosis. Both types of *JAK2* variants showed prominent proliferation in megakaryocytes, with *JAK2* V617F-positive showed pleomorphic megakaryocytes with loose-clustering and occasional clumping. *JAK2* exon 12 mutation displayed monomorphic megakaryocytes uniformly in small size and shape. In contrast, V617F cases exhibited pleomorphic megakaryocytes and presented a higher grade of fibrosis (grade 2-3) compared to exon 12 mutation (grade 0-1). *Discussion:* Distinct morphological differences in megakaryocytes and varying degrees of fibrosis in PV patients with different *JAK2* variants, highlighting megakaryocyte abnormalities as key drivers of fibrosis and disease severity, especially in *JAK2* V617F mutations.

MW-OR02: *JAK2*, *CALR*, and *MPL* Mutations in *BCR::ABL1*-Negative Myeloproliferative Neoplasms: Distribution and Impact on Complete Blood Count Profiles

Yetti Hernaningsih[1,2], Alvin Hartanto Kurniawan[3], Siprianus Ugroseno Yudho Bintoro[2,4]
[1]*Department of Clinical Pathology, Faculty of Medicine, Universitas Airlangga, Indonesia 60131;* [2]*Dr. Soetomo General Academic Hospital, Surabaya, Indonesia 60286;* [3]*Student of Specialist, Clinical Pathology Study Program, Faculty of Medicine, Universitas Airlangga, Indonesia 60131;* [4]*Department of Internal Medicine, Faculty of Medicine, Universitas Airlangga, Indonesia 60131*

Introduction: JAK2 V617F, *CALR*, and *MPL* mutations are the causes of myeloproliferative neoplasms (MPN). i.e polycythaemia vera (PV), essential thrombocythemia (ET) and primary myelofibrosis (PMF). Data on mutations in these three genes are still

limited in Indonesia. The aim of this study was to determine the distribution of mutations in patients with *BCR::ABL1*-negative myeloproliferative neoplasms, and to compare complete blood count (CBC) parameters between mutation groups. *Materials and Methods:* This study was conducted on adult patients at the Oncology Outpatient Department, Dr. Soetomo General Hospital, Indonesia. Patients with clinical, CBC and blood smear evaluation were suspected of having myeloproliferative neoplasms was drawn for venous blood specimen, then were put in EDTA tubes. The specimens were processed for DNA extraction, then were tested with qualitative PCR for *BCR::ABL1* to rule out chronic myelocytic leukaemia. When *BCR::ABL1* were negative, continued testing for *JAK2* V617F, *CALR*, and MPL using qualitative PCR. *Results:* A total of 42 NPM patients were identified, resulting in 34 samples positive for *JAK2* V617F, 5 samples positive for *CALR*, and 3 samples negative for three mutations. Of the 34 *JAK2* V617F-positive samples, 20 were consistent with PV, and the remaining 14 were consistent with ET. Five *CALR* mutations and triple-negative cases were consistent with ET. The white blood cell count (WBC) and mean platelet volume showed significant differences among the three mutations, with the highest count in *JAK2* V617F, followed by triple negative and *CALR* mutation. Meanwhile, the parameters of haemoglobin, red blood cell count, haematocrit, mean corpuscular volume, mean corpuscular haemoglobin, mean corpuscular haemoglobin concentration, platelet, and plateletcrit showed no significant differences between mutations ($p>0.05$). *Discussion:* The V617F mutation is the most common in *BCR::ABL1* negative MPN, followed by the *CALR* mutation. The highest WBC counts and MPV were found in the *JAK2* V617F, followed by triple negative then *CALR* mutation.

MW-OR03: *FCGR* Gene Polymorphisms in Immune Thrombocytopenia Patients

Nurul Izzah Abdul Razak[1], Muhamad Aidil Zahidin[1], Hisham Atan Edinur[2], Noor Haslina Mohd Noor[1]
[1]Department of Haemotology, School of Medical Sciences, Universiti Sains Malaysia Health Campus, Kubang Kerian, Kelantan, Malaysia, [2]Department of Forensic Sciences, School of Health Sciences, Universiti Sains Malaysia Health Campus, Kubang Kerian, Kelantan, Malaysia.

Introduction: Fc gamma receptors (FcγRs), encoded by the *FCGR* gene family, are critical mediators of antibody-dependent cellular cytotoxicity and phagocytosis. Genetic polymorphisms in these genes can affect IgG binding affinity, potentially influencing autoimmune disease susceptibility, severity, and treatment response, including in immune thrombocytopenia (ITP). Hence, this study aims to investigate the association between *FCGR* gene polymorphisms and susceptibility to ITP. *Materials and Methods:* A case-control study was conducted involving 30 patients diagnosed with ITP and 30 healthy controls. Genotyping of 12 selected *FCGR* gene polymorphisms was performed using PCR Allele Competitive Extension (PACE). Associations of genotypic distributions with clinical and treatment data were compared using chi-square tests. *Results:* Genotyping *Results* showed a significantly higher frequency of the *FCGR*2A homozygous HH genotype in controls compared to ITP patients (56.7% vs. 30.0%, $p=0.037$). Conversely, the *FCGR*3A heterozygous FV genotype in controls was significantly higher compared to patients (66.7% vs. 23.3%, $p=0.008$). However, no significant differences were found in the genotype frequencies of other *FCGR* variants ($p>0.05$). Further clinical analyses showed that patients with the *FCGR*3A 158FV genotype had significantly more severe thrombocytopenia compared to other genotypes ($p=0.024$). Additionally, a significant association was found between *FCGR*3A genotype and treatment type ($p=0.010$). Notably, immunosuppressants were more frequent in patients with mutant genotypes (83.3%) while patients with the homozygous wild-type genotype were more commonly treated with corticosteroids alone (50.0%) or remained untreated (33.3%). *Discussion:* Our findings suggest that *FCGR*2A and *FCGR*3A genes may have implications for clinical outcomes and treatment strategies, highlighting the importance of *FCGR* genotypes in ITP pathogenesis and management. Further research involving larger prospective studies is needed to confirm these associations and explore their clinical relevance.

MW-OR04: Early Mortality Predictors in AML Patients: Insights from a Single-Center Cohort Study

Mas Aditya Senaputra[1,2], Dian Ariningrum[1,2], Maria Immakulata Diah Pramudianti[2], Amri Muzzammil[1], Bastomy Eka Rezkita[1]
[1]Clinical Pathology Department, Faculty of Medicine, Universitas Sebelas Maret Surakarta, [2]Clinical Pathology Laboratory, Dr. Moewardi General Hospital, Surakarta

Introduction: Acute Myeloid Leukaemia (AML) is a haematological malignancy with high mortality rates. Early identification of prognostic factors is essential to improve patient management. Routinely haematological parameters may provide valuable prognostic insights in AML. The purpose of this study is to assess haematology parameters that influence overall survival of AML. *Materials and Methods:* A retrospective cohort of AML patients admitted in 2023 was analysed in July 2024. Patients included had complete blood tests before bone marrow puncture (BMP). Exclusions were based on inadequate BMP samples, previous leukaemia treatment, or incomplete records. Bivariate analysis using the chi-square test, which were further analysed via multivariate logistic regression. Kaplan-Meier analysis was conducted to assess survival. *Results:* Overall, 68 AML patients were included. The most of patients were female (60.3%), and AML M2 was the most common subtype (47.0%). Significant parameters included leukocyte count, platelet count, absolute neutrophil count, and neutrophil-to-lymphocyte ratio (NLR) were associated with mortality in Chi-square bivariate test ($p<0.05$). Multivariate analysis using binary logistic regression revealed that haemoglobin level ≤8 g/dL (OR: 5.08; $p=0.018$), leukocytes count $>50\times10^3/\mu L$ (OR: 5.48; $p=0.014$), platelets count $\leq50\times10^3/\mu L$ (OR: 4.90; $p=0.032$), and NLR ≥2 (OR: 31.99; $p=0.004$) were independently associated with an increased risk of mortality. Kaplan-Meier curves showed significantly reduced median survival in patients with NLR ≥2 (74.8 vs. 129.6 days). *Discussion:* Haemoglobin level ≤8 g/dL, leukocyte count $>50\times10^3/\mu L$, platelet count $\leq50\times10^3/\mu L$, and NLR ≥2 are independent predictors of mortality in AML patients. These findings may assist in early risk stratification and clinical decision-making.

MW-OR05: Genomic Landscape of Acute Promyelocytic Leukaemia in Malaysia: Insights from Whole Exome Sequencing

Ermi Neiza Mohd Sahid[1], Fatin Sofia Mohamad[1], Zahidah Abu Seman[1], Azian Naila Md Nor[1], Norafiza Mohd Yasin[1], Ezalia Esa[1], Mohd Khairul Nizam Mohd Khalid[2], Goh Ai Sim[3], Yuslina Mat Yusoff[1]

[1]Haematology Unit, Cancer Research Centre, Institute for Medical Research, National Institutes of Health, Jalan Setia Murni, Setia Alam, Shah Alam, Selangor, [2]Inherited Error Metabolism and Genetic Unit, Nutrition, Metabolic and Cardiovascular Research Centre Institute for Medical Research, National Institutes of Health, Jalan Setia Murni, Setia Alam, Shah Alam, Selangor, [3]Penang General Hospital, Jalan Residensi, George Town, Pulau Pinang

Introduction: Acute promyelocytic leukaemia (APML) is a distinct subtype of acute myeloid leukaemia, defined by the hallmark *PML::RARA* fusion gene. While this genetic feature is well-characterised, comprehensive mutational profiling of APML in Malaysian patients remains limited. This study aimed to identify somatic variants that may cooperate with *PML::RARA* in APML pathogenesis using Whole Exome Sequencing (WES). *Materials and Methods:* A total of 63 archived DNA samples were analysed. WES libraries were prepared using the Illumina DNA Prep Exome 2.0 Plus Enrichment kit and sequenced on the Illumina NextSeq platform. Variant annotation, classification, and filtering were performed using Qiagen Clinical Insight Interpret (QCII), and gene-level alterations were analysed and visualised. *Results:* The mutational landscape revealed several recurrently mutated genes. NRAS was the most frequent (17.5%), followed by *FLT3* and *WT1* (each 9.5%). Other recurrent mutations included *ARID1B, ATRX, FLT3-ITD, KMT2C, ARID1A, and KRAS* (4.8%–6.4%). Most variants were nonsynonymous single-nucleotide variants (SNVs), with notable frequencies of frameshift indels, in-frame insertions, and stop-gain mutations. *Discussion:* This study provides the first comprehensive WES analysis of APML in a Malaysian cohort. The identification of recurrent pathogenic mutations, particularly in *NRAS* and *FLT3*, highlights key cooperating genetic events in APML. These findings enhance our understanding of its molecular landscape and support the integration of genomic data into precision medicine strategies. Future work will focus on characterising novel variants and elucidating their roles in disease pathogenesis and therapeutic intervention.

MW-OR06: Leukaemic Gingival Hyperplasia as the Primary Manifestation in a Middle-Aged Woman with de novo Acute Myelomonocytic Leukaemia (AML-M4): A Diagnostic Challenge

Aditea Etnawati Putri[1*], Paulus B Notopuro[2]

[1]Department of Clinical Pathology, Faculty of Medicine- Airlangga University, Universitas Airlangga Hospital, Surabaya, Indonesia, [2]Department of Clinical Pathology, Faculty of Medicine- Airlangga University, Dr. Soetomo Hospital, Surabaya, Indonesia.

Introduction: Swelling of the gums that is often associated with dental problems (gingivitis/periodontitis) can delay the diagnosis of leukaemia. It occurs in only 15-20% of leukaemic patients (associated with monoblast infiltration in the gum tissue). Confirmation of immunophenotyping and Blood Smear/Bone Marrow Aspiration strengthens the validity of the case. *Case Report*: A 41-year-old woman sought emergency care because of a 2-month history of worsening gingival enlargement and weakness. Physical examination identified extensive, diffuse gingival overgrowth at upper and lower gingival surfaces. Complete blood count showed marked leucocytosis of 121,940/µL, with 81% monoblast. Bone marrow biopsy revealed 87% monoblasts and promonocytes. The CD45/SSC-defined blasts showed bright CD34 expression in flowcytometric and showed HLA-DR, CD33, CD13, MPO, CD64 (bright), CD117 (subset), CD14 (subset) as well. Chemotherapy was not administered, and the patient succumbed one month after diagnosis. *Discussion*: Gingival enlargement may represent either a localized metastatic lesion or indicate haematologic malignancies. Establishing a definitive diagnosis presented substantial challenges. A 41-year-old woman rapid gingival enlargement accompanied by weakness prompted the patient to hospital, complete blood count showed leucocytosis with morphologic evaluation of peripheral blood and bone marrow was consistent with FAB M5. However, immunophenotypic analysis revealed an incongruent cell profile incompatible with this subtype. Thus, this case underscores the imperative for immunophenotyping to verify leukaemia subtypes when gingival infiltration is observed, given its association with aggressive variants.

E-POSTER PRESENTATION

MW-EP01: A Retrospective Study of Overnight Transfusions and Their Impact on Patient Outcomes at HPUSM

Mohamad Nor Farhan Mudri, Mohd Nazri Hassan and Marini Ramli

Department of Haematology, School of Medical Sciences, Universiti Sains Malaysia, Kubang Kerian, Malaysia, Hospital Pakar Universiti Sains Malaysia, Kubang Kerian, Malaysia

Introduction: Blood transfusion is essential in managing conditions such as symptomatic anaemia and major bleeding. While daytime transfusions (DT) are generally preferred for safety, overnight transfusions (OT) remain common, particularly for urgent clinical needs. However, OT carries unique challenges including reduced staffing and monitoring, which may increase risks of transfusion reactions and errors. This study aims to evaluate OT practices at Hospital Pakar Universiti Sains Malaysia (HPUSM), focusing on prevalence, indications, blood product types, and associations with transfusion reactions and hospital length of stay (LOS). *Materials and Methods:* A cross-sectional retrospective study analyzed 600 transfusion events between July and December 2023, randomly selected from 4,353 total events at HPUSM. Transfusions were categorized as OT (8 PM–8 AM) or DT (8 AM–8 PM). Data included patient demographics, blood products transfused (PRBC, platelet concentrate, FFP, cryoprecipitate), clinical indications, and LOS. Statistical analyses were performed using the Chi-square test and Mann–Whitney U test, with significance set at $p < 0.05$. *Results:* OT accounted for 28.8% (n=173) of transfusions, predominantly packed red blood cells (75.1%). Most OT (87.4%) were for acute clinical needs such as bleeding and severe anaemia; only 1.2% had no clear indication. There was no significant difference in transfusion reaction rates between OT and DT groups (1.1% vs. 2.1%, p=0.431). OT was significantly associated with shorter LOS compared to DT (p=0.018). *Discussion:* Most OTs at HPUSM were clinically justified and not linked to increased transfusion reactions. OTs were associated with shorter hospital stays, reflecting timely management of acute cases.

Optimising scheduling, monitoring, and staffing during overnight hours is essential to enhance transfusion safety and resource utilisation.

MW-EP02: *Exosomal miR-6879* and *miR-107* from Blood Plasma as a Predictive Biomarkers for Dengue Disease Progression
Gokulakannan Venkatesan[1], Wan Suriana Wan Ab Rahman[1,2*], Wan Nazatul Shima Shahidan[1], Salfarina Iberahim[3], Edinur Hisham Atan[4], Norliana Ghazali[1]
[1] School of Dental Sciences, Universiti Sains Malaysia Health Campus, Kota Bharu, Kelantan, Malaysia; [2]Hospital Universiti Sains Malaysia Health Campus, Kota Bharu, Kelantan, Malaysia; [3]School of Medical Sciences, Universiti Sains Malaysia Health Campus, Kota Bharu, Kelantan, Malaysia; [4]School of Health Sciences, Universiti Sains Malaysia Health Campus, Kota Bharu, Kelantan, Malaysia.

Introduction: Dengue virus infection manifests as either a febrile illness or a severe, life-threatening condition known as dengue haemorrhagic fever. Currently, no predictive diagnostic markers exist to identify individuals at risk of bleeding manifestations. Exosomal microRNAs (miRNAs) may serve as disease-specific diagnostic signatures, enabling the analysis of exosomal plasma miRNA expression in dengue infection. This study investigates the potential of exosomal plasma miRNA expression as a predictive biomarker for bleeding tendencies in dengue infection. *Materials and Methods*: Microarray-based expression profiling of exosomal miRNAs from plasma samples of six dengue patients was performed to identify predictive biomarkers for bleeding manifestations in patients with warning signs compared to those without. Target genes were predicted using TargetScan and miRDB. A protein-protein interaction (PPI) network was constructed and analysed using Cytoscape to identify hub genes. This approach helps elucidate the regulatory roles of miRNAs and their associated genes. Selected miRNAs were validated using reverse transcription quantitative PCR (RT-qPCR). *Results:* Microarray profiling identified 24 upregulated miRNAs (log2 fold change > 2.0) in plasma-derived exosomes of dengue patients with warning signs. Among them, hsa-miR-107 and hsa-miR-6879-5p were reliable candidates for predicting bleeding risk. In silico functional analysis linked their target genes to bleeding manifestations. RT-qPCR validation confirmed their upregulation. *Discussion:* This study identifies *hsa-miR-107* and *hsa-miR-6879-5p* as potential biomarkers for bleeding risk in dengue infection. Their role in disease progression warrants further investigation in larger cohorts to establish clinical utility and association with disease severity.

MW-EP03: Unmasking De Novo Hereditary Spherocytosis in Elderly: A Rare Case Report
Sri Rahayu Sabtu, Rohana Ghazali, Azlinda Abu Bakar, Wan Aswani Wan Yusof, Raudhawati Osman
Hospital Melaka, Jalan Mufti Haji Khalil, 75400 Melaka.

Introduction: Hereditary spherocytosis (HS) is the most common congenital haemolytic anaemia, typically caused by mutations in red blood cell membrane proteins. It usually presents early in life, with the median age of first presentation between 3-5 years. This case report highlights a rare instance of late presentation in an elderly patient. *Case Report*: A 75-year-old gentleman, with history of hypertension and benign prostatic hypertrophy, presented with jaundice and worsening anaemia since August 2024. He had no family history of blood disorders. On examination, mild jaundice was observed but no hepatosplenomegaly was noted. Blood tests revealed Hb: 9.6 g/dl (baseline Hb: 13.0 g/dl), WBC: 5.6 x 10^9/L, Platelets: 97 x 10^9/L and reticulocyte count of 6.7%. Liver function tests showed elevated total and indirect bilirubin (44.7 umol/L and 30.1 umol/L respectively) with normal liver enzymes and alpha-fetoprotein. Full blood picture showed spherocytosis and occasional Howell-Jolly bodies. A negative Coombs test ruled out immune haemolytic anaemia. An osmotic fragility test confirmed fragile cells with a right-shifted osmotic fragility curve. The final diagnosis was hereditary spherocytosis. *Discussion*: HS is an autosomal dominant disorder commonly presented in childhood. However, de novo mutations in elderly individuals can occur without family history of haemolysis (5% of cases). Elderly patients may present with jaundice, haemolytic crises or splenomegaly. Treatment is typically conservative in elderly patients, though splenectomy remains the gold standard for younger individuals.

MW-EP04: Performance Evaluation for Sysmex Automated Coagulation Analyser CN-3000; A Preliminary Study
Salfarina Iberahim, Nor Khairul Putra Mohd Madi, Ahmad Zakwan Mustafa, Rosline Hassan, Noor Haslina Mohd Noor, Zefarina Zulkafli, Shafini Mohamed Yusoff, Mohd Nazri Hassan
Department of Haematology, School of Medical Sciences, Universiti Sains Malaysia, Kelantan, Malaysia

Introduction: The coagulation test measures the time it takes for a blood sample to clot, using either mechanical that uses a small steel ball to detect changes in blood viscosity as a clot forms, indicating clotting time, or optical that uses light transmission changes to track clot formation and turbidity. Both methods offer valuable information about blood clotting, but the choice depends on specific clinical needs and potential confounding factors. Both methods have their strengths and limitations. In this study, we aimed to evaluate the analytical performance of two distinct detection technologies based on optical (CN-3000) and mechanical clot detection (STA-R Max) methodologies and correlate the result of both for PT, APTT, Fibrinogen, and D-dimer. *Materials and Methods:* Method: Sixty-seven randomly selected blood samples from residual patient samples from the Haematology Laboratory, Hospital Universiti Sains Malaysia were selected. PT, APTT, Fibrinogen, and D-dimer were performed using both analysers. Analytical performances were evaluated for correlation studies. *Results:* Correlation coefficient (R2) values obtained from the analysis of PT, APTT, Fibrinogen, and D-Dimer assays on both analysers were 0.98, 0.93, 0.99, and 0.97, respectively. All values show a very strong correlation based on universal R2 interpretation guidelines. *Discussion:* Each analytical methodology was accurate and highly precise for safe use in clinical or research applications. No significant differences or outliers were found in the correlation study performed very rigorously.

MW-EP05: Unexpected Possible Bone Marrow Findings: Gelatinous Bone Marrow Transformation in Patient with Underlying Retroviral Disease

Dr Noraini Amir, Srinorleza Jalil, Norhayati Taufik
Hospital Enche' Besar Hajjah Khalsom, Kluang Johor.

Introduction: Gelatinous bone marrow transformation (GTBM), also known as starvation marrow was first described by Paul Michael in 1930 is a rare condition with unknown definitive pathogenesis. It is associated with a non-specific spectrum of disease such as severe malnutrition, malignancies and young age acquired immunodeficiency syndromes (AIDS). GTBM is a histology finding characterised by bone marrow hypoplasia, fat atrophy and accumulation of extracellular mucopolysaccharides mainly hyaluronic acid. *Case Report:* We report a case of 33 years old cachexic male, underlying Retroviral disease with CD4 count 63 cells/uL, chronic hepatitis B infection and pulmonary tuberculosis presented with prolonged fever, loss of appetite and persistent bicytopenia. Clinically having hepatosplenomegaly and lymphadenopathies. The bone marrow biopsy showed marrow hypoplasia, fat cells atrophy and deposition of gelatinous material that strongly stained for Alcian blue suggestive of GTBM. Immunostaining showed no abnormal lymphoid cells and intracellular organisms detected. The patient died three months after biopsy. *Discussion:* GTBM is relatively uncommon histology findings. These gelatinous materials are not present in the bone marrow normally and must be differentiated from marrow oedema, necrosis or amyloidosis. It is suggested that hyaluronic acid may be a substance that replaces marrow fat cells used in catabolic states of the disease. This interesting case demonstrates GTBM reflects severe underlying systemic and carries poor prognosis.

MW-EP06: Comparative Analysis of *FLT3*-ITD Detection by PCR and Amplicon-Based Next Generation Sequencing in Acute Myeloid Leukaemia

Zahidah Abu Seman, Ermi Neiza Mohd Sahid, Azian Naila Md Nor, Julia Abdullah, Fifi Shawani Abtah, Siti Noorain Mohd Razali, and Yuslina Mat Yusoff
Haematology Unit, Cancer Research Centre, Institute for Medical Research, National Institutes of Health, Ministry of Health Malaysia, 40170 Shah Alam, Selangor, Malaysia

Introduction: FLT3 internal tandem duplication (ITD) mutations are common in acute myeloid leukaemia (AML) cases and are critical markers for risk stratification, and targeted therapies selection. PCR remains the gold standard for *FLT3*-ITD detection due to its sensitivity and ability to determine allelic ratio. Amplicon-based next-generation sequencing (NGS) panels are widely used for comprehensive genomic profiling including *FLT3*-ITD analysis, however their performance in detecting large ITDs remains variable. This study aims to evaluate the performance of amplicon-based NGS and PCR in detecting *FLT3*-ITDs. *Materials and Methods:* A total of 651 AML patient samples collected from October 2023 to December 2024 were analysed. DNA was extracted and amplified for the PCR method using *FLT3*-specific primers flanking exons 14 and 15. Amplicon-based NGS was performed using the AmpliSeq for Illumina Myeloid Panel. Libraries were prepared according to manufacturer protocols and sequenced with 2×150 bp paired-end reads. Allelic ratio assessment was performed using PCR only, as amplicon-based NGS has limited sensitivity for large or complex ITDs and does not provide reliable quantification for allelic ratio Statistical analyses were not conducted in this study. *Results: FLT3*-ITDs were detected in 112 of 651 cases (17.2%) by PCR fragment analysis. In contrast, amplicon-based NGS detected ITDs in only 18 of 651 cases (2.8%). *Discussion:* The significant discrepancy in detection rates reflects limitations of amplicon-based NGS in capturing large or complex ITDs, especially those overlapping primer sites or exceeding typical read lengths. Reliance solely on amplicon-based NGS may lead to false negatives. PCR-based fragment analysis remains essential for accurate detection. Additionally, hybrid capture based NGS panels offer improved sensitivity and may serve as a more comprehensive alternative.

