HKSH
The Hong Kong Society of Haematology

THE HONG KONG SOCIETY OF HAEMATOLOGY

ANNUAL SCIENTIFIC MEETING 2026

PROGRAMME BOOK

epkinly®
epcoritamab

EPKINLY® CONTINUES TO ELICIT DEEP AND DURABLE RESPONSE IN 3L/3L+ DLBCL*[1]

At 25.1-mo median follow-up of the EPCORE NHL-1 trial[1]:

ORR

61.9%

(95% CI, 53.3–70.0)[1]

Estimated complete responders remained in CR at 24mo

61.8%

(95% CI, 44.9–74.9)[1]

DOCR†

Estimated OS rate in complete responders at 24mo

77.4%

(95% CI, 63.6–86.5)[1]

OS†

Epkinly®-treated DLBCL patients achieved mOS of 19.4 mo (95% CI, 11.7–27.7)[1]

Please scan the QR code for the API

*The pivotal EPCORE NHL-1 trial investigated long-term efficacy and safety results in patients with LBCL (N=157) for up to 2 years, with a median follow-up of 25.1 months.[1] Results for DLBCL (n=139) and DLBCL or HGBCL (n=148) subpopulations were also reported.[1]

†On the basis of the Kaplan-Meier estimate.[1]

Abbreviations: 3L, third line; 3L+, third line or later; CI, confidence interval; CR, complete response; DLBCL, diffuse large B-cell lymphoma; DOCR, duration of complete response; HGBCL, high-grade B-cell lymphoma; LBCL, large B-cell lymphoma; mo, months; mOS, median overall survival; ORR, objective response rate; OS, overall survival

Reference: 1. Thieblemont C, et al. *Leukemia.* 2024;38(12):2653–62.

All adverse events should be reported to PVChina@abbvie.com.
For healthcare professionals only
HK-EDLBCL-250001 17/JAN/2025

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epkinly®
epcoritamab

BLINCYTO®
(blinatumomab)

INTERVENE EARLIER FOR ALL THAT'S AHEAD

FIRST AND ONLY APPROVED IMMUNOTHERAPY FOR MRD+ B-ALL PATIENTS

78% of MRD+ ALL patients achieved a complete MRD response after cycle 1[1,2]

Ph-negative complete MRD responders can achieve **longer Relapse-Free Survival and Overall Survival** than MRD non-responders:

- Median Relapse-Free Survival: 23.6 months (P=0.002)[2]
- Median Overall Survival: 38.9 months (P=0.002)[2]

Generally manageable safety profile[1,2]

ALL=acute lymphoblastic leukemia; B-ALL=B-precursor acute lymphoblastic leukemia; MRD=minimal residual disease.

References: 1. BLINCYTO®(blinatumomab) Full Prescribing Information. January 2023. **2.** Gökbuget N, et al. Blood 2018;131(14):1522-1531.

BLINCYTO® (blinatumomab) Abbreviated Prescribing Information
BLINCYTO® powder for concentrate and solution for solution for infusion 35 mcg/vial

INDICATIONS BLINCYTO is indicated as monotherapy for the treatment of adults with CD19 positive relapsed or refractory B-precursor acute lymphoblastic leukaemia (ALL). Patients with Philadelphia chromosome positive B-precursor ALL should have failed treatment with at least 2 tyrosine kinase inhibitors (TKIs)and have no alternative treatment options. BLINCYTO is indicated as monotherapy for the treatment of adults with Philadelphia chromosome negative CD19 positive B-precursor ALL in first or second complete remission with minimal residualdisease (MRD) greater than or equal to 0.1%. BLINCYTO is indicated as monotherapy for the treatment of paediatric patients aged 1 year or older withPhiladelphia chromosome negative CD19 positive B-precursor ALL which is refractory or in relapse afterreceiving at least two prior therapies or in relapse after receiving prior allogeneic haematopoietic stem cell transplantation. BLINCYTO is indicated as monotherapy for the treatment of paediatric patients aged 1 year or older withhigh-risk first relapsed Philadelphia chromosome negative CD19 positive B-precursor ALL as part of the consolidation therapy. **DOSAGE AND ADMINISTRATION** For the treatment of relapsed or refractory B-precursor ALL, hospitalisation is recommended for initiationat a minimum for the first 9 days of the first cycle and the first 2 days of the second cycle. For the treatment of Philadelphia chromosome negative MRD positive B-precursor ALL, hospitalisation is recommended at a minimum for the first 3 days of the first cycle and the first 2 days of subsequent cycles.For paediatric patients with high-risk first relapsed B-precursor ALL, hospitalisation is recommended at aminimum for the first 3 days of the cycle. In patients with a history or presence of clinically relevant central nervous system (CNS) pathology, hospitalisation is recommended at a minimum for the first 14 days of the first cycle. In the second cycle, hospitalisation is recommended at a minimum for 2 days,and clinical judgment should be based on tolerance to BLINCYTO in the first cycle. BLINCYTO infusion bags should be prepared to infuse over 24 hours, 48 hours, 72 hours, or 96 hours. See Prescribing Information for details. Posology: *Relapsed or refractory B-precursor ALL* Patients with relapsed or refractory B-precursor ALL, may receive 2 cycles of treatment. A single cycle oftreatment is 28 days (4 weeks) of continuous infusion. Each cycle of treatment is separated by a 14 day (2 week) treatment-free interval. Patients who have achieved complete remission (CR/CRh*) after 2 treatment cycles may receive up to 3 additional cycles of BLINCYTO consolidation treatment, based onan individual benefits-risks assessment. Recommended daily dose is by patient weight. Patients greater than or equal to 45 kg receive a fixed-dose and for patients less than 45 kg, the dose is calculated using the patient's body surface area (BSA). See Prescribing Information for details. *High-risk first relapsed B-precursor ALL* Paediatric patients with high-risk first relapsed B-precursor ALL may receive 1 cycle of BLINCYTOtreatment after induction and 2 blocks of consolidation chemotherapy. A single cycle of treatment is 28 days (4 weeks) of continuous infusion. See Prescribing Information for the recommended daily doseby patient weight for paediatric patients. *Premedication and additional medication recommendations:* In adult patients, dexamethasone 20 mg intravenous should be administered 1 hour prior to initiation of each cycle of BLINCYTO therapy. In paediatric patients, dexamethasone 10 mg/m^2 (not to exceed 20 mg) should be administered orally or intravenously 6 to 12 hours prior to the start of BLINCYTO (cycle 1, day 1). This should be followed by dexamethasone 5 mg/m^2 orally or intravenously within 30 minutes prior to the start of BLINCYTO (cycle1, day 1). *MRD positive B-precursor ALL* When considering the use of BLINCYTO as a treatment for Philadelphia chromosome negative MRD positive B-precursor ALL, quantifiable MRD should be confirmed in a validated assay withminimum sensitivity of 10^{-4}. Patients may receive 1 cycle of induction treatment followed by up to 3 additional cycles of BLINCYTO consolidation treatment. A single cycle of treatment of BLINCYTO induction or consolidation is 28 days (4 weeks) of continuous intravenous infusion followed by a 14 day(2 week) treatment-free interval (total 42 days). *Premedication and additional medication recommendations:* Prednisone 100 mg intravenously or equivalent (e.g. dexamethasone 16 mg) should be administered 1 hour prior to initiation of each cycle ofBLINCYTO therapy. Anti-pyretic use (e.g. paracetamol) is recommended to reduce pyrexia during the first 48 hours of eachtreatment cycle. Intrathecal chemotherapy prophylaxis is recommended before and during BLINCYTOtherapy to prevent central nervous system ALL relapse. *Dosage Adjustments*: If the interruption of treatment after an adverse event is no longer than 7 days, continue the same cycle toa total of 28 days of infusion inclusive of days