MW-EP08: A Diagnostic Clue Hidden in Platelet Counts: *CALR* Type 1 Mutation in a Young Adult with Essential Thrombocythemia

Ameerah Nabilah Azhar Hilmy, Zefarina Zulkafli, Mohd Nazri Hassan, Nur Asni Ibrahim
Department of Haematology, School of Medical Sciences, Health Campus Universiti Sains Malaysia, Kubang Kerian, Kelantan, Hospital Pakar Universiti Sains Malaysia, Kubang Kerian, Kelantan

Introduction: CALR (*CALR*eticulin) mutations, especially type 1, are seen in approximately 20–25% of essential thrombocythemia (ET) patients and are more common in younger individuals. Early diagnosis and appropriate monitoring are essential, especially in young individuals, to prevent complications such as thrombosis or progression to myelofibrosis. *Case report:* A 24-year-old single male, an active smoker, was admitted following a first-time seizure episode. He had no significant past medical history and no known family history of haematologic disorders. Laboratory investigations showed persistent thrombocytosis, with platelet counts ranging between 966 and 1372 × 10^9/L throughout his hospital stay. His haemoglobin (Hb) and white blood cell (WBC) counts were within normal ranges, and inflammatory markers such as CRP and ESR were not elevated. There was no evidence of organomegaly or lymphadenopathy. The patient reported no history of thrombotic events, including deep vein thrombosis (DVT), pulmonary embolism (PE), chest pain, blurry vision, or neurological deficits prior to the seizure. A CT venogram of the brain was performed to rule out dural venous sinus thrombosis, and the *Results* were negative. Molecular testing showed a *CALR* type 1 mutation. The diagnosis of ET was made. *Discussion:* This case highlights ET in a young adult, a rare occurrence often leading to delayed diagnosis. The patient's thrombocytosis was incidentally discovered during evaluation for an unrelated neurological issue. *CALR* type 1 mutation, common in younger males and associated with higher platelet counts, lower thrombosis risk, and distinct clinical features compared to *JAK2*-mutated ET. Despite a favourable short-term prognosis, recent studies suggest a potential long-term risk of myelofibrotic transformation. The absence of systemic inflammation and secondary causes confirmed ET. This

case underscores the importance of molecular diagnostics and long-term monitoring in young ET patients.

MW-EP09: Cup-Like Clues: Diagnosing *NPM1*-Mutated AML Without *FLT3* Mutation

Ameerah Nabilah Azhar Hilmy, Cicelia Thomas, Zefarina Zulkafli, Mohd Nazri Hassan
Department of Haematology, School of Medical Sciences, Health Campus Universiti Sains Malaysia, Kubang Kerian, Kelantan, Hospital Pakar Universiti Sains Malaysia, Kubang Kerian, Kelantan

Introduction: NPM1-mutated AML accounts for approximately 30% of adult AML cases. This case highlights the diagnostic features and implications of *NPM1*-mutated AML in an elderly patient. *Case report:* A 77-year-old man with underlying benign prostatic hyperplasia and diabetes mellitus presented with a one-month history of general malaise, poor appetite, and weight loss. He also has an increased urinary frequency and foul-smelling urine over the previous two days. There was no evidence of organomegaly, lymphadenopathy, or bleeding tendency on examination. Laboratory investigations revealed: Total white cell count: 191.69 × 10^9/L (hyperleukocytosis), absolute lymphocyte count: 144.53 × 10^9/, haemoglobin: 8.4 g/dL (moderate normocytic normochromic anaemia) and normal platelet count. Peripheral blood smear showed 84% blast cells, primarily small to moderate in size with fine chromatin, prominent nucleoli, and moderate granular cytoplasm. Some blasts exhibited cup-like nuclei. No Auer rods were observed. Bone marrow aspirate revealed 93% blasts with similar morphology. Flow cytometry identified 84% myeloblasts expressed cyMPO, CD117 (heterogeneous), CD13 (heterogeneous), CD33, CD64 and negative for CD34, and HLA-DR. Molecular analysis detected *NPM1* mutation, with no *FLT3*-ITD or *FLT3*-TKD mutations. Cytogenetic testing revealed a normal male karyotype (46,XY). A diagnosis of acute myeloid leukaemia (AML) with *NPM1* mutation was made. *Discussion:* This case highlights *NPM1*-mutated AML in an elderly patient, now classified as a genetically defined subtype as per WHO 2022. The hallmark of exon 12 mutation causes abnormal cytoplasmic *NPM1* protein localization. Characteristic features include cup-like nuclear invaginations and CD34/HLA-DR negativity with CD33/CD13 expression as seen in this case. The absence of *FLT3*-ITD and normal cytogenetics suggest a favourable prognosis despite the advanced age.

MW-EP10: Underestimated Acquired Haemophilia A: Diagnostic Delay Due to Comorbidities and Absence of Bleeding

Fatin Akmal Baharom[1], Siti Umairah Abd Rahman[1], Nurimatussolehah Sarijan[1], Noraesah Mahmud[2], Sharifah Suryani Syed Rahim Shah[3], Suzana Zainol[1]
[1]Haematology Laboratory, Pathology Department, Hospital Tunku Azizah, Kuala Lumpur; [2]Haematology Unit, Pathology Department, Hospital Kuala Lumpur; [3]Haematology Unit, Medical Department, Hospital Kuala Lumpur

Introduction: Early detection of Factor VIII inhibitors is crucial, yet challenging, particularly in atypical presentations. Misidentification or delayed diagnosis can significantly impact patient outcomes. This case highlights the variability in bleeding presentations, the associated diagnostic challenges, and the laboratory's role in ensuring timely identification. *Case report:* An elderly woman with hypertension, Sjögren's syndrome, and knee osteoarthritis presented with one-month history of painful right knee swelling, requiring surgical procedures. Preoperative investigations revealed isolated prolonged APTT, with mixing studies showing partial correction initially but no correction after two hours, suggestive of time- and temperature-dependent inhibitor. A sample was requested for FVIII activity and inhibitor study. However, due to the absence of significant bleeding, the orthopaedic surgeon proceeded with the arthrotomy and joint washout under fresh frozen plasma cover, considering acquired haemophilia A less likely. A week later, significant bleeding occurred after her second arthrotomy, prompting reassessment. Laboratory tests confirmed acquired haemophilia A with FVIII:C of 4.5% and inhibitor of 27 B.U. Lupus anticoagulant was also detected by the silica test system, possibly due to co-occurrence or test interference especially with APTT-based assay. The patient was promptly treated with steroids and bypassing agent, leading to clinical improvement and normalization of APTT and FVIII:C within a month. *Discussion:* Even in the absence of bleeding symptoms, early recognition of inhibitors is essential, as atypical presentations can delay diagnosis and compromise patient outcomes. This case highlights the diagnostic challenges and critical role of clinician-laboratory collaboration in ensuring timely and accurate diagnosis for optimal patient care.

MW-EP11: Exploring the Role of Immune Cells in Red Blood Cell Alloimmunisation Among Pregnant Women - A Preliminary Study

Nurul 'Adani Sanusi[1], Mohd Nazri Hassan[1], Noor Suryani Mohd Ashari[2], Muhammad Farid Johan[1], Hisham Atan Edinur[3], Noor Haslina Mohd Noor[1]
[1]Department of Haematology, School of Medical Sciences, USM, Kubang Kerian, Kelantan, [2]Department of Immunology, School of Medical Sciences, USM, Kubang Kerian, Kelantan,[3]Forensic Programme, School of Health Sciences, USM, Kubang Kerian, Kelantan

Introduction: Red blood cell (RBC) alloimmunisation occurs when an individual immune system produces alloantibodies against foreign RBC antigens, usually following transfusion or pregnancy. Maternal alloantibodies can cross the placenta and destroy foetal RBC, causing haemolytic disease of the foetus and newborn, and resulting in infant morbidity and mortality. This study aimed to explore the role of immune cells in maternal RBC alloimmunisation. *Materials and Methods*: Peripheral blood mononuclear cell (PBMC) were collected from four alloimmunised (anti-E) and 25 non-alloimmunised pregnant women, all with R1R1 Rhesus phenotype and tested for proliferation assay (stimulated with RhCE peptide) at 24, 48 and 72 hours using MTT test, HLA-blocking assay (using anti-HLA-DR and-DQ antibodies), and flow cytometry analysis for B, T (helper, cytotoxic, regulatory) and plasma cells. The proliferation assay measured as stimulation index (SI). *Results:* The alloimmunised group showed higher PBMC proliferation at 48 (1.041 versus 1.001) and 72 hours (1.142 versus 1.054) compared to the non-alloimmunised group, though the difference was not statistically significant ($p>0.05$). HLA-blocking showed slightly reduced proliferation in both groups compared to control (1.099/0.986 versus 1.142 and 0.987/0.963 versus 1.055), without significant difference ($p>0.05$). Notably, the alloimmunised group exhibited higher total lymphocytes and most immune cell subsets (T, T helper, T cytotoxic, Treg, and plasma cells) except for B cells. Only the increase in total lymphocytes was statistically significant ($p=0.011$). *Discussion:* PBMC proliferation suggested a

delayed yet elevated immune response in the alloimmunised group, while blocking assays showed no significant inhibition. These findings highlight a potential alteration in immune cell composition among alloimmunised pregnant women. Further larger studies are needed to confirm these observations.

MW-EP12: Clinicopathological characteristics and outcome of childhood acute lymphoblastic leukaemia in Hospital Universiti Sains Malaysia (HUSM)

Lishalini Raja[1], Noor Haslina Mohd Noor[1], Marne Abdullah[1], Norsarwany Mohamad[2]
[1]Department of Haematology, School of Medical Sciences, Universiti Sains Malaysia, Kubang Kerian, Kelantan, Malaysia, [2]Department of Paediatrics, School of Medical Sciences, Universiti Sains Malaysia, Kubang Kerian, Kelantan, Malaysia

Introduction: Acute lymphoblastic leukaemia (ALL) is the most prevalent type of cancer in the paediatric population. This study was conducted to describe and explore clinical and laboratory findings with treatment outcomes. *Materials and Methods:* This retrospective cohort study involved 68 paediatric patients aged 1 to 17 years old, diagnosed with ALL in Hospital Universiti Sains Malaysia (HUSM) between January 2013 and December 2022. A thorough analysis of demographic, clinical, laboratory and treatment outcome, which includes prognostic factors and survival study was conducted. Kaplan-Meier survival analysis was used to assess survival differences among clinical subgroups. Cox proportional hazards regression analysis was used to identify significant prognostic factor associated with treatment outcome. *Results:* Most patients had B-ALL (91.2%). Most of them were diagnosed at the age of 1 to 9 years old (79.4%) with a slight male predominance (57.4%). The analysis showed that the overall five-year survival probability of paediatric ALL was 66.1%. After adjustment for potential cofounders, multiple cox regression revealed that the significant prognostic factors were Minimal residual disease (MRD) positive at end of induction (Adj HR 3.43; 95% CI: 1.18, 9.914; $P = 0.023$) and fresh frozen plasma (FFP) transfusion (Adj HR 3.12; 95% CI: 1.089, 8.933; $P = 0.034$) *Discussion:* MRD positivity and FFP transfusion emerged as an important prognostic factor for treatment outcome. As more sensitive novel therapies enter clinical development, ALL management in paediatrics requires evidence-based and risk adapted treatment approaches to guide intensification or de-escalation of therapy.

MW-EP13: Retrospective Analysis of Peripheral Blood Stem Cell Mobilisation Kinetics in Lymphoma and Multiple Myeloma Patients: Experience from a North-eastern Teaching Hospital

Mohd Nazri Hassan[1], Mohd Amirudin Sidik[2], Ahmad Zaki Idris[2], Noor Haslina Mohd Noor[1], Marini Ramli[1], Marne Abdullah[1], Nur Ilyia Syazwani Saidin[1]
[1]Department of Haematology, School of Medical Sciences, USM, Kubang Kerian, Kelantan, [2]Transfusion Medicine Unit, Hospital Pakar USM, Kubang Kerian, Kelantan

Introduction: Autologous peripheral blood stem cell (PBSC) transplantation remains essential in the management of lymphoma and multiple myeloma (MM). However, variations in mobilisation efficacy between different patient groups require further investigation to refine mobilisation strategies. This study aimed to compare PBSC mobilisation kinetics between lymphoma and MM patients. *Material and Methods***:** A retrospective analysis was conducted on 115 patients [91 with 43 non-Hodgkin (NHL) and 48 Hodgkin lymphoma (HL), and 24 with MM] who underwent successful autologous PBSC mobilisation with granulocyte colony stimulating factor and chemotherapy agents, from 2019 to 2024 in Hospital Pakar Universiti Sains Malaysia. Selected mobilisation kinetic parameters evaluated included the day of peripheral blood (PB) CD34+ cells count reached ≥20 cells/μL, the day of leukapheresis, total CD34+ cell dosage and the number of leukapheresis. *Results:* MM patients achieved the target PB CD34+ cell count significantly earlier than lymphoma patients (mean day 10.2 versus 13.4, $p<0.001$), with earlier collection days (mean day 11.0 versus 13.9, $p<0.001$). Subgroup analysis showed significant differences in mobilisation kinetics between MM and both HL and NHL, but not between NHL and HL. Notably, lymphoma patients showed higher mean CD34+ cell dose but statistically insignificant (8.09 versus 6.79×10^7 cells/kg, $p=0.063$) with comparable number of leukapheresis. *Discussion:* These findings indicate that MM tends to exhibit more rapid mobilisation than lymphoma patients, with comparable efficacy in CD34+ yields and collection procedures. These findings highlight the need for tailored mobilisation strategies based on disease type and patient profile to enhance transplant success rates. It is recommended that on day 10 of mobilisation, all patients should have their PB CD34+ cell count measured and be prepared for leukapheresis.

MW-EP14: Distribution of Rh Phenotyping among Malay Patients in Hospital Pakar Universiti Sains Malaysia

Asiah Nabihah Arifin, Nur Ilyia Syazwani Saidin, Siti Asmaa Mat Jusoh, Nurul Izzah Abdul Razak, Noor Haslina Mohd Noor
Department of Haemotology, School of Medical Sciences, Universiti Sains Malaysia Health Campus, Kubang Kerian, Kelantan.

Introduction: The Rh blood group system ranks as the second most clinically important following the ABO system, consisting of more than 50 antigens, particularly D, C, c, E, and e. The D antigen is very immunogenic and crucial to haemolytic transfusion reactions (HTRs) and haemolytic disease in newborns (HDN). This study aims to determine the prevalence of Rh phenotypes among patients at Hospital Pakar Universiti Sains Malaysia (HPUSM), ensuring transfusion safety and effective clinical management. *Materials and Methods:* This retrospective study involved patients screened for Rh phenotypes in the Transfusion Medicine Unit, HPUSM from January to December 2023. Rh phenotyping was carried out for patients with antibody screening positive, and suspected to have Rh alloantibody by using semi-automated system (Bio-Rad) with DiaClon ID Cards. Patients' data was obtained from the laboratory information system known as 'myTransfusi'. The data was analysed using SPSS version 28 for descriptive analysis. *Results:* A total of 101 patients were screened for Rh phenotype. The most prevalent phenotype was R1R1 (DCe/DCe), observed in 55.4% of patients, followed by rr (dce/dce) at 13.9% and R1r (DCe/dce) at 9.4%. Less common phenotypes included R1R2, R2R2, r′r, and R2r. Rare phenotypes such as R0R0, RZR1, and rr″ were each identified in only one patient, respectively. Among Rh-negative individuals, the rr phenotype predominated. *Discussion:* The prevalence of the R1R1 phenotype among Malay patients at HPUSM is consistent with regional trends. These findings highlight the importance of local Rh phenotype databases in

guiding transfusion procedures, reducing alloimmunisation risk, and improving patient outcomes.

MW-EP15: More Than Just Thrombocytopenia: A peripheral Blood Film Unmasks Occult Metastatic Breast Cancer

Siti Alia Izzani Ismail, Ling Pei Chi, Chee Weng Yan Sabrina, Abdul Kadir Rifaie Abd Rashid Khairi, Izian Munirah Ahmad Khusairi, Nor Arsha Sakilla Alladin
Department of Pathology, Hospital Teluk Intan, Jalan Changkat Jong, Teluk Intan, Perak

Introduction: Unexplained cytopenias may indicate underlying bone marrow pathology or infiltration. This case highlights the diagnostic challenges of atypical presentations in malignancy with metastatic bone marrow involvement and the role of peripheral blood film (PBF) in facilitating diagnosis. A timely intervention could prolong patient survival. *Case report:* A 34-year-old woman with no comorbidity presented with nonspecific generalized body aches. Physical examination findings were unremarkable. Initial blood counts revealed leucocytosis, while all other blood parameters were normal. She was re-presented with similar symptoms along with new onset chest pain, shortness of breath, abdominal pain, and vomiting. Repeated blood investigations showed transaminitis, elevated alkaline phosphatase, lactate dehydrogenase, uric acid, and calcium. A new onset thrombocytopenia with persistent leucocytosis, prompting a PBF which revealed leucoerythroblastic with blasts. Computed tomography (CT) abdomen and pelvis was ordered to rule out bowel ischemia incidentally revealed liver lesions and sclerotic lytic bone lesions. A subsequent CT thorax noted right breast mass with multiple lymphadenopathies. Mammography revealed the presence of only a small breast mass. Histology of breast tissue biopsy confirmed the diagnosis of invasive carcinoma of non-special type with strong HER2 receptor and moderate oestrogen receptor positivity. The patient was classified as stage 4 breast cancer and planned for palliative chemotherapy and bone marrow assessment. Unfortunately, shortly after the diagnosis of metastatic breast cancer was established, the patient was admitted for a lung infection and subsequently succumbed to her illness. *Discussion:* This case highlights the role of peripheral blood film in unveiling marrow infiltration when metastatic malignancy presents atypically. In women of reproductive age, the presence of leucoerythroblastic with blasts should prompt evaluation for breast or ovarian cancer and leukaemia, even in the absence of classic malignancy symptoms. A thorough diagnostic approach, including laboratory and imaging studies, is crucial for accurate diagnosis in such atypical cases.

MW-EP16: Secondary Immune Thrombocytopenia in Hospital Pakar Universiti Sains Malaysia: A 25-Year Snapshot

Noor Haslina Mohd Noor[1], Nurul Izzah Abdul Razak[1], Mohd Aidil Zahidin[1], Asiah Nabihah Arifin[1], Shafini Mohd Yusof[1], Rosnah Bahar[1] and Wan Suriana Wan Abdul Rahman[2]
[1]Department of Haemotology, School of Medical Sciences, Universiti Sains Malaysia, Kubang Kerian, Kelantan, Malaysia, [2]School of Dental Sciences, Universiti Sains Malaysia, Kubang Kerian, Kelantan, Malaysia

Introduction: Secondary immune thrombocytopenia (sITP) is an uncommon haematological disorder characterised by low platelet counts associated with underlying conditions. Despite its clinical significance, local epidemiological data in Malaysia remain limited. This study aimed to determine the prevalence, incidence, and associated clinical characteristics of sITP among patients treated at Hospital Pakar Universiti Sains Malaysia (HPUSM) over a 25-year period. *Materials and Methods:* A retrospective review was conducted on adult patients diagnosed with sITP at HPUSM from January 2000 to December 2024. Patient data were obtained from the Haematology Patient Information System and Laboratory Information System databases. Sociodemographic, clinical, and haematological parameters were analysed. Incidence rates were calculated based on regional population data. *Results:* A total of 64 sITP cases were identified, representing 30.2% of all immune thrombocytopenia (ITP) cases (n=211) during the study period. The estimated annual incidence rate was 0.45 per 100,000 person-years (95% CI: 0.42 – 0.70) with a higher rate in females (0.86 per 100,000 person-years, (95% CI: 0.64–1.14) than males (0.26 per 100,000 person-years, 95% CI: 0.15–0.43), yielding a male-to-female ratio of 1:3.27. The median age at diagnosis was 37 years, with most patients aged ≥30 years. The most common associated comorbidity was systemic lupus erythematosus (35.9%), followed by antiphospholipid syndrome and familial thrombocytopenia. Notably, 70.6% of sITP patients were symptomatic at the time of initial diagnosis. Additionally, 56.3% of patients had platelet counts $<30 \times 10^9$/L at diagnosis, indicating a substantial burden of severe thrombocytopenia. Moreover, a total of 70.6% of sITP patients were symptomatic during the initial diagnosis. *Discussion:* sITP is a rare but significant condition, with a notable female predominance and strong association with autoimmune disorders, particularly systemic lupus erythematosus. The majority of patients presented with severe thrombocytopenia, highlighting the importance of early identification and management.

MW-EP17: A Rare Haematologic Overlap: The Diagnostic Conundrum of Myelodysplastic/ Myeloproliferative Neoplasm with Neutrophilia

Shaidatina Mat Yusuf, Ahmad Nasirudin, Noorhana Sofia Ismail, Lee See Lin
Department of Pathology, Hospital Queen Elizabeth, Kota Kinabalu, Sabah.

Introduction: Myelodysplastic/myeloproliferative neoplasm with neutrophilia (MDS/MPN-N), previously termed "atypical chronic myeloid leukaemia", is a rare haematologic malignancy. It is characterised by sustained peripheral neutrophilia, left-shifted granulopoiesis, and absence of the *BCR::ABL1* fusion gene. Diagnosis remains challenging due to the heterogeneous clinical presentation and lack of specific molecular or immunophenotypic markers, making it a diagnosis of exclusion. *Case report*: A 59-year-old man was admitted for occult sepsis. Physical examination revealed hepatosplenomegaly. Initial laboratory investigations showed Hb 6.8 g/dL, WBC 60.94×10^3/μL, and PLT 73×10^3/μL. Peripheral blood smear demonstrated marked leucocytosis with a left shift and dysplastic changes in granulocytes. A manual differential count: blast 2%, promyelocytes 1%, myelocytes 23%, metamyelocytes 4%, neutrophils 54% and lymphocytes 16%). CRP and LDH were elevated (95 mg/L and 684 U/L, respectively), while renal, liver, folate, and vitamin B12 profiles were within normal limits. Bone marrow examination showed marked hypercellularity with predominant myeloid hyperplasia (M:E ratio 9.6:1), significant dysplasia and no excess blasts.

Dysgranulopoiesis was evident, characterised by abnormal cytoplasmic granulation, nuclear hypolobation, and hypersegmentation. Remarkable dysmegakaryopoiesis included hypolobated forms, micromegakaryocytes and megakaryocytes with separated nuclear lobes. Molecular testing was negative for *BCR::ABL1*; *JAK2* mutation analysis is pending. *Discussion:* Morphological assessment remains the cornerstone in diagnosing MDS/MPN-N, especially given its clinical and morphological overlap with other *BCR::ABL1*-negative myeloid neoplasms. Accurate classification is crucial, particularly in transplant-eligible patients, due to the relatively poor prognosis associated with this entity. Early recognition and exclusion of mimickers are essential for optimal patient management.

MW-EP18: Beyond the surface: Diagnostic complexities in histiocytic disorders

Geetha Nallappan[1], Kisa Khazanah Uthayarajah[1], Sukanya Banerjee Nair[1], Siti Zubaidah Mustapha[1], Zainura Anita Zainal Abidin[1], Asmawiza Awang[2], Wan Hayati Mohd Yaakob[1]

[1]Department Of Pathology, Hospital Tengku Ampuan Rahimah, Selangor, *[2]Department Of Pathology, Hospital Kuala Lumpur, Wilayah Persekutuan*

Introduction: Histiocytic neoplasms are diagnostically challenging due to their nonspecific fibroinflammatory infiltrates and varied clinical presentations. This case highlights the complexity in reaching a definitive diagnosis. *Case Report:* A 77-year-old man with chronic folate deficiency presented with abdominal pain, constipation, lethargy, and weight loss. Examination revealed ascites and bilateral pleural effusions with negative cytology, but no lymphadenopathy or organomegaly. Laboratory investigations showed pancytopenia, unconjugated hyperbilirubinemia, and hyperferritinaemia. A lipid profile was not performed. Blood film demonstrated mixed nutritional deficiency anaemia without significant schistocytes. Bone marrow aspirate showed macrophage proliferation with PAS positivity and mild phagocytic activity. The trephine biopsy, although suboptimal, revealed aggregates of CD68-positive histiocytes containing intracytoplasmic red cells. There was no emperipolesis or granuloma formation. The histiocytes were S100 and clonality-negative, leaving a histiocytic neoplasm unconfirmed. *Discussion:* Chronic folate deficiency may contribute to a reactive histiocytic response by upregulating pro-inflammatory cytokines. Animal studies suggest that folate deficiency disrupts one-carbon metabolism, impairs lipid synthesis, and induces oxidative stress, potentially contributing to reactive histiocytosis. Although hyperferritinaemia and phagocytic activity raised suspicion for Haemophagocytic Lymphohistiocytosis, the absence of key diagnostic criteria made this unlikely. Lipid storage disorder was considered but ruled out due to the patient's age and lack of typical features. Neoplastic histiocytic disorders, including Langerhans cell histiocytosis, Rosai-Dorfman disease, and Erdheim-Chester disease, can be excluded based on morphology and immunohistochemistry even in the absence of radiological investigation. This case suggests a potential link between folate deficiency and reactive histiocytosis, warranting further research.

MW-EP19: Acquired Platelet Dysfunction with Eosinophilia: Morphological and Functional Diagnostic Insights in an Adolescent

Nur Amalina Adhwa Najibollah[1], Ummi Mohlisi Mohd Asmawi[1,2], Norashikin Saidon[3], Siti Nuur Syahirah Sabran[4]

[1]Department of Clinical Diagnostic Laboratories, Hospital Al-Sultan Abdullah, Universiti Teknologi MARA, Puncak Alam, Selangor, Malaysia, [2]Department of Pathology, Faculty of Medicine, Universiti Teknologi MARA, Sungai Buloh, Selangor, Malaysia, [3]Department of Medicine, Faculty of Medicine, Universiti Teknologi MARA, Selangor, Malaysia, [4]Department of Primary Care Medicine, Faculty of Medicine, Universiti Teknologi MARA, Selangor, Malaysia

Introduction: Spontaneous bruising in adolescents with normal platelet counts and coagulation profiles presents a diagnostic challenge. Acquired platelet dysfunction with eosinophilia (APDE) is a rare, benign, and self-limiting condition that mimics immune thrombocytopenic purpura (ITP), von Willebrand disease, and non-accidental injury. *Case Report:* We report a case of a 15-year-old girl with a one-week history of spontaneous, painless bruising over multiple limbs. Examination revealed ecchymoses without petechiae or systemic symptoms. Laboratory investigations showed normal haemoglobin (12.7 g/dL), platelet count (180 ×10 /L), and coagulation parameters, with leucocytosis (10.6 ×10 /L) and marked eosinophilia (2.89 ×10 /L; 23.7%). Peripheral blood film revealed normochromic normocytic red cells, matured eosinophils, and adequate platelets with a few large and giant forms. Platelet aggregation studies showed loss of response with ristocetin and collagen, and partial loss with ADP. The patient responded to empirical albendazole with resolution of bruising and normalisation of eosinophil counts. *Discussion:* Although platelet morphology in APDE is usually normal, the large platelets in this case highlight morphological variability. While such findings are more characteristic of inherited macrothrombocytopaenia's, their presence in APDE has been noted in some reports and should not exclude the diagnosis. This case emphasises the importance of integrating clinical context, morphological findings, and platelet function testing in evaluating unexplained bruising. Early recognition of APDE can prevent unnecessary investigations and interventions, particularly in adolescents. Morphological examination of the peripheral smear remains essential in guiding diagnosis in resource-limited and primary care settings.

MW-EP20: When Cancer and Cold Agglutinins Collide: A Diagnostic Challenge in Severe Anaemia

Nabilah Rameli[1], Norolhaswati Che Abd Hakim[1], Norazliaisam Mohemed Ali[1], Sumaiyah Adzahar[2]

[1]Department of Pathology, Hospital Dungun, Dungun, Terengganu, [2]Department of Pathology and Medical Laboratory, Faculty of Medicine, Universiti Sultan Zainal Abidin, Kuala Terengganu, Terengganu

Introduction: Cold autoimmune haemolytic anaemia (AIHA) is a rare form of haemolytic anaemia that can be primary or secondary to underlying conditions such as infections, autoimmune diseases, or malignancies. It is characterised by red cell agglutination at temperature below 37°c, leading to haemolysis and diagnostic interference. Nasopharyngeal carcinoma (NPC) is not commonly associated with cold AIHA, making such presentations diagnostically challenging, particularly in patients with unexplained severe anaemia. *Case Report*: We report a case of a 52-year-old male with advanced NPC presenting with severe anaemia (Hb 6.1 g/dL)

prior to planned chemotherapy. Initial full blood count (FBC) showed spuriously elevated MCV and red cell clumping, suggesting cold agglutinin interference. Peripheral blood smear confirmed marked RBC agglutination. Pre-warmed samples revealed corrected FBC parameters. Direct antiglobulin test was strongly positive for C3d and IgG, confirming cold AIHA. Crossmatching was complicated by autoantibody interference, with only one of five packed red cell units compatible after pre-warming. The patient received blood transfusion and proceeded with treatment. *Discussion*: This case underscores the diagnostic complexity of cold AIHA in oncology patients, particularly when anaemia may be initially attributed solely to malignancy. It highlights the importance of recognising analytical discrepancies such as spurious MCHC elevations and the critical role of pre-analytical handling in obtaining accurate *Results*. Timely diagnosis and multidisciplinary coordination were essential to ensure appropriate transfusion support and safe initiation of cancer treatment.

Acknowledgement: We would like to thank all staff of Pathology Lab Hospital Dungun for their endless effort.

MW-EP21: Toward Zero Rejection: Stewardship Hospital Melaka in Action!

Wan Aswani W. Yusof, Mazny Abu Bakar, Nor Ismalaili Desa, Rohana Ghazali, Fazilah Hassan, Noor Ayuni Paseri, Syaziah Ibrahim
Department Of Pathology, Hospital Melaka

Introduction: Pre-analytical errors are the most common laboratory errors, often leading to misdiagnosis, repeated testing, treatment delays, and increased healthcare costs. To address this, the Pathology Department of Hospital Melaka initiated the *"*Stewardship Toward Zero Rejection*"* programme, a year-long initiative from February 2024 to February 2025. This multidisciplinary effort involved liaison officers from the Medical, Paediatric, and Emergency & Trauma (ETD) Departments, aiming to reduce specimen rejection rates by improving compliance with pre-analytical standards. *Materials and Methods:* A cross-sectional study and a mixed-method approach was employed, including pre- and post-intervention audits, structured training sessions, and continuous monitoring of specimen rejection rates. The focus was on departments with high rejection rates due to clotted specimens, insufficient volume, and haemolysis from the ETD, wards C3 and C4 (Medical), and Special Care Nursery (SCN)/Neonatal Intensive Care Unit (NICU) and B2 (Paediatric ward). *Results:* Post-intervention compliance improved significantly: ETD (77.8% to 100%), SCN/NICU (54.3% to 91.2%), B2 (85.7% to 100%), C4 (82.9% to 100%), while C3 slightly declined (100% to 94.3%). Three-month average rejection rates showed mixed *Results*, with reductions in ETD (1.6% to 1.32%) and NICU/SCN (2.46% to 2.29%), but slight increases in C3, C4, and B2. *Discussion:* These findings demonstrate that focused training and collaboration approached significantly improve pre-analytical practice compliance. However, sustaining improvement remains challenging due to staffing limitations and high workloads. Continuous education, interdepartmental cooperation, and quality monitoring are essential to achieve and maintain zero rejection goals.

MW-EP22: Plasma Cell Leukaemia with Flowery-Like Cells: A Rare Diagnostic Challenge

Mas Aditya Senaputra[1,2], Dian Ariningrum[1,2], Amri Muzzammil[1], Bastomy Eka Rezkita[1]
[1]Clinical Pathology Department, Faculty of Medicine, Universitas Sebelas Maret Surakarta, [2]Clinical Pathology Laboratory, Dr. Moewardi General Hospital, Surakarta

Introduction: Plasma cell leukaemia (PCL) is a rare and aggressive haematologic malignancy classified under plasma cell neoplasms (PCNs). It represents an aggressive variant of multiple myeloma (MM) and is characterised by the clonal proliferation of malignant plasma cells in the peripheral blood. Diagnostic criteria include plasma cells exceeding 20% of leukocytes or an absolute count greater than 2×10^9/L in peripheral blood. *Case Report:* This case involves a 69-year-old male who presented with complaints of fatigue and lower back pain. Laboratory evaluation revealed anaemia, significant leucocytosis, thrombocytopenia, and 38% atypical cells in the peripheral blood. These atypical cells demonstrated distinctive flowery-like morphology—medium to large in size, with nuclear pleomorphism, pronounced convolutions and lobulations, coarse chromatin, and prominent nucleoli—these features highly reminiscent of neoplastic lymphocytes seen in Adult T-cell Leukaemia/Lymphoma (ATL). Serum protein electrophoresis showed a monoclonal spike. Bone marrow aspiration revealed normal cellularity with 7.8% plasma cells and more than 30% atypical cells resembling those found in peripheral blood. Immunophenotyping confirmed plasma cell origin, with positivity for CD38, CD56, CD138, and lambda light chain. *Discussion:* This case was diagnosed as primary PCL. The unusual cytologic appearance of the malignant cells complicated the diagnostic process, given their resemblance to T-cell malignancies. This required careful correlation of clinical findings, morphology, and immunophenotyping. Despite supportive measures, the patient's condition worsened rapidly, and he passed away on the seventh day of hospitalisation. This case underscores the need for early recognition of atypical morphological variants in PCL to avoid diagnostic delay and optimise clinical outcomes.

MW-EP23: Splice Site Mutations in the *F8* Gene: Genotype–Phenotype Correlation in Haemophilia A

Abdullah Aziz Nursaedah[1], Lam Kah Yuen[1], Lukman Izzatul Nabila[1], Mohamad Fadilah Nur Hakimah[1], Mat Ghani Siti Nor Assyuhada[1], Salam Syarini[1], Jradeh Bilal[3], Jaslowska Ewa[3], Yee Thynn Thynn[3], Gomez Keith[3], Mohd Sahid Ermi Neiza[1], Mohd Yasin Norafiza[1], Md Nor Azian Naila[1], Somasundram Suguna[1], Md Afandi Faridah[2], Mohd Fathullah Tun Maizura[2], Jamaludin Sufiza[2], Mat Yusoff Yuslina[1]
[1]Haematology Unit, Cancer Research Centre, Institute for Medical Research, National Institutes of Health, Ministry of Health, Selangor, Malaysia, [2]National Blood Centre, Ministry of Health, Kuala Lumpur, Malaysia, [3]Katharine Dormandy Haemophilia Centre and Thrombosis Unit, Royal Free London NHS Foundation Trust, London, United Kingdom.

Introduction: Haemophilia A (HA) is an X-linked recessive bleeding disorder caused by mutations within the *F8* gene, leading to coagulation factor VIII deficiency. Various types of mutations have been reported in HA, including intron 22 and intron 1 inversions, point mutations, insertions, and deletions. Splice site mutations are rarely reported but such mutations can impair

normal mRNA splicing, resulting in reduced or absent functional factor VIII. This study aimed to identify the splice site mutations in HA patients and correlate these mutations with the observed phenotypes. *Materials and Methods:* Genomic DNA was extracted from the blood samples of 203 HA patients. Genetic screening followed World Federation of Haemophilia guidelines. Severe and moderate patients with factor levels <1% to 3% were screened for intron 22 and intron 1 inversion by inverse-shifting PCR and multiplex PCR, respectively. Subsequently, PCR and Sanger sequencing were performed to identify the mutations within the *F8* gene exons and exon-intron borders. Patients' samples with factor levels >3% were directly subjected to *F8* gene sequencing. Negative cases were further screened for large deletions or duplications by multiplex ligation-dependent probe amplification. *Results:* Splice site mutations were detected in 6 out of 203 patients screened (2.96%), comprising 1 moderate and 5 severe HA cases. Ethnically, 2 patients were Chinese and 4 were Malay. None of the patients developed factor VIII inhibitors. The identified variants were c.5999-1G>A, c.670+1G>A, c.6116-1G>A, c.6429+2T>C, c.788-2A>G, and c.144-1G>T. Notably, c.6429+2T>C and c.788-2A>G variants were novel. *In silico* analyses by Franklin and VarSome classified these novel variants as likely pathogenic according to ACMG guidelines. *Discussion:* Splice site mutations, though rare, significantly contribute to HA pathogenicity and severity. This underscores the need for comprehensive genetic testing, including intronic regions, to ensure accurate diagnosis and inform clinical management.

MW-EP24: Evaluating AI for Peripheral Blood Film Analysis

Ramlah Mohamed Ibrahim [1], Hafiza Alauddin[1], Nor Farisah A. Razak[2]
[1]Department of Pathology, Faculty of Medicine, Universiti Kebangsaan Malaysia, Kuala Lumpur, Malaysia, [2]Department of Laboratory Diagnostic Services, Hospital Canselor Tuanku Muhriz, Universiti Kebangsaan Malaysia, Kuala Lumpur, Malaysia

*Introduction***:** Peripheral blood film (PBF) examination is fundamental in haematology but is limited by manual effort and observer variability. The SigTuple AI100, an artificial intelligence-assisted platform, aims to automate and standardise PBF analysis. This study evaluates its diagnostic accuracy against manual microscopy. *Materials & Methods***:** One hundred peripheral blood films (20 normal & 80 abnormal) were analysed using SigTuple AI100 and manual microscopy. Correlation coefficients (R^2) and Bland-Altman plots were applied for statistical comparison. *Results:* Strong correlations were observed for neutrophils ($R^2 = 0.92$), lymphocytes ($R^2 = 0.89$), and monocytes ($R^2 = 0.87$) with minimal bias seen in Bland-Altman plots. Moderate correlations were found for eosinophils ($R^2 = 0.75$), basophils ($R^2 = 0.68$), and blasts/abnormal lymphoid cells ($R^2 = 0.65$). AI demonstrated 100% sensitivity for normocytes, but lower sensitivity for microcytes (78.8%) and macrocytes (23%). Fragmented red cells had moderate sensitivity (50%) but high specificity (100%). Platelet counts correlated well with FBC analysers ($R^2 = 0.98$), though specificity was affected by debris misclassified as clumps. *Discussion:* SigTuple AI100 shows high concordance with manual microscopy for common leukocyte subsets and platelet estimation. Moderate correlation for eosinophils, basophils, and blasts/abnormal lymphoid cells may stem from limited representation of rare cells, morphological overlap with other cell types, and difficulty distinguishing immature forms. Technical factors such as slide quality, staining variation, and variability in expert annotations may also contribute. Additionally, analysis is limited to the monolayer region, possibly missing atypical cells. Broader training datasets and algorithm refinement are needed to improve accuracy for rare and morphologically complex cells.

MW-EP25: A Decade in Review: Epidemiological Trends of Acute Lymphoblastic Leukaemia Cases Referred to Institute for Medical Research

Nadirah Zainal Abidin[1], Azian Naila Md Nor[1], Noraizan Ahmad[1], Rafizie Rafiq Adha[1], Nabilah Huda Abd Rahman[1], Heather John @Nicholas[1], Nurul Aqilah Ali[1], Ermi Neiza Mohd Sahid[1], Norafiza Mohd Yasin[1], Suguna Somasundram[1], Nor Rizan Kamaluddin[1], Ezalia Esa[2], Yuslina Mat Yusoff[1]
[1]Haematology Unit, Cancer Research Centre, Institute for Medical Research, National Institutes of Health, Ministry of Health Malaysia, Selangor, Malaysia, [2]Virology Unit, Infectious Disease Research Centre, Institute for Medical Research, National Institutes of Health, Ministry of Health Malaysia, Selangor, Malaysia

Introduction: Acute Lymphoblastic Leukaemia (ALL) is a haematological malignancy, requiring early and accurate diagnosis for effective clinical management. This study evaluated the frequency and demographic distribution of ALL cases referred to the Haematology Unit, Institute for Medical Research (IMR), Malaysia. *Materials and Methods:* A retrospective review of 1,525 referral samples received from 2017 to 2024 was conducted. Demographic data including age, sex, and ethnicity were analysed alongside molecular test *Results*. Fusion gene detection was performed using the Quandx® PCR-based kit to identify leukaemia-associated chromosomal translocations. *Results:* Of the samples analysed, 63% (n=960) were paediatric (0-14years old) and 37% (n=565) adult; 58% were male and 42% female. Ethnic distribution included Malay (60.4%), Chinese (15.4%), Indian (4.7%), Others (18.5%), and non-Malaysians (2%). Gene fusions were detected in 30% (n=458), not detected in 64%
(n=972). ALL cases increased notably from 23 in 2017 to 85 in 2024. Among 290 fusion- positive cases, the most frequent was t(12;21)(p13;q22) (60%), followed by t(1;19) (11.4%), t(4;11) (9.7%), *TAL1* deletion, t(6;11), t(10;11), and del(1)(p32). *Discussion:* The predominance of t(12;21) underscores its diagnostic relevance in paediatric ALL, while the variety of other fusions highlights disease heterogeneity. The increasing number of samples detected for ALL reflects improved clinical awareness and increased use of molecular diagnostics. The genetic abnormalities are crucial for risk prognostication and management of patient. For example, B-ALL t(12;21)(p13;q22) associated with favourable prognosis. These findings underscore the importance of expanding access to molecular testing and integrating genetic profiling into diagnostic pathways and prognostication to enable timely and effective treatment.

MW-EP26: Acute Erythroid Leukaemia with *NPM1::MLF1* Fusion: A Rare Case Highlighting Morphologic and Molecular Complexity

Azian Naila Md Nor[1], Ermi Neiza Mohd Sahid[1], Norafiza Mohd Yasin[1], Ezalia Esa[1], Suguna Somasundram[1], Nadirah Zainal Abidin[1], Nurul Aqilah Ali[1], Liew Chiu Yee[2], Mashitah Abdul Hamid[3], Hafizah Hashim[3], Yuslina Mat Yusoff[1] *[1]Haematology Unit, Cancer Research Center, Institute for Medical Research, National Institutes of Health, Selangor, Malaysia, [2]Jabatan Patologi, Hospital Pulau Pinang, Pulau Pinang, Malaysia, [3]Jabatan Patologi, Hospital Sultanah Bahiyah, Kedah, Malaysia*

Introduction: Acute erythroid leukaemia (AEL) is a rare and aggressive subtype of acute myeloid leukaemia (AML), characterised by predominant erythroid proliferation and frequent cytogenetic abnormalities. The *NPM1::MLF1* fusion, recently classified under "AML with other defined genetic alterations" in the fifth edition of the WHO Classification of Haematolymphoid Tumours, is exceptionally rare and remains poorly characterised in the context of AEL. We present a case of AEL harbouring this fusion, emphasising the diagnostic challenges and the value of integrated morphologic and molecular assessment. *Case Report*: A 58-year-old Chinese man presented with dizziness and chest pain. Full blood picture revealed severe anaemia, thrombocytopenia, and 4% circulating blasts. Bone marrow aspirate was markedly hypercellular, with erythroid precursors comprising approximately 90% of nucleated cells, including 10% proerythroblasts with bluish, blebbed, and vacuolated cytoplasm. Marked dyserythropoietic features were observed, such as intranuclear bridging and multinuclearity. Bone marrow trephine biopsy confirmed erythroid predominance with strong Glycophorin A positivity. Immunohistochemistry showed cluster positivity for E-cadherin and CD117, with no significant increase in CD34 expression. Molecular testing using real-time polymerase chain reaction (qPCR) revealed the rare *NPM1::MLF1* fusion, consistent with cytogenetic findings. *Discussion:* This case illustrates the diagnostic complexity of AEL and underscores the essential role of detailed morphologic evaluation combined with molecular profiling. The rare *NPM1::MLF1* fusion points to a broader and more diverse genetic landscape in AEL than previously recognised. It contributes to the evolving understanding of AEL and highlights the need for further studies to clarify its prognostic significance and role in leukaemogenesis.

MW-EP27: A Diagnostic Challenge in Myelodysplastic/myeloproliferative Neoplasms Overlap Syndrome: A Case Report

Wan Asmuni Wan Mohd Saman, Zalizah Khalid, Fatmawati Kamal
Department of Pathology, Faculty of Medicine, Universiti Teknologi Mara, Sungai Buloh,Selangor, Malaysia, Department of Clinical Diagnostic Laboratory, Hospital Al-Sultan Abdullah, Puncak Alam, Selangor, Malaysia

Introduction: Myelodysplastic/myeloproliferative neoplasms (MDS/MPN) represent a group of clonal haematological malignancies that exhibit overlapping features of both myelodysplastic syndromes and myeloproliferative neoplasms, making their classification particularly challenging. *Case report*: We present a case that highlights the diagnostic dilemma in differentiating between MDS/MPN with neutrophilia (formerly known as atypical chronic myeloid leukaemia, aCML) and chronic myelomonocytic leukaemia (CMML). A 70-year-old male ex-smoker with a medical history of hypertension, dyslipidaemia, and gouty arthritis presented with a two-month history of chronic cough, night sweats, weight loss, and splenomegaly. Laboratory investigations revealed marked leucocytosis with a white blood cell count of 141.1 × 10 /L, haemoglobin of 10.8 g/dL, and platelet count of 110 × 10 /L. Peripheral blood film showed dysplastic neutrophils, myelocytes, 8% monocytes, and 4% circulating blasts. Serum lactate dehydrogenase was elevated at 1458 U/L. Bone marrow aspirate demonstrated hypercellularity with granulocytic proliferation and trilineage dysplasia, raising suspicion for MDS/MPN. Trephine biopsy confirmed hypercellular marrow with peripheral blood contamination. Molecular analyses were negative for BCR::ABL1 and *JAK2* V617F mutations. Cytogenetic analysis identified monosomy 7, with no other chromosomal abnormalities. DNA sequencing was not performed due to financial constraints. The patient was referred to another centre for further management. *Discussion*: This case highlights the complexities in subclassifying MDS/MPN disorders, especially when molecular profiling is limited or unavailable. Accurate diagnosis is essential for appropriate prognostication and treatment planning, highlighting the importance of integrating clinical, morphologic, cytogenetic, and molecular data in MDS/MPN classification.

MW-EP28: BPDCN with Non-Classical Presentation: Expanding the Clinical and Diagnostic Spectrum

Fairuz Amil[1], Nadia Arina Zainudin[1], Nor Fadhilah Shafiii[1], Suryati Hussin[1], Nor Azah Farhah Abdul Ayah @ Aziz[2], Mimi Azreen Abdullah[1]
[1]Haemotology unit, Pathology Department, Hospital Raja Perempuan Zainab II, Kota Bharu Kelantan, [2]Pathology and Transfusion Department, Hospital Tanah Merah, Tanah Merah Kelantan

Introduction: Blastic Plasmacytoid Dendritic Cell Neoplasm (BPDCN) is a rare and highly invasive haematological malignancy that originates from plasmacytoid dendritic cells. Despite it being an aggressive disease it usually starts with an indolent skin lesion. It may involve the bone marrow, lymph nodes, peripheral blood and other organs. *Case Report*: A 26-year-old Malay male presented with fever, nasal blockage, dysphagia, and cervical swelling for 1 month. Clinical examination revealed multiple cervical lymphadenopathies and hepatosplenomegaly. Complete blood count showed leucocytosis, anaemia, and thrombocytopenia, with 30% circulating abnormal mononuclear cells. Bone marrow aspiration revealed 70% blastoid looking cells, described as moderate to large with abundant bluish agranular cytoplasm containing microvacuoles and cytoplasmic projections. The nuclei were round with finely dispersed chromatin and prominent nucleoli. Flow cytometry analysis demonstrated a population of cells expressing CD45, moderate SSC, HLA-DR, CD33, CD7, CD4, CD123 and negative for other myeloid, monocytic, lymphoid and immature markers. Trephine biopsy revealed hypercellular marrow with diffused infiltrate of atypical cells positive for CD4, CD56, and CD68. The Ki-67 was greater than 70%. Lymph node biopsy showed similar findings and positive for additional markers of LCA, CD7, CD123, TCL1 and CD33. Cytogenetic analysis revealed complex karyotypes with 8q24/MYC rearrangement, relatively

common in BPDCN. *Discussion and conclusion*: The unique aspect of this case is that the patient was young and presented with extracutaneous involvement. It is extremely rare for BPDCN cases to present without skin lesions. The morphologic features of the neoplastic cells, along with the expression of CD4 and CD56, and the presence of markers specific to plasmacytoid dendritic cells (CD123 and TCL1), in the absence of lymphoid and myeloid lineage-associated antigens, support the diagnosis of BPDCN and help exclude other diagnostic mimickers.