before and after the interruption in that cycle. If an interruption due to an adverse event is longer than 7 days, start a new cycle. If the toxicity takes more than 14 days to resolve, discontinue BLINCYTO permanently, except if described differently in the Prescribing Information. *Method of administration:* Administer BLINCYTO as a continuous intravenous infusion delivered at a constant flow rate using an infusion pump over a period of up to 96 hours. The pump should be programmable, non- elastomeric, and have an alarm. The starting volume (270 mL) is more than the volume administered to the patient (240 mL) to account for the priming of the intravenous tubing and to ensure that the patient will receive the full dose of BLINCYTO. *Infuse prepared BLINCYTO final infusion solution according to the Instructions on the pharmacy label on the prepared bag at one of the following constant infusion rates:* Infusion rate of 10 mL/h for a duration of 24 hours, OR Infusion rate of 5 mL/hfor a duration of 48 hours, OR Infusion rate of 3.3 mL/h for a duration of 72 hours, OR Infusion rate of 2.5 mL/h for a duration of 96 hours. Administer prepared BLINCYTO final infusion solution using intravenous tubing that contains a sterile, non-pyrogenic, low protein-binding 0.2 micrometre in-line filter. Important note: Do not flush the BLINCYTO infusion line or intravenous catheter, especially whenchanging infusion bags. Flushing when changing bags or at completion of infusion can result in excess dosage and complications thereof. When administering via a multi-lumen venous catheter, BLINCYTO should be infused through a dedicated lumen. The choice of the infusion duration should be made by the treating physician considering the frequency of the infusion bag changes and the weight of the patient. The target therapeutic dose of BLINCYTO delivered does not change. The infusion bag must be changedat least every 96 hours by a healthcare professional for sterility reasons. See Prescribing Information for reconstitution of BLINCYTO and preparation of BLINCYTO Infusion Bag. **CONTRAINDICATIONS** Hypersensitivity to the active substance or to any of the excipients. Breast-feeding. **SPECIAL WARNINGS AND PRECAUTIONS** Neurologic events: Neurologic events including events with a fatal outcome have been observed. Grade 3 or higher (severe or life-threatening) neurologic events following initiation of blinatumomab administration included encephalopathy, seizures, speech disorders, disturbances in consciousness, confusion and disorientation, and coordination and balance disorders. Infections: In patients receiving blinatumomab, serious infections, including sepsis, pneumonia, bacteraemia, opportunistic infections and catheter site infections have been observed, some of which were life-threatening or fatal. Cytokine releasesyndrome and infusion reactions: Cytokine release syndrome (CRS) which may be life-threatening or fatal has been reported in patients receiving BLINCYTO. Serious adverse events that may be signs and symptoms of CRS included pyrexia, asthenia, headache, hypotension, total bilirubin increased, and nausea; uncommonly, these events led to BLINCYTO discontinuation. Disseminated intravascular coagulation (DIC) and capillary leak syndrome have been commonly associated with CRS. Haemophagocytic histiocytosis/macrophage activation syndrome (MAS) has been uncommonly reported in the setting of CRS. Tumour lysis syndrome: Tumour lysis syndrome (TLS), which may be life- threatening or fatal has been observed in patients receiving BLINCYTO. Appropriate prophylactic measures including aggressive hydration and anti-hyperuricaemic therapy should be used for the prevention and treatment of TLS during BLINCYTO treatment, especially in patients with higher leukocytosis or a high tumour burden. Neutropenia and febrile neutropenia: Neutropenia and febrile neutropenia, including life-threatening cases, have been observed in patients receiving BLINCYTO. Elevated liver enzymes: Treatment with BLINCYTO was associated with transient elevations in liver enzymes. Pancreatitis: Pancreatitis, life-threatening or fatal, has been reported in patients receiving BLINCYTO in clinical trials and the post-marketing setting. High-dose steroid therapy may have contributed, in some cases, to the pancreatitis. Leukoencephalopathy including progressive multifocal leukoencephalopathy: Cranial magnetic resonance imaging (MRI) changes showing leukoencephalopathyhave been observed in patients receiving BLINCYTO, especially in patients with prior treatment with cranial irradiation and anti-leukaemic chemotherapy. Due to the potential for progressive multifocal leukoencephalopathy (PML), patients should be monitored for signs and symptoms. CD19-negative relapse: CD19-negative B-precursor ALL has been reported in relapsed patients receiving BLINCYTO. Particular attention should be given to assessment of CD19 expression at the time of bone marrow testing.Lineage switch from ALL to acute myeloid leukaemia (AML): Lineage switch from ALL to AML has been rarely reported in relapsed patients receiving BLINCYTO, including those with no immunophenotypic and/or cytogenetic abnormalities at initial diagnosis. All relapsed patients should be monitored for presence of AML. Immunisations: Vaccination with live virus vaccines is not recommended for at least 2 weeks prior to the start of BLINCYTO treatment, during treatment, and until recovery of B-lymphocytes to normal ranges following last treatment cycle. Due to the potential depletionof B-cells in newborns following exposure to blinatumomab during pregnancy, newborns should be monitored for B-cell depletion and vaccinations with live virus vaccines should be postponed until the infant's B-cell count has recovered. Contraception: Women of childbearing potential have to use effectivecontraception during and for at least 48 hours, after treatment with BLINCYTO. Medication errors: Medication errors have been observed with BLINCYTO treatment. It is very important that the instructions for preparation (including reconstitution and dilution) and administration are strictly followed to minimise medication errors (including underdose and overdose). **INTERACTIONS** No formal drug interaction studies have been performed. Results from an *in vitro* test in human hepatocytes suggest that blinatumomab did not affect CYP450 enzyme activities. Initiation of BLINCYTO treatment causes transient release of cytokines during the first days of treatment that maysuppress CYP450 enzymes. Patients who are receiving medicinal products that are CYP450 and transporter substrates with a narrow therapeutic index should be monitored for adverse effects (e.g. warfarin) or drug concentrations (e.g. cyclosporine) during this time. The dose of the concomitant medicinal product should be adjusted as needed. **FERTILITY, PREGNANCY AND LACTATION** Pregnancy: There are no data from the use of blinatumomab in pregnant women. Blinatumomab shouldnot be used during pregnancy unless the potential benefit outweighs the potential risk to the foetus. Women of childbearing potential have to use effective contraception during and for at least 48 hours aftertreatment with blinatumomab. Breast-feeding: Breast-feeding is contraindicated during and for at least 48 hours after treatment with blinatumomab. Fertility: No studies have been conducted to evaluate theeffects of blinatumomab on fertility. **EFFECTS ON ABILITY TO DRIVE AND USE MACHINES** Blinatumomab has major influence on the ability to drive and use machines. Confusion and disorientation, coordination and balance disorders, risk of seizures and disturbances in consciousness canoccur. Due to the potential for neurologic events, patients receiving blinatumomab should refrain from driving, engaging in hazardous occupations or activities such as driving or operating heavy or potentiallydangerous machinery while blinatumomab is being administered. Patients must be advised that they mayexperience neurologic events. **UNDESIRABLE EFFECTS** The most serious adverse reactions that may occur during blinatumomab treatment include: infections (22.6%), neurologic events (12.2%), neutropenia/febrile neutropenia (9.1%), cytokine release syndrome (2.7%), and tumour lysis syndrome (0.8%). The most common adverse reactions were: pyrexia (70.8%), infections – pathogen unspecified (41.4%), infusion-related reactions (33.4%), headache (32.7%), nausea(23.9%), anaemia (23.3%), thrombocytopenia (21.6%), oedema (21.4%), neutropenia (20.8%), febrile neutropenia (20.4%), diarrhoea (19.7%), vomiting (19.0%), rash (18.0%), hepatic enzyme increased (17.2%), cough (15.0%), bacterial infectious disorders (14.1%), tremor (14.1%), cytokine release syndrome (13.8%), leukopenia (13.8%), constipation (13.5%), decreased immunoglobulins (13.4%), viral infectious disorders (13.3%), hypotension (13.0%), back pain (12.5%), chills (11.7%), abdominal pain (10.6%), tachycardia (10.6%), insomnia (10.4%), pain in extremity (10.1%), and fungal infectious disorders (9.6%). Paediatric population: The most frequently reported serious adverse events were pyrexia (11.4%), febrileneutropenia (11.4%), cytokine release syndrome (5.7%), sepsis (4.3%), device-related infection (4.3%), overdose (4.3%), convulsion (2.9%), respiratory failure (2.9%), hypoxia (2.9%), pneumonia (2.9%), andmulti-organ failure (2.9%). The adverse reactions in BLINCYTO-treated paediatric patients were similarin type to those seen in adult patients. Adverse reactions that were observed more frequently (≥ 10% difference) in the paediatric population were anaemia, thrombocytopenia, leukopenia, pyrexia, infusion-related reactions, weight increase, and hypertension. Other special populations: Elderly patients with MRD positive ALL treated with BLINCYTO may havean increased risk of hypogammaglobulinaemia compared to younger patients. It is recommended that immunoglobulin levels are monitored in elderly patients during treatment with BLINCYTO. Immunogenicity: In clinical studies of adult ALL patients treated with BLINCYTO, less than 3% testedpositive for anti-blinatumomab antibodies.