MW-EP29: A Rare and Unique Childhood Hematopoietic Disorder: A Case Report of Juvenile Myelomonocytic Leukaemia in a 4-year-old girl.

Siti Fatimah Mohamed Kamaruzzaman, Nabihah Mohd Shakri ,Nornattasa Mohmad Sallih, Rathiga Krishnamani
Haematology Unit, Department of Pathology, Hospital Sultan Ismail Johor Bahru

Introduction: Juvenile Myelomonocytic Leukaemia (JMML) is a rare, clonal abnormality of pluripotent haematopoietic stem cells of early childhood with incidence of 1.2 per million children per year. JMML is now classified as a MPN of childhood. *Case Report:* We report a case of a 4-year-old girl presented with 10-day history of fever associated with generalized petechial rashes and marked hepatosplenomegaly. Full blood picture demonstrates bicytopenia with leucoerythroblastic picture and presence of 4-5% circulating blasts in the background of neutrophilia and monocytosis with dysplastic neutrophils. Diagnostic bone marrow examination showed hypercellular marrow with myelomonocytic hyperplasia in the background of trilineage dysplasia with presence of 3-4% blasts and increased in histiocytes with prominent Haemophagocytic activity. Further investigations reveal high Hb F level (59.8%) and absence of *BCR::ABL1* fusion gene. Later, the gene mutation analysis *Results* confirmed the presence of *RAS* pathway mutations. *Discussion:* The diagnosis of JMML can be challenging as the clinical and morphologic findings may resemble other myeloproliferative neoplasms such as chronic myeloid leukaemia or mimic viral infections. Our present case is representative of an exemplary JMML case that fulfils all categories of the WHO diagnostic criteria. Clinical characteristics, morphological and molecular studies are required for an accurate diagnosis of JMML, risk stratification and therapy methods. Considering the aggressiveness of this disease with high-risk features that our patient had, HSCT was planned while Azacitidine bridging therapy was initiated.

MW-EP30: Morphological Variants of Plasma Cell Myeloma: A Case Series of Diagnostic Mimics.

Sarah Abdul Halim, Dhamirah Nazihah Mohd Nasiruddin, Norlelawati A Talib
Department of Pathology and Laboratory Medicine, Kulliyyah of Medicine, International Islamic University Malaysia, Kuantan, Pahang, Malaysia

Introduction: Plasma cell myeloma (PCM) exhibits notable morphological heterogeneity beyond the classical plasma cell phenotype. Recognition of these variants is critical for accurate diagnosis and avoiding misclassification, particularly when they mimic other haematological or non-haematological malignancies. We report three PCM cases with distinct morphological features posing diagnostic challenges. *Case Series:* Case 1: A 65-year-old woman with multiple lytic skull lesions. Bone marrow examination revealed large cells with irregular, lobulated nuclei and abundant basophilic cytoplasm with vacuoles and blebs, morphologically resembling monocytes. Immunohistochemistry (IHC) demonstrated CD138 positivity, kappa light chain restriction, and aberrant CD56 and CD117 expression. Case 2: A 62-year-old woman with renal impairment and reversed albumin-to-globulin ratio showed marrow infiltration by plasmablasts with high nuclear-to-cytoplasmic ratios, dispersed chromatin, and prominent nucleoli. Trephine biopsy revealed sheets of CD138-positive plasma cells with kappa light chain restriction. Case 3: A 66-year-old man with relapsed PCM and a concurrent neuroendocrine tumour of the nasopharynx demonstrated bone marrow involvement by pleomorphic, large sized, basophilic cells with cytoplasmic blebs. An initial impression of metastatic infiltration was revised following trephine biopsy, which showed sheets of CD138+, MUM1+, CD56+ plasma cells with kappa restriction; cytokeratin AE1/AE3 was negative. *Discussion:* This case series highlights the morphological diversity of PCM, which may mimic other neoplastic processes. Reliance on morphology alone may lead to misclassification. A combination of careful morphological assessment, supported by immunophenotyping and clinical correlation is essential for accurate diagnosis and prevent delays in treatment.

MW-EP31: Association of ABO and Rhesus D Blood Groups with Risk of COVID-19 Infection: A Case-Control Study in a Single Institution

Muruges A/L Perumal[1], Mohd Nazri Hassan[2], Noor Haslina Mohd Noor[2]
National Cancer Institute, Putrajaya, [2]Department of Haematology, School of Medical Sciences, USM, Kubang Kerian, Kelantan

Introduction: ABO blood groups have been associated with susceptibility to various infectious and non-infectious diseases. Emerging evidence suggests a potential link between ABO blood types and the risk of COVID-19 infection. However, data from Malaysia on the relationship between ABO or Rhesus D (RhD) blood groups and COVID-19 remains limited. This study aimed to investigate the association of ABO and RhD blood groups with the risk of COVID-19 infection. *Material and Methods:* This case-control study included 214 confirmed COVID-19 patients and 363 non-COVID-19 patients admitted to Hospital Putrajaya between 2022 and 2024. Blood grouping for ABO and RhD was performed using the tube method with monoclonal anti-A, anti-B, and anti-D reagents. ABO groups were confirmed by both forward and reverse typing. Patients with a recent history of red cell transfusion were excluded. Multivariate logistic regression (MLR) was used to assess the association between blood groups and COVID-19 infection, with statistical significance set at $p<0.05$. *Results:* Most participants were Malay (81.1%), with a mean age of 46.2 ± 17.1 years. Blood group O was the most common (41.4%), followed by A (26.5%), B (24.5%), and AB (7.5%). Nearly all participants were RhD-positive (99.8%). MLR analysis indicated that O blood groups (adjusted OR=0.595, p=0.004), were significantly associated with a lower risk (40.5% lower odds) of COVID-19 infection compared to non-O group. RhD status showed

no significant association. *Discussion:* Blood Group O may confer a protective effect against COVID-19, whereas non-O group appears to be associated with a higher risk. These findings highlight a potential immunohaematologic role in COVID-19 susceptibility.

MW-EP32: Acute Megakaryoblastic Leukaemia in Adult: Diagnostic Challenge in The Absence of Classical Markers and Association with Bone Marrow Fibrosis

Maskur Fahmi Adi Bhaskoro[1], Mas Aditya Senaputra[2], Dian Ariningrum[2]
[1]*Clinical Pathology Resident of Faculty of Medicine of Sebelas Maret University / Dr Moewardi General Hospital, Surakarta,* [2]*Clinical Pathology Department of Faculty of Medicine of Sebelas Maret University / Dr Moewardi General Hospital, Surakarta*

Introduction: Acute Megakaryoblastic Leukaemia (AMKL) is a rare subtype of acute myeloid leukaemia (AML), accounting for only 1–2% of adult AML cases. It is characterised by ≥20% blasts with megakaryocytic differentiation and expression of specific markers such as CD41, CD61, or CD42b. Fibrosis may accompany AMKL due to increased megakaryocyte proliferation. *Case Report:* A 31-year-old woman presented with generalized weakness and abdominal enlargement persisting for 4 months. Physical examination revealed anaemic conjunctiva and splenomegaly. Haematological analysis showed anaemia (haemoglobin of 6.8 g/dL), leucocytosis (white blood cell of $24\times10^3/\mu L$), and thrombocytosis (platelet of $361\times10^3/\mu L$). Tear drop shaped red blood cell, megakaryoblast and micromegakaryocyte were found in peripheral blood smear (PBS). Bone marrow aspiration (BMA) revealed 38.2% megakaryoblasts and 3.8% promegakaryocytes. Immunophenotyping showed CD34(+), CD13(+), CD33(+), HLA-DR(+), and CD36(+), while cyMPO and CD41 were negative. Markers for monocytic (CD14 and CD64) and erythroid (CD21 and CD235a) lineages were absent. CD61 and CD42b as another marker for megakaryocytic lineage were not available. Bone marrow biopsy analysis confirmed bone marrow fibrosis. *Discussion:* The diagnosis of AMKL was made based on clinical presentation, morphology, immunophenotyping, and exclusion of other AML subtypes. Although CD36 is not specific marker for megakaryocytic lineage, the absence marker for another myeloid lineage leukaemia, monocytic leukaemia and erythroid leukaemia, along with morphology in PBS and BMA supported the diagnosis of AMKL. This case highlighted the diagnostic complexity of AMKL, especially when classical markers are absent. This case also illustrates the association between AMKL and marrow fibrosis.

MW-EP33: Demographic and Severity Profile of Haemophilia B Patients in Malaysia

Lukman Izzatul Nabila[1], Abdullah Aziz Nursaedah[1], Lam Kah Yuen[1], Mohamed, Roslida[1], Md Afandi Faridah[2], Mohd Fathullah Tun Maizura[2], Esa Ezalia[1], Mat Yusoff Yuslina[1]
[1]*Haematology Unit, Cancer Research Centre, Institute for Medical Research, National Institutes of Health, Ministry of Health, Selangor, Malaysia,* [2]*National Blood Centre, Ministry of Health, Kuala Lumpur, Malaysia.*

Introduction: Haemophilia B (HB) is an X-linked recessive bleeding disorder caused by coagulation factor IX deficiency. It results in impaired blood clotting and a tendency for spontaneous or prolonged bleeding, which can lead to joint damage, muscle hematomas, and life-threatening haemorrhages if untreated. The severity of bleeding symptoms typically correlates with the residual activity of factor IX. Management includes replacement therapy with factor IX concentrates to prevent or control bleeding episodes. This study analysed blood samples from 33 HB patients referred by government hospitals between 2014 and 2021. Factor IX levels were determined by the referring institutions. The cohort was multiethnic, comprising 57.6% Malay, 21.2% Chinese, 12.1% Indian, and 9.1% from other ethnic groups. Patients ranged in age from newborn to 33 years and were categorized as severe (n=22), moderate (n=6), or mild (n=5) based on factor IX levels. *Materials and Methods:* Demographic and clinical data, including age, ethnicity, and disease severity, were collected from the patient records. *Results:* The majority of patients were Malay, followed by Chinese and Indian ethnicities, reflecting Malaysia's population diversity. Severe HB was the most common clinical category. Only one severe patient developed an inhibitor to factor IX replacement therapy**.** *Discussion:* This study highlights the demographic profile of HB patients in Malaysia, emphasising ethnic and age distribution within the cohort. Understanding these demographics is essential for healthcare planning and resource allocation.

MW-EP34: Severe Marrow Dysplasia associated with Vitamin B12 Deficiency in a 2-Year-Old Boy

Elsiana Laurencia[1], Sienny Linawaty[2], JB Suparyatmo[3], Brigitte Rina Aninda Sidharta[3], Dian Ariningrum[3], M. I. Diah Pramudianti[3], Mas Aditya Senaputra[3]
[1]*Clinical Pathology Resident of Faculty of Medicine of Sebelas Maret University / Dr Moewardi General Hospital, Surakarta,* [2]*Clinical Pathology Department of Faculty of Medicine of Sebelas Maret University, Surakarta,* [3]*Clinical Pathology Department of Faculty of Medicine of Sebelas Maret University / Dr Moewardi General Hospital, Surakarta*

Introduction: Bone marrow dysplasia is characterised by the inability of the bone marrow to produce blood cells properly. One of the causes of dysplasia, especially in children, is vitamin B12 deficiency, which may result in pancytopenia. *Case Report*: A 2-year-old boy presented with intermittent fever and pallor that had persisted for one year. The patient's older brother was deceased at 2 years old with similar symptoms and diagnosed with myelodysplasia. The patient was anemic, acral hyperpigmented, and showing signs of malnutrition. Complete blood count showed pancytopenia with Hb level 4.6 g/dL, WBC count $3.6\times10^3/\mu L$, and platelet count $25\times10^3/\mu L$. Peripheral blood smear revealed macrocytosis, normoblastaemia, and hypersegmented neutrophils. Bone marrow aspiration showed severe dysplasia of more than 30% in all three hematopoietic lineages suggesting the diagnosis of Myelodysplastic Syndrome (MDS), especially in erythropoietic lineage (38.5%) and granulopoietic lineage (16.7%). Further investigation revealed decreased serum vitamin B12 level (<125 pg/mL). *Discussion*: The haematologic manifestations of vitamin B12 deficiency often mimic other serious conditions such as myelodysplastic syndrome (MDS), with dysplastic changes in the bone marrow that can lead to diagnostic confusion. Therefore, additional tests such as serum vitamin B12 levels, flow cytometry, and gene mutation that leads to congenital vitamin B12 deficiency are needed. If these tests are not available, a blind supplementation of vitamin B12 may be considered, and the response can help to confirm the diagnosis.

MW-EP35: Acute Myeloid Leukaemia with Unusual Presentation and Difficult Histiologic Pattern Mimicking Histiocytic Sarcoma

Nur Juliana Idris[1] , Nur Aliaa Anuar[1] , Asmawiza Awang[2]
[1]Department of Pathology, Hospital Tuanku Ja'afar Seremban, Negeri Sembilan, Malaysia
[2]Department of Pathology, Hospital Kuala Lumpur, Wilayah Persekutuan Kuala Lumpur, Malaysia

Introduction: Cutaneous presentation of Acute Myeloid Leukaemia (AML) is rare. Distinguishing AML with monocytic differentiation (AMoL) from Histiocytic Sarcoma (HS) can be challenging as both express monocytic markers. *Case Report:* We herein report a case of a 33-year-old female patient who presented with fever and purplish nodular lesions over her trunk and face. No palpable hepatosplenomegaly or lymphadenopathy. Peripheral blood film identified ~20% blastic-looking cells with irregular nuclei, slightly open chromatin pattern and ample basophilic cytoplasm. Bone marrow aspiration was dry tap. Peripheral blood immunophenotyping identified ~0.5% of the myeloblasts population. Trephine biopsy showed diffuse infiltration by sheets of malignant cells. The cells were round to indented nuclei with condensed chromatin patterns and abundant cytoplasm. They were strongly positive for CD45, CD68 and lysozyme associated with a high proliferative index (Ki67 > 80%). Some tumour cells were focally positive for Myeloperoxidase (MPO). Other lineage-specific and additional markers including CD3, CD5, CD 20, CD79 alpha, PAX5, CD138, CD30,CD 123, CD1a and S100 were all negative. They lacked expression of all immature markers ie CD34, CD117, TdT and CD99. Her skin biopsy was preliminarily reported as HS. However, this diagnosis was revised, and the final diagnosis was AMoL based on the morphology and positive MPO stain. *Discussion:* AMoL was misreported as HS from the skin biopsy in this case due to its unusual presentation and less definitive immunohistochemistry pattern. The morphology of the cells described was more characteristic of AML than HS, in which HS often shows greater pleomorphism. Other myeloid-specific stains, such as CD13 and CD33 (not performed in this case), may further support the diagnosis.

MW-EP36: Hereditary Spherocytosis Masquerading as Congenital Dyserythropoietic Anaemia: A Genetic Revelation

Intan Noorliza Mohd Sopani[1], Liana Yusof[2], Norazlina Azman[3], Mimi Azura Aziz[3], Zulaiha Muda[2], Chee Weng Yan Sabrina[4]
[1]Department of Pathology, Faculty of Medicine and Health Sciences, Universiti Putra Malaysia, Serdang, Selangor, Malaysia, [2] Department of Paediatric Haemato-oncology, Hospital Tunku Azizah, Kuala Lumpur, Malaysia, [3]Department of Pathology, Hospital Tunku Azizah, Kuala Lumpur, Malaysia, [4]Department of Pathology, Hospital Teluk Intan, Teluk Intan, Perak, Malaysia.

Introduction: Congenital dyserythropoietic anaemia (CDA) and hereditary spherocytosis (HS) are inherited red cell disorders with overlapping features, often leading to diagnostic uncertainty. Genetic testing is a critical tool to differentiate these conditions. We report a case initially managed as CDA but later genetically confirmed as HS due to a pathogenic **SLC4A1** mutation. *Case Presentation:* A 2-year-6-month-old Malay girl presented with incidental pallor on day 15 of life. Haemoglobin was 4.6 g/dL initially with reticulocytosis (28.8%) and a leucoerythroblastic film with spherocytes. Negative Coombs test; other cell lines normal. She is transfusion-dependent (3–4 weekly) to maintain Hb >8 g/dL. Both parents had normal FBCs and Hb analyses. Bone marrow aspiration at 10 months revealed a mildly hypercellular marrow with marked erythroid hyperplasia and dyserythropoiesis (binucleation, karyorrhexis), suggestive of CDA. A hereditary haemolytic anaemia gene panel (Invitae) at 18 months of age revealed no CDA-related mutations. Instead, a heterozygous pathogenic SLC4A1 variant (c.88C>T; p.Gln30) was identified in both the patient and her father, along with a silent variant of uncertain significance (c.2511G>A). This mutation is consistent with autosomal dominant HS. South East Asian Ovalocytosis (SAO), which is commonly associated with SLC4A1**,** was excluded with no Band 3 deletion in molecular testing. Distal renal tubular acidosis features were absent. *Discussion/Conclusion*: This case is illustrative of the challenges of neonatal anaemia diagnosis. Bone marrow examination was suggestive of CDA, but genetic examination confirmed the diagnosis of HS. Early molecular diagnosis is essential to support accurate diagnosis and guide appropriate management.

MW-EP37: CML's Secret Weapon: Myeloid Sarcoma Emergence

Mohamad Saiful Ridzuan Mohd Khairi[1], Ramlah Mohamed Ibrahim[1], Azlin Ithnin[1], Qhasmira Abu Hazir[2], Lailatul Hadziyah Mohd Pauzy[2]
[1]Department of Pathology, Faculty of Medicine, Universiti Kebangsaan Malaysia, Kuala Lumpur, [2]Department of Diagnostic Laboratory Services, Hospital Canselor Tuanku Muhriz, Universiti Kebangsaan Malaysia, Kuala Lumpur

Introduction: Chronic Myeloid Leukaemia (CML) is a clonal disorder originating in the hematopoietic stem cells which is characterised by presence of the Philadelphia (Ph) chromosome. Myeloid Sarcoma (MS) on the other hand is a rare neoplasm composed of immature myeloid cells in extramedullary tissue has been reported in rare cases of CML. We report a case of CML in chronic phase (CP) complicated with MS over the left lower limb. *Case Report:* A 35-year-old Malay male newly diagnosed with CML in CP not compliance to imatinib presented with continuous fever. He was treated for bacteraemia and left lower limb panniculitis. However, despite high-end antibiotics, his lower limb showed no improvement, and the patient subsequently developed neutropenic sepsis. A skin punch biopsy of the left leg reported as atypical cells positive for MPO and CD117, which is consistent with myeloid sarcoma (AML) with underlying acute inflammation. The bone marrow aspirate and trephine biopsy shows hypercellular marrow with no excess of blasts and cytogenetic analysis shows t(9;22)(q34;q11) with no additional rearrangement. *Discussion:* Myeloid sarcoma may precede, coincide with, or follow the diagnosis of CML. Its presentation in soft tissues can mimic infections or inflammatory conditions, delaying diagnosis. Studies have also suggested that MS might be the first symptom of progressive illness prior to haematological progression of CML. This case underscores the importance of clinical suspicion and prompt identification in persistent, non-resolving lesions in CML patients.

MW-EP38: Verification of Rosner Index Cut-offs in Coagulation Mixing Test

Naqibah Abdul Malik, Nabeelah Abdul Khalid, Teng Jia Ying
Haematology Unit, Pathology Department, Hospital Pulau Pinang, Malaysia

Introduction: Mixing test is a relatively simple procedure used in a laboratory as a first-line investigation following an abnormal coagulation screening test. There are several methods available to interpret mixing studies as having either "corrected" or "not corrected". One commonly used method is the index of circulating anticoagulant (ICA) also known as the "Rosner Index" (RI) which was initially developed for Lupus Anticoagulant (LA) investigation. This study aimed to establish optimal cut-off values for the RI with the current analyser and reagent used. *Materials and Methods:* A retrospective study was conducted on 222 patient samples collected from 25 patients with LA and 197 from individuals with haemophilia, both newly diagnosed and known cases. This includes haemophilia patients with (n=17) and without inhibitors (n=180). *Results:* The result demonstrated that RI above 16% consistently indicated the presence of inhibitor, whereas RI below 13% strongly suggested factor deficiency. For cases with RI between 13% and 16%, it is recommended to either repeat test or to proceed with factor assay, inhibitor assay or LA test. *Discussion:* The Rosner Index offers the advantage of simple calculation, and an interpretation applied against a given target value. However, its main disadvantage is the necessity for each laboratory to establish its own cut-off to differentiate factor deficiency from inhibitors. These findings provide a clearer framework for interpreting mixing test *Results*, thereby improving diagnostic accuracy and guiding subsequent patient management.

MW-EP39: Rouleaux Formation in Acute Myeloid Leukaemia with COVID-19: A Diagnostic Clue to Co-infection

Kaveta Balasubramaniam, Zainina Seman, Sabariah Md Noor
Department of Pathology, Faculty of Medicine and Health Sciences, UPM, 43400 Serdang, Malaysia.

Introduction: Patients with acute myeloid leukaemia (AML) are immunocompromised and thus highly susceptible to opportunistic infections such as COVID-19. Blood film morphology may reflect not only haematologic malignancy but also additional systemic pathology, particularly in the setting of infection. *Case Report:* A 66-year-old man with diabetes mellitus presented with incidental initial blood parameters: Hb 9 g/dL, WBC 20 × 10^9/L, platelets 16 × 10^9/L. Bone marrow showed 29% blasts positive for MPO, CD34, CD117, CD13, HLA-DR, CD36, CD33, and CD123, confirming AML. Two weeks later, before chemotherapy, he developed a fever and lethargy. COVID-19 PCR was positive. Repeat blood film showed 68% blasts and an additional prominent rouleaux formation. Multiple myeloma, chronic liver disease, anaemia of chronic disease, and connective tissue disorders were excluded. His condition deteriorated with hematemesis, abdominal distension, and rectal bleeding. Endoscopy revealed portal hypertensive gastropathy and oesophageal varices. *Discussion:* This case demonstrates rouleaux formation as a significant additional morphological finding in AML with COVID-19 co-infection. The appearance of rouleaux in AML patients should alert clinicians to investigate co-existing disorders, as it may indicate disease progression or concurrent complications. The rouleaux likely resulted from viral-induced inflammatory changes affecting red cell surface properties. Unfortunately, the patient was referred to another centre, preventing follow-up on treatment outcomes. However, this case highlights the urgent need for heightened vigilance in morphological assessment when AML patients develop concurrent infections. Recognition of evolving blood film morphology provides valuable diagnostic insights and may prompt timely intervention in managing these complex cases.

MW-EP40: Development of Fragmented Red Cell Flagging in Haematology Analyser without Reticulocyte Channel

Kaveta Balasubramaniam[1], Nor Ayuni Khuzaimah[1], Nabeelah Abdul Khalid[1], Teng Jia Ying[1], Farisyah Syakirah Mohd Zain[2], Tuan Hulwani Tuan Mohamad[2]
[1]Haematology Unit, Department of Pathology, Hospital Pulau Pinang, Ministry of Health Malaysia, Georgetown, Penang, Malaysia, [2]Haematology Unit, Department of Pathology, Hospital Seberang Jaya, Ministry of Health Malaysia, Penang, Malaysia

Introduction: The presence of fragmented red blood cells, or schistocytes, plays a fundamental role in diagnosing thrombotic microangiopathy (TMA). Haematology analysers equipped with a reticulocyte (RETIC) channel can generate flagging for these findings. However, reticulocyte channel is not available in smaller laboratory haematology analyser due to lower workload, additional reagent required, and therefore extra cost incurred. This study was aimed to develop an algorithm to generate flagging using information from the RBC/PLT impedance channel. *Methods*: A total of 324 EDTA samples were randomly selected and analysed for complete blood counts (CBC) using RBC/PLT impedance, HGB, WNB, DIFF, and RETIC channels. Two sets of blood smears were prepared for manual schistocyte examination under an optical microscope by two independent examiners. Samples with significant RBC fragments (≥1% schistocytes confirmed by manual microscopy) were selected, and their impedance channel characteristics were analysed. *Results:* Cases with significant fragments exhibited distinct characteristics, including abnormal RBC and PLT histogram patterns. When these features were combined with parameters such as MCH <80 as a new algorithm and applied to a set of retrospective data, the algorithm demonstrated strong performance for flagging samples with fragments. The positive predictive value (PPV) was 86.8%, the negative predictive value (NPV) was 94%, the sensitivity was 70.2%, the specificity was 97.8%, and the accuracy was 93%. Prospective validation also showed acceptable performance. *Conclusion*: This study highlights a cost-effective method to screen for RBC fragments, thus enhancing early TMA diagnosis in resource-limited settings.

MW-EP41: Howell-Jolly Body-like Inclusions in Neutrophils in Immunosuppressed Patient: A Case Report

Azizan Hanny Azahar, Wan Zuhairah W Embong, Wan Amal Hayati Wan Hassan, Nor Ainiza Mansor, Azly Sumanty Ab Ghani, Zakiah Nurasyikin Mohamed Halim

Haematology Unit, Department of Pathology, Hospital Sultanah Nur Zahirah
Introduction: Howell-Jolly bodies are nuclear remnants usually found in red blood cells. Howell-Jolly body-like inclusions (HJBLIs) are detached nuclear fragments resembling Howell-Jolly bodies but are found within neutrophils. These have been reported in association with conditions such as HIV, COVID-19, immunosuppression and Myelodysplastic Syndrome. We present a case with Howell-Jolly body-like inclusions in an immunosuppressed patient. *Case Report:* A 10-year-old Malay girl with underlying T-cell acute lymphoblastic leukaemia, clinically and morphologically in remission. She has been receiving pulse therapy of vincristine and dexamethasone. She did not have any infection. FBP sent for surveillance showed anaemia and leukopenia with neutropenia. Smear showed neutrophils containing cytoplasmic inclusions resembling Howell-Jolly bodies. *Discussion:* HJBLIs have also been reported in immunosuppressed patients, likely due to therapy-related factors as demonstrated in this case. Morphological identification in smear may help in recognising HJBLIs. However, confirmation of DNA-based origin by immunofluorescence microscopy was not performed due to unavailability.

MW-EP42: A Case of Life Saving Transfusion in a RhD-Negative Woman of Childbearing Potential: Management and Outcomes

Norhanis Nazar Shah[1], Wan Nurul Husna Wan Alkamar Shah[2], Eusni Rahayu Mohd Tohit[1], Veena Selvaratnam[3], Sheikh Ridzuan Shaikh Awadz[4], Prakash Selvanatham[2], Nurul Azwin Rasilin[2]
[1]Department of Pathology, Faculty of Medicine and Health Sciences, Universiti Putra Malaysia, Serdang, Selangor Darul Ehsan, [2]Department of Pathology, Blood Transfusion Unit, Hospital Ampang, Ministry of Health Malaysia, Selangor Darul Ehsan, [3]Department of Haematology, Hospital Ampang, Ministry of Health Malaysia, Selangor Darul Ehsan, [4]Department of Obstetrics & Gynaecology, Hospital Ampang, Ministry of Health Malaysia, Selangor Darul Ehsan.

Introduction: In critical, time-sensitive clinical scenarios, physicians may need to initiate transfusions before a full blood compatibility profile is established. The RhD-negative blood group is a rare occurrence among Malaysians, with Malays and Chinese exhibiting an extremely low prevalence of typically around 1% or even lower. On the other hand, the frequency is noticeably higher among Indians, reaching up to 8%, making them a distinct exception within the rich genetic diversity of our nation. Given the rarity of RhD-negative blood group in Malaysia, emergency transfusion strategies primarily rely on group O RhD- positive blood as the designated "Safe O", ensuring a swift solution for critical cases where precise blood type matching cannot be instantly secured. *Case Report:* Herein, we report the case of a 32-year-old Malay woman, primigravida, with an uncertain last menstrual period, who presented with a short history of severe lower abdominal pain and vaginal bleeding. These symptoms led to the diagnosis of a ruptured left ectopic pregnancy via abdominal ultrasonography and a positive urine pregnancy test. Despite fluid resuscitation, persistent signs of hemodynamic instability necessitated the transfusion of one pint of "Safe O" blood, with immediate planning for an emergency laparotomy and left salpingectomy operation. In a twist of events, crossmatching at the blood bank confirmed her RhD-negative blood group. *Discussion:* Our case report emphasises on the collaboration of a multidisciplinary team in managing this case according to standard guidelines, outlines monitoring strategies, offers recommendations for future pregnancies, and ultimately ensures the best possible care for the patient.

MW-EP43: Transformation of MDS to AML Associated with Trisomy 6: A Rare Cytogenetic Event

Nur Ilyia Syazwani Saidin[1], Fatma Basyira Jamallodin[1], Nurul Asyikin Nizam Akbar[1], Razan Hayati Zulkeflee[1], Nur Syafira Kamaruzzaman[2]
[1]Department of Haemotology, School of Medical Sciences, Universiti Sains Malaysia, Kota Bharu, Kelantan, [2]Department of Internal Medicine, School of Medical Sciences, Universiti Sains Malaysia, Kota Bharu, Kelantan

Introduction: Myelodysplastic syndrome (MDS) is a clonal haematopoietic stem cell disorder with variable risk of progression to acute myeloid leukaemia (AML). The transformation risk is influenced by cytogenetic abnormalities, molecular mutations, and bone marrow blast percentage. Case Report: We present a case of a 56-year-old woman diagnosed with multilineage MDS, initially presenting with pancytopenia and <5% blasts. Cytogenetic analysis was inconclusive due to failure to obtain metaphases. Over four months, she developed worsening pancytopenia: WBC 2.94 × 10^9/L, haemoglobin 8.3 g/dL, and platelets 35 × 10^9/L. Peripheral blood smear showed 24% circulating blasts. Bone marrow aspiration and immunophenotyping confirmed AML with myelomonocytic differentiation: 13.2% myeloblasts, 5.7% monoblasts, and 4.3% promonocytes. Trephine biopsy revealed extensive infiltration by immature cells positive for MPO and CD117, with partial CD34 and CD68 positivity. Cytogenetic studies showed a karyotype of 47,XX,+6. *Discussion:* This case demonstrates a rapid progression of MDS to AML with trisomy 6. While less common than other chromosomal abnormalities seen in MDS (e.g., del(5q), del(7q), +8, and del(20q)), trisomy 6 has been reported in both de novo AML, MDS and aplastic anaemia. The mechanisms by which trisomy 6 contribute to leukaemogenesis are largely unknown, but two processes have been suggested. The first contributes is a gene-dosage effect as a direct result of the trisomy, with the extra copies of genes leading to overexpression. The second is a mutation of a gene(s) on the trisomy chromosome. This case highlights the rare cytogenetic finding of trisomy 6 in a patient with MDS who experienced a rapid transformation to AML. Although trisomy 6 is an uncommon aberration in myeloid malignancies, its presence, especially in isolation, maybe a marker of clonal evolution and disease progression.

MW-EP44: A myth or a truth? Lineage switching infant leukaemia in *KMT2A* gene rearrangements; A case report.

Norhanis Nazar Shah[1], Eusni Rahayu Mohd Tohit[1], Fatin Adlia Arifin[2], Nurimatussolehah Sarijan[2], Koh-Su-Yee[2], Norazlina Azman[2], Suzana Zainol[2], Shenaz Banu Said Khan[2], Siti Shahrum Muhamed Said[2], Mimi Azura Aziz[2], Chin-Loi-Khim[2], Kogilavani Gunasagaran[3]

[1]Department of Pathology, Faculty of Medicine and Health Sciences, Universiti Putra Malaysia, Serdang, Selangor Darul Ehsan, [2]Department of Pathology, Hospital Tunku Azizah, Ministry of Health Malaysia, Kuala Lumpur. [3]Department of Paediatrics, Haemato-oncology Unit, Hospital Tunku Azizah, Ministry of Health Malaysia, Kuala Lumpur.

Introduction: Infantile leukaemia, diagnosed before the first birthday, is an exceptionally rare condition, comprising only 3.7% of childhood leukaemia cases. Studies indicate that acute lymphoblastic leukaemia (ALL) is more prevalent than acute myeloid leukaemia (AML) in infants. Its complex genetic instability, particularly the high frequency of *KMT2A* gene rearrangements (*KMT2A-r*) is associated with poorer outcomes in infant ALL compared to AML and shows strong correlation with lineage switch leukaemia. *Case Report:* We report a case of an eight-month-old girl who presented with fever, feeding difficulties, and lethargy. Physical examination revealed pallor and hepatosplenomegaly. Laboratory examination showed striking hyperleukocytosis, severe anaemia, and thrombocytopenia. Due to patient's condition was unstable for bone marrow aspiration (BMA), BALL was diagnosed via peripheral blood immunophenotyping (IPT). No myeloid or T markers were detected at diagnosis. Cytogenetics and molecular studies revealed t(11:19) (q23.3; p13.3) (KMT2A:MLLT1), linking KMT2A ex8+9 to MLLT1 ex2. Chemotherapy was administered based on the Interfant-99 protocol. By day 36 post-induction, a loss of B-lineage markers was observed along with gaining of myelomonocytic markers. Treatment followed with MARAM consolidation phase, OCTADD block reinduction, blinatumomab (BiTE therapy) and maintenance chemotherapy. Patient subsequently underwent an allogeneic bone marrow transplantation from an HLA identical sibling. *Discussion:* Lineage switch (LS) leukaemia, a unique and challenging phenomenon occurs when leukaemia cells change from one lineage to another, often during or immediately after induction chemotherapy. Most cases of LS begin as B-lineage ALL (BALL) but later transform into AML. This case underscores the complex nature of leukaemogenesis, highlighting diagnostic challenges posed by lineage switch, therapeutic strategies, and patient outcomes, while offering valuable insights for future advancements in this area.

MW-EP45: Rare Initial Presentation of Haemolytic Anaemia in Wilson Disease: A Case Report

Azizan Hanny Azahar[1], Wan Zuhairah W Embong[1], Yip Kam Hoo[2], Norfarizal Ashikin Abdullah[3]

[1]Haematology Unit, Department of Pathology, Hospital Sultanah Nur Zahirah; [2]Haematology Unit, Department of Internal Medicine, Hospital Sultanah Nur Zahirah; [3]Department of Ophthalmology, Hospital Sultanah Nur Zahirah

Introduction: Wilson disease is an autosomal recessive disorder of copper metabolism that causes severe damage to various organs. The clinical presentations are mainly hepatic and neurological, with haemolytic anaemia being uncommon. To our knowledge, this is the second reported case of initial presentation haemolytic anaemia in newly diagnosed Wilson disease in Malaysia. *Case presentation:* We report a case of a 15-year-old boy who presented with Coombs-negative haemolytic anaemia as the initial manifestation of Wilson disease. On examination, Kayser Fleischer ring was found. Haematological investigations revealed anaemia with initial haemoglobin level 10.1g/dL, which dropped to 6.5g/dL, red blood cell (RBC) count 2.97×10^{12}/L, MCV of 98.7 fL, MCH of 32.7 pg, MCHC of 33.1 g/dL and haematocrit of 29.3%. Reticulocyte fraction was elevated at 11.7%. There was leucocytosis (21.1×10^{9}/L) and thrombocytopenia (48×10^{9}/L). Elevated serum LDH of 439 U/L, and total bilirubin of 253 µmol/L (indirect 135 µmol/L, direct 118 µmol/L). Direct Coombs test was negative. Blood smear demonstrated polychromasia, a few spherocytic red cells and occasional nucleated RBC. These findings supported the diagnosis of haemolytic anaemia. Heinz bodies were also present with New Methylene Blue stain. Additionally, serum transaminases were persistently elevated and serum ceruloplasmin was low. There was an incidental finding of liver cirrhosis. However, his family declined treatment, and he subsequently died. *Discussion:* Haemolytic anaemia is a rare but recognised manifestation of Wilson disease. A high index of suspicion for Wilson disease is warranted in patients presenting with acute liver failure and Coombs-negative haemolytic anaemia.

MW-EP46: Acute Lymphoblastic Leukaemia with CD34 Negative Expression: A Case Series

Yulia Nadar Indrasari[1,2,3], Aryati [2,3], I Dewa Gede Ugrasena[2,4]

[1]Study Program of Doctoral, Faculty of Medicine Universitas Airlangga, Surabaya, Indonesia, [2] Dr. Soetomo General Academic Hospital, Surabaya, Indonesia, [3]Department of Clinical Pathology, Faculty of Medicine Universitas Airlangga, Surabaya, [4]Haemotology Oncology Division, Department of Child Health, Faculty of Medicine Universitas Airlangga, Surabaya

Introduction: The diagnosis of B-ALL requires confirmation through flow cytometry immunophenotyping to identify the expression of B-cell markers, such as CD19, CD22, and CD79a, and immature markers, including terminal deoxynucleotidyl transferase (TdT) and CD34. Only about 33-34% to 50% of B-ALL cases show negative CD34 expressions. T-acute lymphoblastic leukaemia/lymphoma (T-ALL/LBL) is a neoplasm of lymphoblasts committed to the T-cell lineage, involving bone marrow (BM) and blood (in T-ALL) or with primary involvement of the thymus or of nodal or extranodal sites (in T-LBL). However, the expression of CD34 on T lineage blasts is more inconsistent when compared to B lineage blasts and may be negative in 30-100% of T-ALL cases. This case series focuses on adult acute lymphoblastic leukaemia cases lacking CD34 expressions are presented. *Case Report:* The first patient, a 31-year-old man with clinical manifestations of bruising on both of his hands and legs and his back. CBC *Results* presented anaemia (Hb 8.5 g/dL), leucocytosis (WBC 42,180/µL), and thrombocytopenia (PLT 24,000/µL). Bone marrow morphology suggested ALL L1 (FAB criteria). The second patient is a 39-year-old man with clinical manifestations of nausea and body aches. Complete blood count *Results* presented anemia (Hb 10.6 g/dL), hyperleukocytosis (WBC 158,470/µL), and thrombocytopenia (PLT 20,000/µL). Bone marrow morphology suggested ALL L2 (FAB criteria). Immunophenotyping markers of the first patient revealed CD34 negative, cCD79a negative, CD19+, CD10+, cCD3-, CD5-, CD7-, cMPO- while the second patient revealed CD34

negative, cCD3+, CD5+, CD7+, cCD79a negative, CD19-, CD10-, cMPO-. Both patients underwent outpatient treatment in the haematology oncology medical division, internal medicine department. *Discussion:* The clinical implications of CD34 negativity are often associated with the prognosis of patients with B-ALL although the *Results* are still controversial. Some studies have reported an association between CD34 negative and a higher risk of residual disease (MRD) and relapse after chemotherapy, while other studies have suggested that the absence of CD34 may be associated with complications of lower fever during therapy.

MW-EP47: A Case of Acute Myeloid Leukaemia with t(6;9)(p23;q34)/*DEK::NUP214* in a Young Adult at Hospital Queen Elizabeth, Sabah

Nursyaza Fatinah MD Yusoff, Noorhana Sofia Ismail, Raja Muhamad Zul Hatta Raja Ismail
Department of Pathology, Hospital Queen Elizabeth, Kota Kinabalu, Sabah, Kementerian Kesihatan Malaysia

Introduction: Acute myeloid leukaemia (AML) with the t(6;9)(p23;q34) translocation, forming the *DEK::NUP214* fusion gene, is a rare and aggressive subtype, accounting for approximately 1–2% of AML cases. Clinically, it often presents with myelodysplasia, bone marrow basophilia, and dysplastic features such as ringed sideroblasts, posing significant challenges in management due to high relapse rates. Early intensive treatment, including hematopoietic stem cell transplantation (HSCT), is critical for improving outcomes. *Case report:* A 23-year-old male presented with fever, vomiting and weight loss. Initial full blood count showed bicytopaenia and raised white cell counts. Peripheral blood film showed 34% blasts, while bone marrow aspirate revealed hypercellular marrow with increased blasts (15-18%) with maturation and evidence of erythroid, granulocytic and megakaryocytic dysplasia. Cytogenetics identified the t(6;9)(p23;q34) translocation, forming the *DEK::NUP214* fusion gene. Mutations in *WT1*, *KRAS*, and *NRAS* were detected via next-generation sequencing (NGS). The patient received induction chemotherapy with DA3+7 followed by FLAG-IDA. Post-treatment bone marrow assessments showed no excess of blast, and he is planned for allogenic HSCT. *Discussion:* This case morphologically presented as AML with maturation and background of trilineage myelodysplasia, which may give early indication presence of t(6;9)(p23;q34); *DEK::NUP214*. This also demonstrates how early diagnosis, and aggressive chemotherapy can lead to a favourable initial response in a high-risk AML subtype. Conclusion: The poor prognosis associated with *DEK::NUP214* highlights the need for early diagnosis, close monitoring, and early consideration of HSCT during the first remission to improve long-term outcomes.

MW-EP48: Correlation of Immature Platelet Fraction with Platelet Count and Age: A Retrospective Study

Fatmawati Kamal[1], Wan Asmuni Wan Mohd Saman[1] and Halimatun Radziah Othman[2]
[1]Department of Pathology, Faculty of Medicine, Universiti Teknologi MARA, Sungai Buloh Campus; [2]Clinical Diagnostic Laboratories, Hospital Al-Sultan Abdullah, Universiti Teknologi MARA, Bandar Puncak Alam

Introduction: Immature platelet fraction (IPF) is available as an extended parameter in the reticulocyte/optical platelet channel. Immature platelets contain more ribonucleic acid (RNA) compared to mature platelets and can be identified using flow cytometry techniques. It indirectly provides information on thrombopoiesis, as high IPF indicates increased platelet production by the bone marrow. Its utility has been explored in thrombocytopaenia associated with immune thrombocytopenia, cancer and viral infections. However, fewer publications were found on thrombocytosis or normal platelet counts. This study examined the relationship between IPF and platelet counts, as well as age. *Materials and Methods:* Data were extracted from the Mindray BC6200 database between June 2024 and December 2024. IPF% were analysed based on low, normal and high platelet counts. *Results:* A total of 111 patients, aged between 1 and 84 years (median: 49 years, IQR: 45), were included. Thrombocytopenia was found in 27% of the patients (mean: 72.6 × 10^9/L, ± SD: 41 × 10^9/L). The normal range for IPF% is between 0.7% and 7.6%. IPF% was significantly higher in cases of thrombocytopenic patients (median = 8.55, min = 0.3, max = 46.1) than in patients with normal platelet counts (median = 5.85, min = 1.2, max = 6.4) or thrombocytosis (median = 4.35, min = 2.2, max = 10.7), $p<0.001$. *Discussion:* There was a strong, negative correlation between IPF and platelet counts, rho = –.45, $p < .001$. This study found almost no correlation between IPF and age, with a correlation coefficient of rho=.044, p=0.65. In conclusion, this study showed that IPF is significantly increased in thrombocytopenia, due to platelet recovery, and relatively unaffected by age.

MW-EP49: Enhancing National Thalassemia Surveillance through Standardised Diagnostic Coding and Dashboard Visualization: A Public-Private Collaboration Model in Malaysia

Ooi Theng Choon[1], Marius Micheal[2], Nur Hidayah Zakaria[2], Darwisha Darus[2], Mohamad Adam Narawi[2], Zaini Mat Nor[2], Caroline Jennifer[2], Saira Bahnu Mohamed Yousoof[1,3], Sayyidi Hamzi Abdul Raub[1,2,3], Zubaidah Zakaria[2,3], Mohd Hareeff Muhammad[1,2,3]
[1]Strategic Planning and Innovation, Premier Integrated Labs Sdn Bhd, Brickfields, Kuala Lumpur, Malaysia; [2]Haemotology, Reference Core Laboratory (RCL); [3]Cytogenetics and Molecular Diagnostics Laboratory, Premier Integrated Labs Sdn Bhd, Pantai Hospital Kuala Lumpur, Bangsar, Kuala Lumpur, Malaysia.

Introduction: In January 2024, the Ministry of Health (MOH) Malaysia introduced an updated diagnostic coding system for thalassemia, encouraging its adoption by private laboratories as outlined in circular HS PAT/761/04 (107). Previously, the lack of standardised reporting, particularly in private labs, hindered effective disease surveillance, care coordination, and preventive measures, especially at the primary care level. This study aimed to implement the standardised coding system within a private laboratory setting and to develop an interactive dashboard to support national thalassemia control efforts. *Materials and Methods*: Beginning in June 2024, pathologists at Premier Integrated Labs assigned thalassemia diagnostic codes during the validation of haemoglobin analysis *Results*. Structured diagnostic data were extracted monthly from the Laboratory Information System and used to develop an interactive Thalassemia Registry Dashboard using Power BI. The dashboard supports advanced analytical functions, including drill-down capabilities to explore regional and population-specific patterns by sociodemographic variables such as gender, ethnicity, and age. It also features drill-through functions for detailed evaluation of individual cases. *Results*: Between 1st June 2024 and 31st May 2025, 9,195 haemoglobin analysis samples were received from 1,152 primary care providers. Of these, 4,908 samples

(53.4%) exhibited abnormal findings. These were categorized as follows: α-thalassemia (168 cases; 3.4%), β-thalassemia (787; 16.0%), haemoglobinopathies (486; 9.9%), Hb variants (1,696; 34.6%), and others (1,771; 36.1%). The most commonly assigned diagnostic codes included E1 (iron deficiency anaemia), D16 (α-thalassemia cannot be excluded), and E2 (suspected iron deficiency anaemia). *Discussion:* This initiative illustrates that integrating standardised diagnostic coding in private laboratories can generate high-quality, structured data for national registries and support data-driven public health strategies. Embedding diagnostic codes within laboratory reports also improves communication with primary care providers, facilitating accurate interpretation, carrier identification, and timely referrals for genetic counselling and family screening. This public-private partnership model is scalable and can significantly strengthen thalassemia control efforts nationwide.

MW-EP50: Acute Myeloid Leukaemia with *NPM1* Mutation in Malaysian Patients: Insights from the Institute for Medical Research's Next-Generation Sequencing Experience

Suguna Somasundram, Zahidah Abu Seman, Nadirah Zainal Abidin, Julia Abdullah, Fifi Shawani, Siti Nur Farah Adibah Rajami, Norafiza Mohd Yasin, Azian Naila Md Nor, Ermi Neiza Mohd Sahid, Yuslina Mat Yusoff,
Haematology Unit, Cancer Research Centre, Institute for Medical Research, Shah Alam, Selangor

Introduction: Acute Myeloid Leukaemia (AML) with *NPM1* mutation amounts to one-third of newly diagnosed AML. This study analysed the incidence of *NPM1* mutations from the retrospective data of AML mutation study from October 2023 until April 2025 utilising the Next Gene Sequencing (NGS) method. The molecular types of *NPM1* mutations and co-existence with FLT-ITD mutation were additionally analysed. *Materials and Methods:* DNA was extracted from peripheral blood or bone marrow samples using the QIAamp DNA Blood Mini Kit. Library preparation was then carried out using the AmpliSeq for Illumina Myeloid Panel, a targeted panel designed to detect 40 DNA genes associated with myeloid cancers. Sequencing was performed using the MiniSeq High Output Kit. Data analysis included secondary analysis with DRAGEN DNA Amplicon software and tertiary analysis using QIAGEN Clinical Insight Interpret. *Results:* Out of 651 AML samples analysed, 172 (26.4%) were found to have *NPM1* mutations. The distribution of *NPM1* types is type A (77.9%), type B (5.2%) and type D (6.4%) and the other types (10.5%). The non-type A, B and D are considered rare. Among the 172 cases with *NPM1* mutations, 69 (40%) also harboured concurrent *FLT3-ITD* mutations. *Discussion:* There is disagreement in the literature about whether specific types of *NPM1* mutations represent a poor prognostic factor. According to the 2022 ELN risk classification, mutated *NPM1* without an accompanying *FLT3-ITD* mutation is categorized as favourable and demonstrates a positive response to induction therapy. Detection of this mutation is important for diagnosis, prognosis, treatment stratification and monitoring.

MW-EP51: Silent Infiltration: Invasive Lobular Carcinoma of the Breast Unveiled by Trephine Biopsy in a Lady with Unexplained Cytopenia

Nur Khalili Che Ali[1] Wan Amal Hayati Wan Hassan[1], Wan Zuhairah W Embong[1], Nor Ainiza Mansor[1], Zakiah Nurasyikin Mohamed Halim[1], Azly Sumanty Ab Ghani[1], Yip Kam Hoo[2]
[1]Haematology Unit, Department of Pathology, Hospital Sultanah Nur Zahirah. [2]Haematology Unit, Department of Internal Medicine, Hospital Sultanah Nur Zahirah.