Please read the full prescribing information prior to administration and full prescribing information is available upon request. BLINCYTO® is a registered trademark owned or licensed byAmgen Inc., its subsidiaries, or affiliates. Abbreviated Prescribing Information Version: HKBLIPI02

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HKG-103-0524-80001 Mar 2025

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CSL Behring

Multiple Myeloma Remission doesn't stop infections[1], secondary immunodeficiency still puts outcomes at risk.[1]

Go the extra mile

IgRT was associated with a ten-fold reduction in grade 3–5 infections in MM patients treated with anti-BCMA bispecific antibodies, with an incidence rate ratio of 0.10 (95% CI: 0.01–0.81; p = 0.0307), indicating a 90% reduction in serious infection risk during periods on IVIg versus off.[1]

- 90%

Disclaimer Individual shown is a model.
Image used for illustration only.

Before prescribing, please review the approved Hong Kong Package Insert, November 2021

Privigen Human normal immunoglobulin solution for infusion (10%)

Indication: Replacement therapy in adults, and children and adolescents (0-18 years) in: • Primary immunodeficiency syndromes (PID) with impaired antibody production; • Secondary immunodeficiencies (SID) in patients who suffer from severe or recurrent infections, ineffective antimicrobial treatment and either proven specific antibody failure (PSAF)* or serum IgG level of <4 g/l. Immunomodulation in adults, and children and adolescents (0-18 years) in: • Primary immune thrombocytopenia (ITP), in patients at high risk of bleeding or prior to surgery to correct the platelet count; •Guillain-Barré syndrome; • Kawasaki disease(in conjunction with acetylsalicylic acid); • Chronic inflammatory demyelinating polyneuropathy (CIDP). Only limited experience is available of use of intravenous immunoglobulins in children with CIDP; • Multifocal motor neuropathy (MMN). ** PSAF = failure to mount at least a 2-fold rise in IgG antibody titre to pneumococcal polysaccharide and polypeptide antigen vaccines.* **Dosage: In replacement therapy the dose may need to be individualised for each patient depending on the clinical response.** *Replacement therapy in primary immunodeficiency (PID) syndromes.* The recommended starting dose is 0.4 to 0.8 g/kg body weight (bw) given once, followed by at least 0.2 g/kg bw every 3 to 4 weeks. *Secondary immunodeficiencies* The recommended dose is 0.2 – 0.4g/kg bw every three to four weeks. *Primary immune thrombocytopenia (ITP)* • 0.8 to 1g/kg bw given on day 1; this dose may be repeated once within 3 days, OR • 0.4 g/kg bw given daily for 2 to 5 days. *Guillain-Barré syndrome* 0.4 g/kg bw/day over 5 days. Kawasaki disease 2.0 g/kg bw should be administered as a single dose. Patients should receive concomitant treatment with acetylsalicylic acid. *Chronic inflammatory demyelinating polyneuropathy (CIDP)* The recommended starting dose is 2 g/kg bw divided over 2 to 5 consecutive days followed by maintenance doses of 1 g/kg bw over 1 to 2 consecutive days every 3 weeks. *Multifocal Motor Neuropathy (MMN)* Starting dose: 2 g/kg given over 2-5 consecutive days. Maintenance