Introduction: Breast cancer is the most common malignancy worldwide. ILC is the second most common type of breast cancer and often presents insidiously, lacking a distinct breast mass. Bone marrow infiltration may manifest as unexplained cytopenia, such as anaemia and thrombocytopenia, accompanied by a leucoerythroblastic blood picture, which may mimic primary haematological disorders. We report a diagnostically challenging case in which a trephine biopsy uncovered the diagnosis of ILC. *Case Report:* A 48 years-old lady with one-month history of lethargy, lower back pain, abdominal pain, loss of appetite and weight loss. No palpable breast lump, lymphadenopathy or hepatosplenomegaly. Peripheral blood film revealed anaemia and thrombocytopenia with a leucoerythroblastic picture and occasional blast cells. Bone marrow aspiration was markedly haemodiluted. Trephine biopsy revealed the presence of non-haematopoietic cells. Histologically these cells were moderate to large in size, with open chromatin, prominent nucleoli and a moderate amount of cytoplasm. The cells was arranged in sheets, and some forming glandular patterns. Immunohistochemistry demonstrated strong positivity for pancytokeratin (CK AE1/AE3), suggestive of metastatic carcinoma. The immunophenotype (CK7 positive, CK20 negative) supported a primary origin from the breast, lung, thyroid, cervix or pancreas. Associated marrow fibrosis and osteosclerosis were also observed. A subsequently CT scan of thorax, abdomen and pelvis revealed diffuse lytic bone lesions and hepatosplenomegaly. Colonoscopy revealed metastatic carcinoma, likely of breast origin. Breast ultrasound and mammography detected a suspicious lesion in the right breast lesion (BIRADS IV). A trucut biopsy of the lesion confirmed a diagnosis of ILC. Unfortunately, the patient succumbed to the disease before oncological treatment could be initiated. *Discussion:* This case highlights the diagnostic challenge of metastatic ILC, particularly when presenting without a palpable breast lump. It underscores the importance of considering metastatic carcinoma in patients with unexplained cytopaenias and leucoerythroblastic blood pictures. Bone marrow aspiration, including trephine biopsy and immunohistochemistry, plays a crucial role in prompt diagnosis and management.

MW-EP52: Unraveling the Immunophenotypic Complexity of MPAL: A Differential Diagnosis Perspective

Shalini Ponnusamy
Department of Pathology, Hospital Raja Permaisuri Bainun, Ipoh,, Perak, Malaysia.

Introduction: Acute myeloid leukaemia (AML) with t(8;21)(q22;q22) *RUNX1::RUNX1T1* is a well-established AML subtype with characteristic genetic and clinical features. Aberrant expression of B-lineage markers is a recognised phenomenon in this subtype and may complicate diagnosis. Early and accurate diagnosis is essential for appropriate treatment and prognosis. *Case Report:* An 11-year-old boy presented with right-sided otalgia and reduced hearing associated with fever and weight loss over three weeks. Initially treated for otitis media, his symptoms persisted alongside rising white blood cell counts. Peripheral blood smear revealed

81% blast cells, with no Auer rods seen. Immunophenotyping performed on bone marrow showed blasts positive for myeloid markers with co-expression of B-lineage markers. Cytochemical staining was MPO-positive, PAS-negative. Immunophenotyping demonstrated expression of precursor markers (CD34, TdT), myeloid markers (CD13, CD33, CD117, cyMPO) and B-lineage markers (CD19, cyCD79a, cyCD22). Molecular and cytogenetic studies identified the t(8;21)(q22;q22) *RUNX1::RUNX1T1* fusion. The patient was treated with AML UK 17 protocol chemotherapy with favourable response and subsequent improvement of counts. *Discussion:* This case highlights the diagnostic importance of integrating morphology, immunophenotyping, and molecular genetics in AML with aberrant B-lineage marker expression to prevent misdiagnosis.

MW-EP53: Parvovirus B19-Associated Pure Red Cell Aplasia Post-Renal Transplant: A Diagnostic and Pathological Challenge

Fahmaa Azizi, Masitah Hamid, Nur Hidayah Muhamad Yasin
Haemotology Unit, Dept of Pathology Hospital Sultanah Bahiyah Alor Setar, Kedah, Malaysia

Introduction: Parvovirus B19 (PB19V)–associated pure red cell aplasia (PRCA) is a rare but clinically significant complication in renal transplant recipients. PB19V, the only known human pathogen in the Parvoviridae family, exhibits tropism for erythroid progenitor cells (EPCs) in the bone marrow. In immunocompromised patients, PB19V can cause persistent infection, leading to erythroid suppression and PRCA. *Case Report:* We report a 43-year-old female with end-stage renal failure secondary to granulomatous nephritis, who underwent renal transplantation 11 months earlier and was on immunosuppressive therapy. She presented with progressive anaemia requiring multiple transfusions. Blood film showed severe normocytic normochromic anaemia with marked reticulocytopenia, while white blood cell and platelet counts remained normal. Bone marrow aspiration revealed marked erythroid hypoplasia with scattered giant proerythroblasts, some with prominent inclusion-like nucleoli. Trephine biopsy demonstrated marked erythroid hypoplasia with predominance of proerythroblast, including giant forms while granulopoiesis and megakaryopoiesis were preserved. PB19V immunostaining showed viral nuclear inclusions and PCR confirmed viremia. The patient responded well to intravenous immunoglobulin (IVIG) therapy. *Discussion*: PB19V-induced PRCA typically presents with isolated anaemia and profound reticulocytopenia. Erythroid aplasia with giant proerythroblasts containing viral inclusions is a hallmark of PB19V infection. Diagnosis relies on a combination of marrow morphology, immunohistochemistry, and molecular testing. Treatment involves IVIG and adjustment of immunosuppression. Persistent anaemia in renal transplant recipients on immunosuppressive therapy necessitates thorough investigation. In the presence of isolated anaemia with reticulocytopenia, PB19V infection should be strongly suspected. Early recognition is crucial to avoid unnecessary transfusions and restore erythropoiesis in transplant patients.

MW-EP54: When Leukaemia Wears a Lymphoma Mask: A Morphological Puzzle in Haematologic Malignancy

Nabilah Rameli[1], Zakiah Nurasyikin Mohamed Halim[2], Nor Ainiza Mansor[2]
[1]Department of Pathology, Hospital Dungun, Dungun, Terengganu, [2]Haematology Unit, Department of Pathology, Hospital Sultanah Nur Zahirah, Kuala Terengganu, Terengganu

Introduction: Differentiating blasts of acute leukaemia from malignant lymphoma cells based on morphology alone can be diagnostically challenging. While leukaemic blasts typically exhibit high nuclear-to-cytoplasmic ratios and fine chromatin, lymphoma cells demonstrate broader morphological variability. Accurate interpretation is critical, particularly in settings with limited access to immunophenotyping and cytogenetics. *Case Report:* A 30-year-old Malay male, smoker, and courier by occupation presented with symptoms of anaemia and multiple lymphadenopathies, without hepatosplenomegaly. Blood tests revealed pancytopenia: WBC 3.95 × 10^3/μL, Hb 3 g/dL, and platelets 40 × 10^3/μL. Liver enzymes were mostly normal aside from mildly elevated ALT. Infectious, autoimmune, and nutritional causes were excluded. Peripheral blood smear revealed a leukoerythroblastic picture with presence of abnormal cells, characterised by small to moderately sized forms with cytoplasmic blebbing, mildly clumped chromatin, and prominent nucleoli. LDH rose from 293 to 406 U/L. Bone marrow aspirate yielded no fragments but revealed small to moderate vacuolated cells with scant bluish cytoplasm; some showed cytoplasmic blebbing. Immunophenotyping demonstrated 45% B-lymphoblasts, with 27% of the blast population positive for nuclear TdT and cyCD79α but negative for CD34, CD20, kappa, and lambda, leading to a diagnosis of precursor B-cell acute lymphoblastic leukaemia (B-ALL). *Discussion:* This case highlights the diagnostic ambiguity of blast cells with atypical morphology mimicking lymphoma. Despite a suggestive morphological picture, definitive lineage determination required immunophenotyping. In resource-limited settings, reliance on morphology alone poses risks of misclassification. This underscores the indispensable role of ancillary testing in accurately diagnosing haematologic malignancies.

MW-EP55: Compound Heterozygous Haemoglobin Hope With β⁰-Thalassemia: Diagnostic Challenges in Variant Co-Inheritance

Nur Mastura Abdul Wahab[1], Nor Ayuni Khuzaimah[1], Hafizah Hashim[2]
[1]Haematology Unit, Pathology Department, Hospital Pulau Pinang, Ministry of Health, Penang, Malaysia, [2]Haematology Unit, Pathology Department, Hospital Sultanah Bahiyah, Ministry of Health, Kedah, Malaysia

Introduction: Haemoglobinopathies, including structural variants and thalassemia, present diagnostic challenges due to overlapping features. Haemoglobin Hope (Hb Hope), a rare β- globin variant (codon 136, GGT→GAT), is usually asymptomatic in heterozygotes but may complicate diagnosis when co-inherited with β-thalassemia. *Case Report:* We report a 49-year-old asymptomatic Chinese female with incidental mild anaemia (Hb 9.9 g/dL, MCH 22.9 pg, MCV 75.7). Capillary electrophoresis (CE) showed a dominant Hb Hope peak (93.7%), elevated HbA2 (5.3%), and absent Hb A, initially suggesting homozygosity. HPLC confirmed an abnormal peak (89.6%, retention time 1.4 min) with elevated HbA2 (6.2%). Molecular analysis revealed compound heterozygosity for Hb Hope and β - thalassemia (codon 41/42, –TTCT). However, no molecular test for alpha-thalassemia was performed. *Discussion:* This case underscores the diagnostic complexity of haemoglobinopathies, where β- thalassemia suppression of Hb A can mimics homozygosity. Elevated HbA2 and absent HbA were key to suspecting concomitant β-thalassemia. Despite compound heterozygosity,

the patient had only mild anemia, highlighting variable expressivity. Co-inheritance of α-thalassemia, which may ameliorate the phenotype, should also be excluded. *Discussion:* The findings emphasise the importance of integrating full blood count indices, haemoglobin typing (CE/HPLC), and molecular analysis for accurate diagnosis. Genotype- phenotype correlation facilitates the prediction of clinical severity, particularly in cases of compound heterozygosity. Precise characterisation is vital for genetic counselling, family screening, and distinguishing benign variants, especially in ethnically diverse populations.

MW-EP56: A Challenging Case of Mixed Phenotypic Acute Leukaemia in a Young Adult: Diagnostic and Therapeutic Dilemmas

Nurul Anis Che Anuar, Shafini Mohamed Yusoff, Nurul Asyikin Nizam Akbar
Department of Haematology, School of Medical Sciences, Health Campus Universiti Sains Malaysia, Kubang Kerian, Kelantan, Hospital Pakar Universiti Sains Malaysia, Kubang Kerian, Kelantan

Introduction: Acute leukaemias of mixed or ambiguous lineage (ALAL) rarely occurred, represents <4% of all acute leukaemia cases. It composed of ≥20% abnormal progenitors that do not show differentiation along a single lineage. Managing patients with mixed phenotypic acute leukaemia (MPAL) is challenging in view of mixed phenotypic expressions of the blasts. *Case report:* A 25-year-old, Malay lady presented with progressively worsening dysphagia for 3 months associated with odynophagia, painless neck swelling, weight loss with no fever and night sweats. Initially she was treated for left tonsillar enlargement with multiple lymphadenopathies and hepatosplenomegaly. Direct laryngoscope and tonsillectomy showed left tonsil infiltration by primary tumour. Full blood picture (FBP) showed leukocytosis with normal haemoglobin (Hb) and platelet count with presence of 85% blasts. Bone marrow & trephine biopsy (BMAT) showed 90.8% blasts exhibiting two distinct populations supported by flowcytometry and immunohistochemical (IHC) staining. Cytogenetic study showed complex karyotype patterns. This patient underwent HyperCVAD followed by maintenance cycle (POMP) while waiting for allogenic-stem cell transplant. However, patient developed Relapsed MPAL while on maintenance cycle. *Discussion:* The rarity of MPAL with the capability to switch lineages under pressure (treatment) defines the challenges in treating MPAL. The debate on the most appropriate induction regimen remains unresolved over the controversy whether initial therapy should be based on immunophenotype, cytogenetic or molecular biology. MPAL has poor prognosis and worse outcomes than standard acute myeloid leukaemia (AML) or acute lymphoblastic leukaemia (ALL).

MW-EP57: She Was Group O, Not A: A Transfusion Catastrophe from A Single Sample

Nur Ilyia Syazwani Saidin[1], Filzah Amalina Mohd Nordin[1], Noor Haslina Mohd Noor[1], Azlan Husin[2], Mohd Nazri Hassan[1]
[1]Department of Haemotology, School of Medical Sciences, Universiti Sains Malaysia, Kota Bharu, Kelantan, [2]Department of Internal Medicine, School of Medical Sciences, Universiti Sains Malaysia, Kota Bharu, Kelantan

Introduction: Transfusion safety relies heavily on accurate patient identification and pre-transfusion testing. Errors in sample labelling or collection can result in severe adverse events, particularly in patients without existing blood group records. *Case Report:* We report a transfusion error involving a 26-year-old female with osteoid osteoma of the L5 vertebra, electively admitted for surgery. A Group, Screen, and Hold (GSH) sample on admission identified her as blood group A positive with a negative antibody screen; however, no previous blood group records were available. Intraoperatively, she experienced a blood loss of 1.2 liters and was transfused with two crossmatch-compatible group A positive red cell units. Post-transfusion, the patient developed haematuria and hypotension, with a haemoglobin drop from 10 to 6 g/dL. Peripheral blood film revealed schistocytes, and she developed disseminated intravascular coagulation (DIC) accompanied by acute kidney and liver injury. Biochemical findings were consistent with ongoing haemolysis, demonstrated by significantly raised LDH and elevated indirect bilirubin levels. A repeat GSH sample showed haemolysis, and crossmatch with group A blood was incompatible. Direct antiglobulin test was positive (IgG 2+), and anti-A antibodies were detected via elution and testing with A1 cells. Repeat blood grouping demonstrated a mixed field reaction with anti-A antisera, confirming the patient's true blood group as O positive. Crossmatch with group O positive blood was compatible. *Discussion:* This case illustrates an acute haemolytic transfusion reaction complicated by DIC and acute kidney injury following a mismatched transfusion. The root cause was the mislabelling of the initial GSH sample. This incident highlights the critical importance of strict adherence to sample collection and labelling protocols. Reinforcement of bedside patient identification practices is vital. Implementing a routine second sample policy for first-time blood groupings may prevent similar errors in the future.

MW-EP58: Bisphenol F Induced Red Blood Cells Morphological Changes in Rats After Subacute and Subchronic Exposure

Izatus Shima Taib[1,2], Zariyantey Abd Hamid[1,2], Nur Natrahanies Suhaimi[2] and Muhammad Afiq Azhar[2]
[1]Centre for Diagnostics, Therapeutics and Investigative Studies, Faculty of Health Sciences, Universiti Kebangsaan Malaysia, Jalan Raja Muda Abdul Aziz, 50300 Kuala Lumpr, Malaysia, [2]Biomedical Sciences Program, Faculty of Health Sciences, Universiti Kebangsaan Malaysia, Jalan Raja Muda Abdul Aziz, 50300 Kuala Lumpr, Malaysia

Introduction: Bisphenol F (BPF) is used in the manufacturing industry as an alternative to bisphenol A, which has been banned due to its toxicity. Mass substitution in the manufacturing industry has led to under-reporting of BPF toxicity studies including the effects of BPF on red blood cells (RBCs). Therefore, this study was conducted to evaluate the effects of BPF on RBCs after subacute and subchronic exposure. *Materials and Methods:* For each subacute and subchronic studies, male Sprague Dawley Rats were divided into four groups (n=7/group): control (received 1 mg/kg corn oil); BPF1 (received 1 mg/kg BPF); BPF5 (received 5 mg/kg BPF); and BPF10 (received 10 mg/kg BPF). BPF were administered via force feed oral for 28 days (subacute) and 60 days (subchronic). At the end of studies, rats were sacrificed and RBCs were isolated for oxidative stress status, fragility test and morphological evaluations and were assessed using ANOVA test. *Results:* No significant changes were observed in all oxidative stress parameters superoxide dismutase, glutathione and malondialdehyde as well as the membrane fragility test between all groups in both subacute and subchronic exposures. However, the morphological observations found target cells in BPF5 (p=0.04) and BPF10 (p=0.032) groups with the percentage is significantly higher as compared to the control and BPF1 groups. *Discussion:* BPF

exposure might directly affects the lipid and protein composition in RBCs' membrane thus induced target cells formation after subacute and subchronic exposures. Future studies can be done in evaluating molecular aspects of RBC's membrane in BPF exposure.

MW-EP59: Haemoglobin Rothschild: A Rare Haemoglobin Variant Detected in Asymptomatic Adolescent During Routine Screening

Sharifah Sarah Syed Mohamad Alwi[1], Faidatul Syazlin Abdul Hamid[1], Syahzuwan Hassan[1], Ezzanie Suffya Zulkifle[1], Nur Aisyah Aziz[1], Sharifah Suhana Syed Abd Rahman[1], Fatin Akmal Baharom[2], Ermi Neiza Mohd Sahid[1], Azian Naila Md Nor[1], Suguna Somasundram[1], Ezalia Esa[3], Norafiza Mohd Yasin[1], Yuslina Mat Yusoff[1]

[1]Haematology Unit, Cancer Research Centre (CaRC), Institute for Medical Research (IMR), National Institute for Health (NIH) Setia Alam, Selangor, Malaysia, [2]Unit Haematology, Department of Pathology, Hospital Melaka, [3]Virology Unit, Infectious Disease Research Centre (IDRC), Institute for Medical Research (IMR), National Institute for Health (NIH) Setia Alam, Selangor, Malaysia.

Introduction: Haemoglobin (Hb) Rothschild is a rare haemoglobin variant caused by a mutation in the beta-globin gene, replacing tryptophan with arginine at position 37 of the beta-globin chain. This haemoglobinopathy is associated with mild to moderate haemolytic anaemia and low oxygen affinity. *Case Report:* 16year old boy with no known medical illness enrolled in Malaysia thalassaemia screening program for Form 4 students. He had no history of anaemia-related admissions and was generally well. On examination he was not in respiratory distress and not cyanosed. Oxygen saturation (SpO_2) under room air was 89% and no hepatosplenomegaly. Full Blood Count (FBC) showed RBC count of 6.23 x 10^6/uL, Hb of 15.2g/dL, MCV of 76.4fL, MCH of 24.4pg, RDW 14.2%. Hb analysis using High-performance liquid chromatography (HPLC) revealed HbA of 51.4% with HbA2 (2.9%) and presence of abnormal peak at zone 6 of 41.9%, while Hb analysis capillary electrophoresis (CE) revealed HbA_2 (2.5%) and presence of abnormal peak seen at RT 4.06 min of 41.9%. Direct beta sequencing revealed heterozygous state of Codon 37 [TGG>CGG] Hb Rothschild mutation. *Discussion:* The presence of an abnormal peak resembling Hb D has also been reported in other cases of this variant. Some reported cases seen with low SpO_2 and low partial pressure of oxygen (PaO_2) by blood gas analysis with or without presence of cyanosis and fatigue where the clinical correlations were unknown. This case report illustrates the subtle presentation of Hb Rothschild and highlights the importance of molecular diagnostics in accurately diagnose rare haemoglobin variants, especially in the context of population screening programs.

MW-EP60: Challenges in Meeting The 45-Minute Turnaround Time for Urgent Full Blood Count: A Retrospective Review of Emergency Department Samples

Halimatun Radziah Othman[1], Zetty Zuraini Shamsubahar[1], Syarifah Syariena Muhamad Jaiz[1], Madyhah Abdul Monir[1,2]

[1]Haematology and Transfusion Medicine Unit, Department of Clinical Diagnostic Laboratories, Hospital Al-Sultan Abdullah UiTM, Bandar Puncak Alam, Selangor Malaysia; [2]Faculty of Medicine Universiti Teknologi MARA Sungai Buloh, Selangor Malaysia

Introduction: The Ministry of Health Malaysia (MOH) has established a 45-minute laboratory turnaround time (LTAT) for urgent Full Blood Count (FBC) *Results* as a key performance indicator (KPI), targeting >90% compliance. Our haematology laboratory adopted this KPI as part of quality monitoring; however, achieving consistent compliance remains a challenge. This study evaluates LTAT compliance for Emergency Department (ED) FBC samples and identifies contributing factors to delays. *Materials and Methods:* A retrospective analysis was conducted on ED FBC samples processed by the Clinical Diagnostic Laboratories Department, Hospital Al-Sultan Abdullah (HASA) UiTM, from January to December 2024. LTAT was defined as the duration from sample receipt to manual result validation. Delayed cases were grouped into Category A (46–60 minutes) and Category B (>60 minutes) and analysed to determine the causes of delay. *Results:* ED samples represented over one-third of the total FBCs received in 2024. Of 1,453 delayed cases, 52% were in Category A and 48% in Category B. Category A delays were mainly due to manual result review, whereas Category B delays were associated with instrument-flagged abnormalities requiring microscopic examination. These flagging included platelet clumps, >3% immature granulocytes, marked leucocytes or platelet abnormalities and cold agglutinin cases requiring sample incubation. Despite these challenges, monthly LTAT compliance exceeded 90% throughout 2024, except in September (88.8%). *Discussion:* Key contributors to delays included reflex microscopic examination, manual staining and manual result verification. Optimising workflow through autoverification and strengthening microscopic competency among laboratory technologists may enhance compliance. These findings highlight the operational challenges of timely reporting in a manual-dependent environment.

MW-EP61: When Dengue Isn't Just Dengue: A Case of Hidden Acute Promyelocytic Leukaemia (APML)

Nuur Qamarina Mohd Bakri, Chitranjini Nadaraja, Nur Atikah Md Nor

Department Of Pathology, Hospital Kuala Lumpur

Introduction: Severe dengue characterised by plasma leakage, coagulopathy and organ dysfunction, is a major public health concern in endemic regions. APML is a distinct subtype of acute myeloid leukaemia associated with high risk disseminated intravascular coagulation (DIC) due to abnormal promyelocytes releasing procoagulant substances. The coexistence of severe dengue and APML is exceedingly rare but present a critical diagnostic dilemma. Here, we report a case of severe dengue in a newly diagnosed APML. *Case Report:* A 51-year-old woman came in with 5 days of fever, dizziness, and heavy menstrual bleeding. When examined, she had a fever, hypotensive and tachycardic. Laboratory investigations revealed pancytopenia. Dengue serology was positive. During convalescent phase, patient had refractory menorrhagia. A peripheral blood smear showed 91% abnormal promyelocytes with PML-RARA translocation. The patient was started on supportive care and all-trans retinoic acid (ATRA) was initiated. Unfortunately, patient succumbed to death after 1st cycle of chemotherapy. *Discussion:* Pancytopenia is not the most common haematological finding in dengue, but it can occur, especially in moderate to severe cases. Studies show pancy211topenia occurs in 5–10% of hospitalised dengue patients. In APML and dengue, the promyelocytic leukaemia protein (PML) plays a significant role as a restriction factor against viral infection. PML is an intrinsic antiviral defence that restricts viral replication, but in APML,

its loss may blunt the host's defence against dengue. Research suggests that PML restricts dengue virus DENV replication and disrupts PML-nuclear bodies PML-NBs during infection. *Conclusion:* APML should be considered in dengue patients presenting with prolonged cytopenias or unexplained haemorrhagic manifestations, particularly in the convalescent phase. Timely diagnosis and initiation of ATRA-based therapy are essential to prevent potentially fatal outcomes.

MW-EP62: Unmasking Lead Toxicity: The Power of a Peripheral Blood Smear

Nazurah Khalid, Norsafina Zainal, Siti Nur Azwa Md Shahid
Haematology Unit, Department of Pathology, Hospital Sultan Idris Shah, Serdang.