dose: 1 g/kg every 2 to 4 weeks or 2 g/kg every 4 to 8 weeks. Method of administration: For intravenous use. Privigen should be infused intravenously at an initial infusion rate of 0.3 ml/kg bw/hr for approximately 30 min. If well tolerated, the rate of administration may gradually be increased to 4.8 ml/kg bw/hr. In PID patients who have tolerated the infusion rate of 4.8 ml/kg bw/hr well, the rate may be further gradually increased to a maximum of 7.2 ml/kg bw/hr. Contraindications: Hypersensitivity. Patients with selective IgA deficiency who developed antibodies to IgA. Patients with hyperprolinaemia type I or II. Precautions: Not indicated in patients with selective IgA deficiency where the IgA deficiency is the only abnormality of concern. Caution for hypersensitivity, haemolytic anaemia, aseptic meningitis syndrome, thromboembolism, acute renal failure, pulmonary adverse reactions, interference with serological testing, possibility of transmissible agents. In case of adverse reaction, IVIg products should be administered at the minimum rate of infusion and dose practicable. Privigen does not contain sucrose, maltose or glucose. Privigen contains less than 2.3 mg sodium per 100 ml. Undesirable effects: Headache, pain, pyrexia, influenza like illness, anaemia, haemolysis β, leukopenia, hypersensitivity, dizziness, hypertension, flushing, hypotension, dyspnoea, nausea, vomiting, diarrhoea, abdominal pain, hyperbilirubinaemia, skin disorder, myalgia, fatigue, asthenia, decreased haemoglobin, Coombs' (direct) test positive, increased alanine aminotransferase, increased aspartate aminotransferase, increased blood lactate dehydrogenase. Date of last revision of PI: Nov 2021

STAY AHEAD OF SID

CSL Behring

Abbreviation Key
IgRT: Immunoglobulin Replacement Therapy; IVIg: Intravenous Immunoglobulin; BCMA: B-Cell Maturation Antigen; MM: Multiple Myeloma; CI: Confidence Interval; OR: Odds Ratio.

Learn More
1.Lancman et al., 2023

CSL Behring Asia Pacific Limited
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HKG-HIZ-0010
Date of production: Nov 2025

WELCOME MESSAGE

Dr. Vivien MAK Wai Man
Chairperson
The Hong Kong Society of Haemaotlogy

Dear Members and colleagues,

On behalf of the Organizing Committee, it is my great pleasure to welcome you to the 52nd Annual Scientific Meeting (ASM) of The Hong Kong Society of Haematology (HKSH). As we enter a transformative era in blood sciences, driven by the convergence of digital technology, molecular biology, and big data analytics, our meeting serves as a vital platform for clinicians, researchers, and healthcare professionals to exchange insights on the therapies reshaping our field.

Our program covers the broad spectrum of haematology, from therapies on malignant haematology to life-changing developments in benign conditions. We are honoured to have distinguished international speakers share the updated information on the management of haematological diseases and their experiences with us. For the presidential symposium, we are privileged to have Prof. HUANG He of the First Affiliated Hospital, Zhejiang University School of Medicine, to speak to us on the rapidly evolving landscape of CAR-T Therapy in China.