Introduction: Basophilic stippling arises from incomplete or failed degradation of ribosomal RNA, leading to the aggregation of ribosomes or their remnants within mature circulating erythrocytes. In cases of lead toxicity, the heavy metal inhibits pyrimidine-5'-nucleotidase, an enzyme essential for ribosomal RNA breakdown. As such, basophilic stippling is considered a pathognomonic finding of lead poisoning, particularly when accompanied by anaemia. *Case Report:* A 37-year-old man presented with unexplained fever, chest pain, bilateral lower limb swelling, multiple lymphadenopathies and transaminitis. Despite undergoing several investigations and receiving empirical treatments, his condition showed minimal improvement. A peripheral blood smear revealed a substantial amount of basophilic stippling with moderate normochromic normocytic anaemia. These findings prompted further testing for lead levels in blood and urine, both of which were significantly elevated. The patient was subsequently treated with intravenous ethylenediaminetetraacetic acid (EDTA) for five days. Following chelation therapy, his symptoms gradually improved, and the fever resolved. *Discussion:* Basophilic stippling is a common manifestation of various haematological disorders; however, it can also serve as an indicator of heavy metal toxicity. In this case, the presence of basophilic stippling alongside unexplained anaemia raised clinical suspicion for lead poisoning, later confirmed by elevated blood and urine lead levels. Despite the patient's non-specific symptoms and inconclusive initial workup, a simple peripheral blood smear provided a vital diagnostic clue. Early recognition enabled prompt treatment, leading to clinical improvement. This case highlights the diagnostic value of blood smears in systemic illness and underscores the importance of considering heavy metal toxicity in persistent, unexplained presentations.

MW-EP63: Epigenetic Landscape of Acute Myeloid Leukaemia Cell Lines

Norfarhana Mazlan[1,3], Mohd Khairul Nizam Mohd Khalid[2], Zahidah Abu Seman[1], Saiful Effendi Syafruddin[3], Mohamad Aimanuddin Mohtar[3], Muhammad Redha Abdullah Zawawi[3], Noor Atiqah Fakharuzi[1], Shaik Ahmad Kamal Shaik M. Fakiruddin[1], Ezalia Esa[1], Azian Naila Md Nor[1], Ermi Neiza Mohd Sahid[1], Yuslina Mat Yusoff[1]*
[1]Haematology Unit, Cancer Research Centre, Institute for Medical Research, National Institutes of Health, Section U13 Setia alam, Shah Alam, Selangor, [2]Inborn Error of Metabolism and Genetic Unit, Nutrition, Metabolism and Cardiovascular Research Centre, Institute for Medical Research, National Institutes of Health, Section U13 Setia alam, 40170 Shah Alam, Selangor, [3]UKM Medical Molecular Biology Institute, Jalan Yaacob Latif, Bandar Tun Razak, Cheras, Kuala Lumpur.

Introduction: Aberrant DNA methylation plays a crucial role in gene regulation and leukaemogenesis, particularly in acute myeloid leukaemia (AML). Understanding this patterns in specific genes can provide insights into disease mechanisms and uncover potential therapeutic targets. This study investigates genome-wide DNA methylation patterns in AML using whole genome bisulfite sequencing (WGBS) in two AML cell lines (CG-SH and THP- 1) with distinct cytogenetic profiles. Materials and methods: WGBS was performed on genomic DNA extracted from CG-SH and THP-1 cell lines to generate single-base resolution methylomes. Comparative analyses were conducted to assess differences in methylation signatures between the cell lines to identify patterns associated with cytogenetic abnormalities. *Results:* A striking pattern of complete hypomethylation was observed in several tumour suppressor genes (i.e. CDKN2A and CDKN2B) in THP-1 cells with t(9;11) translocation. In contrast, CG-SH cells which lack chromosomal translocation, displayed heterogeneous methylation patterns across the same loci. Additionally, differential methylation patterns were observed in other AML-related genes such as *CEBPA* and *MLLT3*, between the two cell lines. *Discussion:* These preliminary findings highlight the heterogeneity in epigenetic signatures between the two cell lines. Further work is underway to identify unique regions with potential to serve as therapeutic targets for AML.

MW-EP64: Five-Year Review of Leukaemia Cases at the Institute for Medical Research (2020–2024): Demographic and Clinical Insights

Heather John @ Nicholas, Azian Naila Md Nor, Noraizan Ahmad, Rafizie Rafiq Adha, Nabilah Huda Abd Rahman, Nurul Aqilah Ali, Norfarhana Mazlan, Nadirah Zainal Abidin, Ermi Neiza Mohd Sahid, Norafiza Mohd Yasin, Nor Rizan Kamaluddin, Yuslina Mat Yusoff
Haematology Unit, Cancer Research Centre, Institute for Medical Research, National Institutes of Health, Ministry of Health Malaysia, Persiaran Setia Murni U13/52, Seksyen U13 Setia Alam, Shah Alam, Selangor, Malaysia.

Introduction: Leukaemia is a cancer of the blood and bone marrow, marked by the abnormal growth of white blood cells. It is the sixth most common cancer in Malaysia and the leading malignancy among children. Leukaemia incidence is increasing globally, contributing significantly to cancer-related morbidity and mortality. Understanding its demographic and clinical patterns is essential to improve early diagnosis, guide treatment strategies, and optimise healthcare planning. *Materials and Methods*: This retrospective study analysed leukaemia cases from 2020 to 2024 using clinical and demographic data from the Haematology Unit, Institute for Medical Research. The aim was to evaluate disease patterns, demographic trends, and variations in laboratory parameters. *Results:* A total of 4,086 leukaemia cases were recorded from 2020 to 2024, with the highest number in 2024. Acute Myeloid Leukaemia (AML, 46.3%) was the most common subtype, followed by Acute Lymphoblastic Leukaemia (ALL, 28.4%) and Chronic Myeloid Leukaemia (CML, 12.5%). Most cases were male (55.2%) and of Malay ethnicity (54.3%). CML had the highest mean white blood cell (WBC) count (97.0 $\times 10^9$/L), followed by ALL (53.0 $\times 10^9$/L) and AML (49.3 $\times 10^9$/L). Both ALL and AML cases commonly presented with anaemia (mean haemoglobin: 8.5 g/dL and 8.1 g/dL, respectively) and thrombocytopenia (mean platelet count:

102.7×10³/μL and 100.3×10³/μL), with slightly higher values observed in ALL. The most common translocation observed in ALL was t(9;22)(q34;q31), while t(15;17)(q24;q21) was most frequent in AML. *Discussion:* The clinical and demographic variations observed underscore the biological diversity of leukaemia. Integrating clinical and laboratory data can help improve diagnosis, guide targeted treatments, and support better healthcare planning, especially in resource-limited areas.

MW-EP65: Co-occurrence of Viral-associated Hemophagocytic Lymphohistiocytosis and Langerhans Cell Histiocytosis in a Child: A Rare and Challenging Diagnosis.

Noor Hayati Sabtu[1], Yap Lih Ping[1], Roihan Awang Isa[1], Kimberly Fe Joibi[1], Lee Sen Lin[2]
[1]Unit Hematologi, Jabatan Patologi dan Perubatan Transfusi, Hospital Wanita dan Kanak-Kanak Sabah, Karung Berkunci 187, Kota Kinabalu, Sabah, [2]Unit Histopatologi, Jabatan Patologi, Hospital Queen Elizabeth I, Karung Berkunci 2029, Kota Kinabalu, Sabah.

Introduction: Langerhans cell histiocytosis (LCH) involves clonal dendritic cell proliferation with multisystem organ involvement. Hemophagocytic lymphohistiocytosis (HLH) is a life-threatening hyperinflammatory syndrome driven by dysregulated macrophage and T-cell activation. HLH can be primary or secondary to infections, malignancies, or autoimmune diseases. Their coexistence presents significant diagnostic complexity and therapeutic challenges. *Case Report:* A 14-month-old male presented with prolonged fever, generalized rash, cervical lymphadenopathy, hepatosplenomegaly, anaemia, and thrombocytopenia. Peripheral blood smear revealed 25% abnormal mononuclear cells. Bone marrow aspirate showed numerous large, atypical mononuclear cells suggestive of Langerhans cell histiocytosis (LCH). Histopathological analysis of bone marrow and cervical lymph node biopsies confirmed LCH and demonstrated haemophagocytosis. Immunohistochemistry was positive for CD1a, S100, and Langerin. He fulfilled 5 of 8 HLH-2004 criteria (fever, splenomegaly, bicytopenia, hypertriglyceridemia, haemophagocytosis), and Epstein–Barr virus (EBV) DNA was detected at 1270 IU/mL. A diagnosis of EBV-associated secondary HLH and LCH were established, and treatment was initiated with dexamethasone (10 mg/m2/day) and etoposide per HLH-2004 protocol. *Discussion:* Diagnosing LCH and HLH remains challenging due to their nonspecific clinical presentations and overlap with infection or malignant conditions. Approximately 50% of patients develop HLH concurrently with or shortly after LCH diagnosis, often during active disease. Infections with Epstein–Barr virus (EBV) are the most frequently identified in HLH cases. LCH lacks pathognomonic features; thus, definitive diagnosis relies on histopathological and immunohistochemical findings, particularly CD1a, Langerin, and S100 positivity. Early diagnosis and management require integration of clinical assessment, peripheral smear, bone marrow aspirate, and trephine biopsy for optimal outcomes.

MW-EP66: Assessment of Corrected MCHC Values for Pre- and Post-incubation Utilising Mean Frequent Volume (MFV) and RBC-O in the Reticulocyte Channel.

Veronica Ruth Packianathan, Liana Abd Ghafar, Ramakrishnan Kandasamy, Norsyila Shamsudin, Syarifah Zuraitul Aida Syed Edros, Nor Eshah Salleh, Ruth Shamini Arokiasamy, Kohilavani Tanggaiah, Puteri Azizah Megat Zainon, Muhamad Faizul Abdul Rahman, Wan Suriati Che Mohd Salleh, Azwa Anuar, Nurul Atikah Mohd Fauzi, Intan Khalida Ahmad Iskandar Shah, Hana Shafinaz Jamaluddin, Mardziah Mohamad
Laboratory Medicine Department, University Malaya Medical Centre, Kuala Lumpur, Malaysia.

Introduction: Cold agglutination in EDTA blood samples can lead to falsely elevated mean corpuscular haemoglobin concentration (MCHC) values due to RBC clumping, which distorts RBC count and haematocrit (HCT) measurements. Traditional correction methods involve incubating samples at 37°C for an hour, which delays *Results*. This study aims to evaluate a rapid method for correcting MCHC without prolonged incubation, hypothesising no significant difference between pre-incubation and post-incubation MCHC values. *Materials and Methods:* Retrospective data from University Malaya Medical Centre (January–June 2025) were analysed. Cold-aggregated samples, identified by delta RBC (ΔRBC) > 5%, were assessed. HCT correction was done using mean frequent volume (MFV), and corrected MCHC values were calculated. Statistical analyses included Passing–Bablok regression to assess agreement and paired two-tailed t-test for mean comparison, with p-value > 0.05 considered non-significant. *Results:* The Passing–Bablok regression showed constant and proportional biases (intercept = -98.80 g/L, slope = 1.299). The Cusum test confirmed linearity (p = 0.99), and residual variability was moderate (RSD = 11.66 g/L). The paired t-test revealed no significant difference between pre- and post-incubation MCHC values (mean difference = 0.26 g/L, p = 0.913). *Discussion:* The *Results* support that the rapid pre-incubation correction method is statistically equivalent to the conventional post-incubation technique, offering a time- efficient and accurate alternative for MCHC correction in clinical practice.

MW-EP67: Cold Autoimmune Haemolytic Anaemia Precipitated by Urosepsis in a Diabetic Patient on Traditional Medication

Abdul Hanan Abdullah[1], Nur Farhanie Mahadi[2], Nur Ilyia Syazwani Saidin[2], Mohamad Zikir Ismail[1]
[1]Department of Internal Medicine, School of Medical Science, Universiti Sains Malaysia, Kota Bharu, Kelantan, [2]Department of Haemotology, School of Medical Science, Universiti Sains Malaysia, Kota Bharu, Kelantan.

Introduction: Cold autoimmune haemolytic anaemia (AIHA) is a subtype of AIHA mediated by cold-reactive autoantibodies, often secondary to infections, lymphoproliferative disorders, or autoimmune conditions. Timely recognition and appropriate therapy are essential to prevent life-threatening complications. *Case Report:* We report a case of a 46-year-old woman with type 2 diabetes mellitus and bilateral ureterocele with hydronephrosis, who presented with lethargy, dizziness, and tea-coloured urine. She had been consuming traditional remedies for diabetes over the past year. On examination, she appeared both pale and jaundiced. Laboratory evaluation revealed leucocytosis (white blood cell of 12 × 10⁹/L), severe anaemia (haemoglobin of 6.6 g/dL), thrombocytosis (platelets of 704 × 10⁹/L), reticulocytosis (9.29%), indirect hyperbilirubinemia, elevated LDH (679 U/L), and marked red blood cells agglutination on peripheral blood smear. The direct Coombs test was positive for anti-C3d (3+) but negative for anti-IgG, consistent with cold AIHA. Serologic tests for infection screening were negative. However, urine culture grew *Klebsiella pneumoniae*, indicating a urosepsis trigger. She was treated with intravenous (IV) hydrocortisone followed by methylprednisolone and a full

course of IV piperacillin-tazobactam. Due to severe anaemia and signs of tissue hypoperfusion (altered sensorium, lactate acidosis), packed cell transfusion was initiated with no incompatibility issues noted during crossmatching. Haemoglobin stabilized following transfusion. *Discussion:* This case highlights the importance of considering cold AIHA in patients presenting with haemolytic anaemia and recent infection. The absence of bleeding and rapid haemoglobin decline, coupled with a background of traditional medication use, complicated the diagnostic process. *Conclusion:* Cold AIHA should remain a differential diagnosis in cases of unexplained anaemia, particularly post-infection. Peripheral smear morphology and prompt immunohaematological workup are critical for diagnosis and management.

MW-EP68: Haematological and Biochemical Profiles in Dengue Patients with and without Warning Signs

Wan Suriana Wan Ab Rahman[1,2*] Gokulakannan Venkatesan[1], Wan Nazatul Shima Shahidan[3], Salfarina Iberahim[2,4]
[1]School of Dental Sciences, Universiti Sains Malaysia Health Campus, Kota Bharu, Kelantan, Malaysia. [2]Hospital Universiti Sains Malaysia Health Campus, Kota Bharu, Kelantan, Malaysia. [3]Department of Pharmacology, School of Medical Sciences, Universiti Sains Malaysia Health Campus, Kota Bharu, Kelantan, Malaysia. [4]Department of Haematology, School of Medical Sciences, Universiti Sains Malaysia Health Campus, Kota Bharu, Kelantan, Malaysia

Introduction: Dengue fever presents with a range of clinical manifestations. Early identification of laboratory markers associated with disease severity is crucial for timely clinical decision making and improved patient outcomes. This study aims to compare haematological and biochemical profiles in dengue patients categorized as with and without warning signs, based on World Health Organization 2009 classification criteria. *Material and Methods:* This retrospective study analysed laboratory records of confirmed dengue patients. For those without warning signs, parameters assessed included haemoglobin, haematocrit, platelet count, white blood cell count, and red blood cell count. In patients with warning signs, additional variables were evaluated, including coagulation profiles, and liver enzymes. Statistical comparisons between the groups were performed using appropriate parametric or non-parametric tests based on data distribution. *Results:* Preliminary analysis demonstrates that dengue patients presenting with warning signs exhibit significantly lower platelet counts, elevated haematocrit levels, and increased hepatic transaminases, relative to patients without warning signs. Additionally, prolonged coagulation profile was more frequently observed in the warning-sign cohort, indicating potential early hepatic dysfunction and coagulopathy. Although comprehensive statistical analysis is ongoing, these initial findings suggest that specific haematological and biochemical parameters serve as early indicators of disease severity and progression. *Discussion:* The utility of current haematological and biochemical markers is affirmed as valuable tools in the clinical management of dengue. Their use in early assessment facilitates timely risk stratification, improves patient monitoring, and supports informed decision-making, ultimately contributing to better clinical outcomes and resource optimisation during dengue outbreaks.

MW-EP69: Beyond Deletion: A Retrospective Study of the Geta-globin Gene (*HBB*) Cluster Mutations Driving High HbF in Malaysian Beta-thalassaemia

Nur Aisyah Aziz[1,3*], Ezzanie Suffya Zulkefli[1], Faidatul Syazlin Abdul Hamid[1], Syahzuwan Hassan[1], Syahira Lazira Omar[1], Velintina Matnih[1], Melanie Ling Mohd Din[1], Nurul Amira Jamaludin[1], Wan Rohani Wan Taib[3], Azian Naila Md Nor[1], Ermi Nieza Mod Sahid[1], Ezalia Esa[2], Norafiza Mohd Yasin[1], Yuslina Mat Yusoff[1]
[1]Haematology Unit, Cancer Research Centre (CaRC), Institute for Medical Research (IMR), National Institutes of Health (NIH), Ministry of Health (MOH), Setia Alam, Shah Alam, Selangor, Malaysia, [2]Virology Unit, Infectious Disease Research Centre (IDRC), Institute for Medical Research (IMR), National Institutes of Health (NIH), Ministry of Health (MOH), Setia Alam, Shah Alam, Selangor, Malaysia, [3]Faculty of Health Sciences (FSK), University Sultan Zainal Abidin, Kuala Terengganu, Terengganu, Malaysia.

Introduction: Beta-thalassemia, caused by mutations in the *HBB* gene, exhibits a range of clinical phenotypes influenced by foetal haemoglobin (HbF) levels. While deletions within the beta-globin gene cluster impact HbF, elevated HbF (>5%) can also arise from other genetic factors, including diverse *HBB* mutations and co-inheritance of multiple mutations. This study aims to characterise *HBB* mutations in beta-thalassemia patients with elevated HbF to improve molecular diagnostics. *Materials and Methods:* This retrospective analysis encompasses patients referred from January 2020 to December 2024, with HbF levels exceeding 5%, detected via HPLC and/or capillary electrophoresis. DNA extracted from peripheral blood samples was examined using PCR, MLPA, and Sanger sequencing to identify mutations. Haematological parameters and HbF levels were compared across different genotype groups. *Results:* A total of 779 cases were included and categorized based on their genotypes: heterozygous beta- thalassemia (n=36, 4.6%), double heterozygous beta-thalassemia (n=9, 1.2%), homozygous beta-thalassemia (n=5, 0.6%), heterozygous delta/beta-thalassemia (n=639, 82%), compound heterozygous delta/beta-thalassemia with beta-thalassemia (n=9, 1.2%), double heterozygous delta/beta-thalassemia (n=1, 0.1%), homozygous delta/beta-thalassemia (n=5, 0.6%), and individuals with a normal beta-globin gene (n=75, 9.6%). The Kruskal-Wallis H test indicated significant differences ($p \leq 0.001$) in RBC, Hb, Hct, MCV, MCH, RDW, and HbF (CE and HPLC) in at least one of the genotype groups. MCHC did not demonstrate a significant difference ($p = .101$). *Discussion:* The cohort mainly consisted of delta/beta-thalassemia patients and normal individuals. Variations in haematological parameters highlight profiles linked to different genotypes. Molecular characterisation is important for diagnosis, prognosis, and counselling. The genetic diversity of beta-globin mutations in Malaysia contributes to clinical variability; understanding mutations related to elevated HbF can optimise molecular testing, streamline diagnosis, and improve cost-efficiency.

MW-EP70: Determining the Normal Range of Erythrocyte Sedimentation Rate at Hospital Pakar Universiti Sains Malaysia

Zefarina Zulkafli[1], Nurul Asyikin Nizam Akbar[1], Wan Suriana Wan Ab Rahman[2], Mohd Nazri Hassan[1], Noor Haslina Mohd Noor[1], Shafini Mohamed Yusoff[1], Salfarina Iberahim[1], Rosnah Bahar[1], Marne Abdullah[1], Marini Ramli[1]
[1]Haemotology Department, School of Medical Sciences, Health Campus, Universiti Sains Malaysia, Kubang Kerian, Kelantan, Malaysia, [2]School of Dental Sciences, Health Campus, Universiti Sains Malaysia, Kubang Kerian, Kelantan, Malaysia

Introduction: Erythrocyte sedimentation rate (ESR) is often used as an indicator of inflammation or other medical conditions.

Understanding the normal ESR range for blood donors is essential to accurately interpret the result and ensure that potential health conditions are appropriately identified. This study aims to determine the normal range for ESR for Haematology Laboratory at Hospital Pakar Universiti Sains Malaysia (HPUSM), providing a reference for clinical evaluation. *Material and Methods:* A total of 241 (121 females and 120 males) healthy individuals were included in the study, with ESR measurements taken using The MIX-RATE® X20 ESR analyser. *Results*: This study showed that the range of ESR for male was 1–22 mm/hour and female was 0-16, consistent with typical ranges observed in healthy adults. *Discussion*: The study successfully established the normal range of ESR for Haematology Laboratory at HPUSM. These findings contribute valuable data for clinical decision-making, ensuring accurate interpretation of ESR *Results* in the region. Further research may be needed to explore additional factors influencing ESR values, such as demographic differences, lifestyle habits, and environmental factors, to refine the reference range and enhance its clinical utility.

MW-EP71: Clinical Utility of Extended Red Blood Cell Parameters in Diagnosing Iron Deficiency Anaemia Among Children in Hospital Universiti Sains Malaysia

Fatma Basyira Jamallodin[1], Marini Ramli[1], Wan Suriana Wan Ab Rahman[2], Nur Ilyia Syazwani Saidin[1]
Department of Haematology, School of Medical Sciences, Health Campus University Sains Malaysia, Kubang Kerian, Kelantan, Hospital Pakar Universiti Sains Malaysia, Kubang Kerian, Kelantan; [2]School of Dental Sciences, Health Campus, Universiti Sains Malaysia, Kubang Kerian, Kelantan, Malaysia

Introduction: Iron deficiency anaemia (IDA) is the most common anaemia affecting children under five years of age, often characterised by hypochromic and/or microcytic red cells. Therefore, the aim of this study was to determine the proportion of iron IDA among hypochromic patient, and to correlate the extended red blood cell (RBC) parameters with traditional markers of iron deficiency. *Material and Methods*: A cross-sectional study was conducted among hypochromic children, aged between six to 60 months who were treated at the paediatric clinic and ward Hospital Pakar Universiti Sains Malaysia (HPUSM) from January 2024 to April 2025. The FBC and extended FBC parameters was performed using Mindray BC6200 analyser and serum iron, TIBC and ferritin were performed using Roche Cobas 6000 analyser. The result data were obtained from Laboratory Information System (LIS). The Mentzer index and transferrin saturation (TSAT) were calculated using standard formula. *Result*: The mean age of the patients was 1.69±1.27 years. Among all children, 67 (57.8%) were boys with a male-to-female ratio of 1.45 among those with iron deficiency. A total of 93 children (80.2%) were found to be iron deficient. By using Mentzer Index, 22.5% of iron deficient were possibly thalassaemic, 31.2% was latent iron deficient and 46.3% was IDA. The higher incidence of IDA was observed in the 6-to-24-month age group (39%) compared to the 24-to-60-month group (31%). The mean haemoglobin level among children with IDA was 9.35±1.26 g/dL. The mean reticulocyte haemoglobin equivalent (RHe) was 17.1±8.99pg with a median of 17.70pg. There was a statistically positive significant linear correlation between RHe with haemoglobin ($r=0.453$; $p<0.001$) and MCH ($p=0.274$; $p=0.038$). There was a significant negative correlation with ferritin ($r=-0.613$, $p=<0.001$). *Discussion:* IDA was prevalent among hypochromic children under five years old at HPUSM, with the higher incidence observed in those aged 6 to 24 months. RHe showed strong correlations with traditional iron markers, particularly haemoglobin and serum ferritin, indicating its potential value as an early biomarker of iron deficiency. However, the lack of correlation between RHe and TSAT suggests that RHe may not fully replace traditional tests. In conclusion, RHe can enhance early detection of IDA in children, especially when used alongside established diagnostic parameters.

MW-EP72: Megakaryocytes Beyond the Marrow: Striking Peripheral Blood Finding in Sepsis

Aznor Wahidah Khairul Nor Azman, Kautsar Mohd Zahari, Siti Nur Azwa Md Shahid
Hospital Sultan Idris Shah Serdang, Jalan Puchong, 43000 Kajang, Selangor.

Introduction: Megakaryocytes are large, specialised bone marrow cells that responsible for the production of platelets, which are essential for blood clotting and haemostasis. The presence of megakaryocytes in the peripheral blood is a rare finding, typically indicative of significant bone marrow pathology. However, they can temporarily enter the bloodstream even in non-haematological diseases during intense bone marrow stress and naturally presence in neonates or young infants. *Case Report:* A 21-day-old baby presented with signs of systemic inflammatory response and was diagnosed with septic shock secondary to meningitis and enterocolitis. Blood count revealed leucocytosis with neutrophilia, monocytosis and thrombocytosis. Peripheral blood smear noted the unusual presence of large cells with multilobulated nuclei and abundant weakly basophilic granular cytoplasm at the feathered edge, signifies circulating megakaryocytes. There was no evidence of haematologic malignancy and the megakaryocytes was no longer detected on follow-up smear. *Discussion:* Given that megakaryocytes can be present at the tail of blood smears, careful examination of the feathered edge is advised. The appearance of megakaryocytes in peripheral blood may reflect severe bone marrow stress in the setting of systemic infection. In our case, the findings were attributed to reactive changes secondary to intense systemic inflammation. Awareness of this occurrence is crucial to avoid misinterpreting it as evidence of a primary haematologic disease. Therefore, evaluation of a peripheral blood smear should always be interpreted in conjunction with the clinical context and additional laboratory findings. This case also illustrates the dynamic response of bone marrow under extreme inflammatory conditions.

MW-EP73: Analysis of DNA Repair and MicroRNAs in Mouse Foetal-derived Haematopoietic Stem/Progenitor Cells in response to In-utero Benzene Exposure

Zariyantey Abd Hamid[1]*, Nur Afizah Yusoff[1], Izatus Shima Taib[1], Siti Balkis Budin[1] and Siti Razila Abdul Razak[2]
[1]Biomedical Science Programme, Centre of Diagnostic, Therapeutic and Investigative Studies,Faculty of Health Sciences, Universiti Kebangsaan Malaysia, Jalan Raja Muda Abdul Aziz,Kuala Lumpur, Malaysia, [2]Department of Biomedical Science, Advanced Medical and Dental Institute, Universiti Sains Malaysia, Kepala Batas, Pulau Pinang, Malaysia

Introduction: Benzene is haematotoxic and leukaemogenic. Maternal exposure confers haematotoxic risk to developing foetus,

especially during hematopoietic organogenesis in foetal liver by targeting haematopoietic stem/progenitor cells (HSPCs) niche. This study investigates the effect of in-utero exposure of Hydroquinone (HQ), a benzene metabolite on the DNA repair and microRNAs (miRNAs) targeting foetal HSPCs comprising of myeloid, erythroid and pre-B lymphoid lineages. *Materials and Methods:* Twenty-four pregnant ICR mice grouped as Control, HQ25, and HQ50, received 25 and 50 mg/kg HQ orally on gestational days (GD) 12, 14, and 16. Foetal liver cells were isolated on day 18, cultured and plated on colony-forming unit (CFU) assay for 7-14 days to grow HSPCs. DNA repair proteins [RAD51 (HR) and DNA-PKcs (NHEJ)] and miRNAs associated with self-renewal (*miR-196b, miR-29a*) and differentiation (*miR-181a*) were analysed. *Results:* In utero HQ exposure significantly ($p<0.05$) increased DNA-PKcs and RAD51 protein expression in a dose-dependent manner in foetal liver cells and HSPCs. A significant ($p<0.05$) reduction of *miR-196b* (both HQ) and *miR-181a* (HQ25) with increased in *miR-29a* (both HQ) was noted in liver cells. Significant ($p<0.05$) increased of *miR-196b* expression in all HSPCs, as well as *miR-181a* in myeloid (HQ50) and Pre-B lymphoid progenitors (both HQ) were notable. miR-181a expression was reduced significantly ($p<0.05$) in myeloid (HQ25) and erythroid (both HQ). A significant ($p<0.05$) reduction in *miR-29a* was observed only in Pre-B lymphoid progenitor at HQ50. *Discussion:* Conclusively, in utero HQ exposure induces DNA repair and alters miRNA expression in a lineage-specific manner, indicating potential haematotoxicity in foetal HSPCs.

MW-EP74: Adoption of Machine Learning in Morphological Features Analysis of Colony Forming Unit for Mouse Haematopoietic Stem / Progenitor Cells

Zariyantey Abd Hamid[1], Ramanaesh Rao Ramakrishna[1], Wan Mimi Diyana Wan Zaki[2], Aqilah Baseri Huddin[2]
[1]Biomedical Science Programme, Centre for Diagnostic, Therapeutic and Investigative Studies, Faculty of Health Sciences, Universiti Kebangsaan Malaysia, Kuala Lumpur, Malaysia, [2]Department of Electrical, Electronic & Systems Engineering, Faculty of Engineering & Built Environment, Universiti Kebangsaan Malaysia, Bangi, Malaysia

Introduction: Hematopoietic colony-forming unit (CFU) assay assesses the proliferative and differentiation potential of hematopoietic stem and progenitor cells (HSPCs). Colony characteristics (size, shape, colour, morphology) indicate progenitor lineage. Traditionally, CFU analysis involves manual classification of colonies under a microscope, which is time-consuming, subjective, and requires expert training. Machine learning provides a solution for automated, standardised, and high-throughput CFU analysis. This study adopts a machine learning approach to analyse morphological features of 5 common CFU types as follows: erythroid (CFU-E), myeloid (CFU-G; CFU-GM; CFU-M), and lymphoid pre-B (CFU-PB). *Materials and Methods:* Freshly isolated mouse bone marrow cells were cultured for 24 hours prior to CFU assay for 7-14 days according to progenitor lineage. Colonies were observed under inverted light microscope. Images were captured using a smartphone device attached to microscope lens adapter. 638 images were augmented, resulting into 5104 images that later went through pre-processing, segmentation, feature extraction, feature selection and classification. Eleven morphological features were extracted using region props. Significant features ($p<0.05$) were selected for classification of CFU types using the Support Vector Machine (SVM) classifier. *Results*: Analysis showed that 8 from the 11 extracted morphological features showed significant differences ($p<0.05$) for the 5 types CFUs. The effectiveness of the features to classify the 5 types CFUs were tested using SVM. Combined morphological features had the accuracy of 75.1 % in classifying CFUs. *Discussion:* Conclusively, the adoption of machine leaning for analysing morphological features shows promise as an automated classification system for CFUs. The invention can foster CFU assay application in clinical and research concerning the study of HSPCs.

MW-EP75: An Uncommon Path: A Rare Transformation of Chronic Myeloid Leukaemia (CML) to Acute Megakaryoblastic Leukaemia

Rosmaniza Muhamat Yusoff, Raja Muhamad Zul Hatta Raja Ismail, Nizmah Mahani Mokhri
Haemotology Unit, Department of Pathology, Hospital Queen Elizabeth, 88586 Kota Kinabalu, Sabah.

Introduction: Chronic myeloid leukaemia (CML) typically progresses from chronic phase to blast crisis, most commonly of myeloid or lymphoid lineage. Transformation into acute megakaryoblastic leukaemia (AML-M7) is extremely rare and associated with a poor prognosis. We report a case of CML in chronic phase that progressed to AML-M7, highlighting its clinical course and treatment challenges. *Case Report:* A 21-year-old male was diagnosed with chronic phase CML at the age of eight in July 2011 and was started on imatinib therapy. Cytogenetic and molecular analyses showed t (9;22) and the presence of an ASXL1 mutation, without evidence of the *BCR::ABL1* T315I mutation. During follow-up in November 2024, noted increasing white cell count (WCC) and physical examination revealed splenomegaly. Full blood counts demonstrated Hb 9.6 g/dL, WCC 69.9 x 10^3/µl, and platelets 478 x 10^3/µl, with 20% circulating blasts on peripheral blood smear. Bone marrow examination confirmed disease progression to acute myeloid leukaemia (AML), subtype M7 from trephine biopsy. The patient received induction chemotherapy with DA 2+5 and FLAG regimens; however, post-therapy marrow assessment indicated persistent disease, with no remission achieved. *Discussion:* Blast transformation of CML into AML, particularly the megakaryoblastic subtype (AML-M7), is exceedingly rare and indicates a poor prognosis. The presence of an ASXL1 mutation in CML is associated with resistance to imatinib and an increased risk of disease progression. This case underscores the importance of vigilant molecular surveillance and highlights the limited efficacy of conventional induction regimens in managing such rare transformations.

MW-EP76: Caught off Guard: Acute Promyelocytic Leukaemia (APML) in a Cardiac Patient without Warning Signs

Nur Amanina Abd Nasir, Nazurah Khalid, Siti Nur Azwa Md Shahid, Norsafina Zainal
Haematology Unit, Department of Pathology, Hospital Sultan Idris Shah, Serdang.

Introduction: APML is a distinct subtype of acute myeloid leukaemia characterised by the t(15;17) translocation, leading to the *PML::RARα* oncogenic fusion protein, which disrupts blood cell development. Diagnosis relies on identifying this genetic abnormality and monitoring treatment response. In clinically stable patients, particularly those with underlying comorbidities such

as two-vessel coronary artery disease, diagnosing and managing APML can be complex. The absence of symptoms may delay recognition, risking poorer outcomes. Treatment in such patients requires a careful balance to prevent exacerbating cardiovascular conditions, considering drug interactions and potential side effects like myelosuppression and cardiac complications. *Case Report:* A 61-year-old man with stable ischemic heart disease was found to have leucopoenia and thrombocytopenia during a routine evaluation for elective percutaneous coronary intervention, despite being asymptomatic with white blood cell of 1.65×10^3/uL, haemoglobin 15.3 g/dL, and platelets 58×10^3/uL, prompting further evaluation. The peripheral blood smear reveals abnormal promyelocytes, raising concern for APML, which warrants urgent diagnosis and treatment initiation. The final molecular and cytogenetic result shows a *PML::RARα* translocation t(15;17). Nevertheless, the patient denies constitutional symptoms such as fever or weight loss and has not experienced any bleeding tendencies. *Discussion:* Managing APML in this context necessitates a heterodox approach: tailoring therapy to avoid cardiac decompensation, vigilant monitoring for adverse effects, and coordinating multidisciplinary care to address both leukaemia and cardiovascular risk. Early diagnosis and careful management are vital for improving outcomes in such complex cases.

MW-EP77: Beyond Blue: Verifying Perl's Stain for Accurate Bone Marrow Iron Quantification

Kasmawati Abu Bakar[1], Hajariah Awang Maharum[1], Azly Sumanty Ab. Ghani[1], Zakiah Nurasyikin Mohamed Halim[1], Ton Ali Isram Ton Abdullah[1], Ahmad Shahabuddin Mamat[1], Ehram Jamian[2], Noor Hidayat Hussain[3]

[1]Haemotology Unit, Pathology Department, Hospital Sultanah Nur Zahirah (HSNZ), Ministry of Health Malaysia, Kuala Terengganu, Terengganu Darul Iman; [2]Haemotology Unit, Pathology Department, Hospital Sultanah Aminah (HSA), Ministry of Health Malaysia, Johor Bahru, Johor Darul Takzim; [3]Chemical Pathology Unit, Pathology Department, Hospital Sultan Idris Shah (HSIS), Ministry of Health Malaysia, Serdang, Selangor Darul Ehsan

Introduction: In the meticulous world of Haematology, where every cell tells a story, verification of staining methods is often overlooked, yet it's a critical requirement under MS ISO 15189:2022. According to CLSI EP12-Ed3-IG-2023 guideline, manufacturer's performance claims must be verified before reporting patient *Results* to ensure that a new examination's performance aligns with its stated claims. Additionally, whenever new reagent lots, their consistency with current lots should be verified before use. Notably, MS ISO 15189:2022 (clause 7.3.1) mandates that laboratories use examination methods validated for their intended purpose to ensure clinical accuracy in patient testing. This study aimed to verify the precision, sensitivity, and specificity of Perl's stain, a key diagnostic tool for detecting iron (Fe^{3+}) in haematological specimens. *Materials and Methods:* Among commercially available options, Haematognost Fe (Sigma Aldrich) was evaluated using 20 bone marrow aspirate (BMA) specimens (10 positive, 10 negative) from Hospital Sultanah Nur Zahirah (HSNZ) as per CLSI EP12-Ed3-IG-2023 guidelines and analysed microscopically by Haematologists. *Results:* Sensitivity, along with positive predictive value (PPV) and negative predictive value (NPV), was calculated using a 2×2 contingency table, while specificity was determined through observational assessment. *Results* demonstrated 100% sensitivity, PPV, and NPV for nuclei, cytoplasm, and free iron granules, aligning with manufacturer claims. Specificity was confirmed by the absence of non-specific background staining. *Discussion:* These *Results* verify the precision of the Perl's stain, confirming its suitability for diagnostic use. As evidenced, Perl's stain reliably detects bone marrow iron levels, aiding in anaemia and hemochromatosis diagnosis. Strict protocol compliance ensures result accuracy and better patient care.

MW-EP78: Primary Plasma Cell Leukaemia: Morphological Insight and Diagnostic Clarity in an Unusual Presentation

Adibah Daud[1,2], Nur Rasyidah Mohd Kamal[3], Daniel Hazim Mohd Shukri[2], Muhammad 'Aqil Nazahah Mohamad Mustafa[2], Wan Amal Hayati Wan Hassan[3], Wan Zuhairah W Embong[3], NorAiniza Mansor[3], Zakiah Nurasyikin Mohamed Halim[3], Azly Sumanty Ab Ghani[3], Sumaiyah Adzahar[1,2], Yip Kam Hoo[4]

[1]Department of Pathology and Medical Laboratory, Faculty of Medicine, Universiti Sultan Zainal Abidin, Kuala Terengganu; [2]Department of Pathology and Medical Laboratory, Hospital Sultan Zainal Abidin, Kuala Nerus; [3]Department of Pathology, Hospital Sultanah Nur Zahirah, Kuala Terengganu; [4]Department of Medicine, Hospital Sultanah Nur Zahirah, Kuala Terengganu

Introduction: Plasma cell leukaemia (PCL) is a rare and aggressive form of plasma cell dyscrasia, accounting for less than 5% of plasma cell neoplasms. The International Myeloma Working Group (IMWG) recently updated the diagnostic criteria, defining PCL as the presence of ≥5% circulating plasma cells or an absolute plasma cell count of $\geq 0.5 \times 10^9$/L in the peripheral blood. PCL may present as a primary condition or as a transformation from multiple myeloma. *Case Report:* We report a case of primary PCL in a previously healthy 66-year-old woman who presented with a one-month history of productive cough and weight loss. The full blood count showed mild anaemia, leucocytosis, and borderline thrombocytopenia. The peripheral blood film demonstrated 20% circulating plasma cells. Her bone marrow aspiration revealed 65% plasma cells with morphological heterogeneity, including mature, immature, and plasmablastic cells. Immunophenotyping showed a presence of 32.7% clonal plasma cells with positive cytoplasmic kappa. Serum protein electrophoresis revealed a monoclonal IgA Kappa spike with a free light chain ratio of >500. *Discussion:* This case meets the latest diagnostic criteria by the IMWG for primary PCL. The clinical history reflects the non-specific and often insidious onset of PCL. The presence of leucocytosis and borderline thrombocytopenia further underscores the haematological disruption typical of this malignancy. This case also highlights the diverse morphological features of the clonal plasma cells observed in the bone marrow. Due to the disease's aggressive nature and poor prognosis, early recognition and diagnosis are essential for initiating prompt and effective treatment.

MW-EP79: Leukaemoid Features in Melioidosis Concealing Underlying Acute Myeloid Leukaemia: A Diagnostic Oversight

Farhas Salisah Derani, Sabariah Md Noor, Faridah Idris

Department of Pathology, Faculty of Medicine and Health Sciences, UPM, Serdang, Malaysia, Hospital Sultan Abdul Aziz Shah, Persiaran Mardi - UPM, Serdang, Selangor

Introduction: Patients with AML are usually presented with systemic symptoms. However, diagnostic challenges emerge with

concurrent systemic illness notably during infection. *Case Report:* A 58-year-old man with diabetes mellitus came with fever and lethargy. On examination, hepatomegaly noted. Initial blood *Results*: haemoglobin of 3.6 g/dL, white blood cells of 8.47×10^9/L, and platelets 54×10^9/L. Blood film shows leucoerythroblastic pictures with presence of 4% suspicious abnormal mononuclear cells. Ultrasound reveals liver abscess. *Burkholderia pseudomallei* antibody IgM was detected, suggestive of melioidosis. Antibiotics were commenced. After completed treatment, the patient was discharged. Repeated blood film showing >20% blasts, which are small to moderate in size with minimal amount of bluish cytoplasm with conspicuous nucleoli, suggestive of acute leukaemia. His condition was stable during this interval; thus, the blasts are considered as a leukaemoid reaction secondary to disseminated melioidosis. The patient has been advised for further investigation such as bone marrow aspiration to rule out acute leukaemia. Regrettably, the patient refused. Repeated blood film showing increasing blasts to 44% with similar morphology. After serial blood film monitoring, bone marrow aspiration was done; showed 41.1% blasts, which were positive for CD34, HLA-DR, CD117, CD13, CD33 and CD4, confirming AML. Before chemotherapy, he developed a right thigh nodule. Biopsy performed and histopathologic examination confirmed diagnosis of leukaemia cutis. Fortunately, the patient responded well to chemotherapy. *Discussion:* Infection can cause formation of blast looking myeloid leukaemoid reactions. This case highlights that the persistence of bicytopenia with presence of >20% blasts should warrant clinical suspicion of additional underlying conditions, as it may indicate disease progression or concurrent complications.

MW-EP80: Extramedullary myeloid blast phase in Chronic Myeloid Leukaemia involving the gastrointestinal system: A case report

Nuur Qamarina Mohd Bakri[1], Asmawiza Awang[1], Yuslina Mat Yusoff [2], Nurimatussolehah Sarijan[3], Chin Loi Khim[3], Emynurshiela Anuar[3], Nur Ashikin Azemi[3], Sharifah Suryani Syed Rahim Shah[4], Munirah Md Noor[5], Nur Azura Suliman[1*]

[1]Department of Pathology, Hospital Kuala Lumpur, Wilayah Persekutuan Kuala Lumpur, [2]Haematology Unit, Cancer Research Centre (CaRC), Institute for Medical Research (IMR), National Institute for Health (NIH) Setia Alam, Selangor, [3]Department of Pathology, Hospital Tunku Azizah, Wilayah Persekutuan Kuala Lumpur, [4]Department of Medicine, Hospital Kuala Lumpur, Wilayah Persekutuan Kuala Lumpur, [5]Department of Pathology, Hospital Queen Elizabeth, Kota Kinabalu, Sabah

Introduction: Blast phase in CML is defined by presence of ≥20% blasts in peripheral blood and/or bone marrow, evidence of extramedullary blast proliferation, or detection of lymphoblasts in peripheral blood or bone marrow. *Case Report:* We report a 35-year-old male, diagnosed with chronic phase CML in 2022 and treated with tyrosine kinase inhibitor (TKI). The treatment was not optimised due to thrombocytopenia, and he was unable to achieve major molecular response. He later presented with upper gastrointestinal bleeding and hyperleukocytosis. Physical examination revealed hepatosplenomegaly without lymphadenopathy. Peripheral blood film, bone marrow aspirate, immunophenotyping, and trephine biopsy showed features of CML in chronic phase with presence of <10% myeloblasts. Oesophagogastroduodenoscopy revealed an infiltrative duodenal mass with a Forrest IIa ulcer. Histopathology examination confirmed extramedullary myeloid blast phase in CML. Cytogenetic study showed an abnormal male karyotype with t(9;22) and addition on 5q. Molecular testing showed detection of major *BCR::ABL1* fusion transcript. Real-time quantitative PCR of *BCR::ABL1* p210 transcripts after induction chemotherapy was 13.05% (IS). Next-generation sequencing detected ABL1:c.1375G>A;p.E459K mutation, which is associated with acquired resistance to TKI. *Discussion:* Extramedullary blast proliferation in CML can involve various site, particularly lymph nodes, skin, bone and central nervous system. This case is notable due to rare gastrointestinal tract involvement. Worsening full blood count results despite TKI treatment should raise suspicion of disease progression or TKI resistance. This case emphasises the importance of recognising atypical extramedullary presentations in CML and highlights the clinical utility of molecular tests in identifying mutations that can influence patient management.

MW-EP81: Rare *SET::NUP214* Fusion in Paediatric B-cell Acute Lymphoblastic Leukaemia: A Case of High-Risk Genetic Lesion

Siti Shahrum Muhamed. Said[1], Suzana Zainol[1], Norazlina Azman[1], Shenaz Banu Said Khan[1], Nurimatussolehah Sarijan[1], Fatin Adlia Arifin[1], Wee Nee Tan[2], Mimi Azura Aziz[1]

[1]Department of Pathology, Hospital Tunku Azizah, Kuala Lumpur, Malayzia; [2]Department of Paediatric, Hospital Tunku Azizah, Kuala Lumpur, Malaysia.

Introduction: B-cell Acute Lymphoblastic Leukaemia (B-ALL) is the most common cancer in children, typically affecting those aged 2 to 5 years, with a survival rate of around 85–90%. Prognosis depends on factors such as age, white blood cell count, genetic mutations, CNS involvement and presence of minimal residual disease (MRD) after treatment. The *SET::NUP214* fusion gene, a rare abnormality typically found in T Acute Lymphoblastic Leukaemia in adult male, is exceptionally rare in paediatric B-ALL. It is linked to high-risk features including chemoresistance and complex genetic alterations, contributing to poorer prognosis. *Case Report:* We report on a 2-year-old girl who presented with one month history of fever, lethargy, joint pain and leucocytosis (WBC 28.4 x 10^9/L). Peripheral blood smear showed numerous blasts and bone marrow examination confirmed 70% blasts. B-ALL was diagnosed by immunophenotyping by flow cytometry. The CSF smear examination showed no blast. While conventional karyotyping was normal, molecular testing revealed the rare *SET::NUP214* fusion gene, indicating a high-risk genetic lesion. She was treated according to the ALL-BFM protocol. MRD was detected at 1.4% on Day 15, which became undetectable by Day 36. Due to the presence of a high-risk fusion gene, therapy was intensified with blinatumomab. *Discussion*: This case underscores the power of molecular testing in identifying cryptic mutations like *SET::NUP214,* even in the absence of abnormal karyotypes. Targeted therapies such as blinatumomab, paired with early MRD monitoring, offer hope for improving outcomes in high-risk paediatric B-ALL. This case highlights a shift towards personalized treatment, where genetic findings are pivotal in guiding clinical decisions and optimising patient care.

MW-EP82: Portal vein obstruction in the context of Myeloproliferative Neoplasm with Normal Blood Counts: A Diagnostic Challenge

Beverly Kuok Li Lie
Unit Haematology, Department of Pathology, Hospital Pulau Pinang

Introduction: Portal vein is a rare site for venous obstruction, which is often associated with underlying pathological conditions such as myeloproliferative neoplasms (MPNs). MPNs are clonal hematopoietic disorders characterised by overproduction of mature blood cells. Rarely, patients may present with non-classical features, such as normal peripheral blood counts and isolated splenomegaly, which can mask the diagnosis of MPNs. *Case report:* A 27-year-old Chinese woman with a background of non-cirrhotic portal hypertension and portal vein obstruction experienced recurrent upper gastrointestinal bleeding from oesophageal varices, treated with banding. Physical examination showed splenomegaly without hepatomegaly or lymphadenopathy. Her full blood count revealed normal haemoglobin, white blood cell, and platelet levels. Peripheral blood film showed platelet anisocytosis, with no leucoerythroblastic changes or blasts. Further molecular testing detected the *JAK2 V617F* mutation. Bone marrow biopsy demonstrated a hypercellular marrow with megakaryocytic hyperplasia, pleomorphic megakaryocytes, and significant fibrosis (WHO reticulin grade 2). Levels of protein C, protein S, Factor V, and Factor X were reduced, likely secondary to underlying liver dysfunction. Probable diagnosis of myeloproliferative neoplasm not otherwise specified (MPN-NOS) was made. The patient remains asymptomatic and is not on anticoagulation therapy due to bleeding risk. *Discussion:* MPNs typically present with elevated peripheral blood counts and increased risk of thrombosis, driven by mutations in *JAK2*, *MPL*, or *CALR*. However, rare cases with normal blood counts can make diagnosis challenging. Without molecular testing, these atypical presentations may be missed or delayed. In conclusion, the discovery of genetic mutations has revolutionized the diagnostic framework for MPNs, supporting earlier detection, improved prognostic stratification, and targeted management strategies.