Similar to the previous Annual Scientific Meetings, we are proud to feature two other essential components in the meeting: Young Fellow & Best Abstract Presentations, a dedicated platform for our emerging specialists to showcase their research, and the Nursing Symposium, recognizing the indispensable role of specialized nursing in patient outcomes.

This meeting is more than just a series of lectures; it is an opportunity to reconnect with colleagues, foster new collaborations, and discuss how these international advancements can be integrated into our local practice. We thank you for your continued dedication to the field and look forward to an inspiring and fruitful meeting.

MonoFer®
iron isomaltoside 1000

Moving with the times

Take a step forward in treating iron deficiency*

*Innovative matrix technology to restore iron levels and treat fatigue with a robust safety profile[1-3]

ROUTINE PHOSPHATE MONITORING NOT REQUIRED[1^]

Restore iron need from ONE visit[1]

With Monofer®, your patients need ONE IV infusion when their required iron is up to 20 mg/kg body weight[1,2,4,5]

Rapidly improves fatigue[†6]

In the PHOSPHARE-IBD[#] study, Monofer® was associated with a greater improvement in patient-reported fatigue score at Day 35, compared with ferric carboxymaltose (p=0.005)[6]

Robust safety profile[1,3]

Well tolerated across patient groups with an uncommon incidence of hypophosphataemia (0.1% to 1% of patients)[1,3]

[†]In the PHOSPHARE-IBD[#] study, Monofer® was associated with greater improvement in patient-reported fatigue scores at Days 35 and 49, compared with ferric carboxymaltose (p=0.005 and 0.009 respectively).[6]

[#]A multicentre, randomised, head-to-head comparison of IV Monofer® and ferric carboxymaltose in patients with both IBD and IDA. The primary endpoint for this study was the incidence of hypophosphataemia (serum phosphate < 2.0 mg/dL) at any time, Day 0-35 (SAS). Change in FACIT Fatigue Scale score (prespecified exploratory outcome; ITT) was a secondary endpoint in this study. A greater improvement in fatigue scores was observed among Monofer®-treated patients compared to ferric carboxymaltose-treated patients at Day 35 (p=0.005) and Day 49 (p=0.009), despite the difference not being significant at Day 70.[6]

[^]Hypophosphatemia is an uncommon adverse drug reaction observed during clinical trials and post-marketing experience.

Abbreviations: IV, intravenous; IBD, inflammatory bowel disease; IDA, iron deficiency anaemia; FACIT, Functional Assessment of Chronic Illness Therapy; ITT, intention to treat; SAS, statistical analysis system.

Monofer® 100 mg/ml solution for injection/infusion
Composition: 1 ml contains 100 mg iron as iron(III) isomaltoside 1000. Available in vials/ampoules of 1 ml and 5 ml. **Indications:** Monofer® is indicated for the treatment of iron deficiency in the following conditions: (1) Oral iron preparations are ineffective or cannot be used. (2) There is a clinical need to deliver iron rapidly. The diagnosis must be based on laboratory tests. **Dosage and Administration:** Monitor patients for hypersensitivity reactions during and following each administration. Administer only when staff trained to evaluate and manage anaphylactic reactions is available. Calculate iron need using the Simplified Table or Ganzoni formula. Do not exceed 20 mg iron/kg body weight per week. Not recommended for children and adolescents <18 years. **IV bolus injection:** up to 500 mg up to three times a week, administered undiluted or diluted in max. 20 ml sterile 0.9% NaCl at a rate of up to 250 mg iron/min. **IV infusion:** up to 20 mg iron/kg body weight, administered undiluted or diluted in max 500 ml sterile 0.9% NaCl (to no less than 1 mg iron/ml). **Contraindications:** Hypersensitivity to the active substance or any excipients. Known serious hypersensitivity to other parenteral iron products. Non-iron deficiency anaemia. Iron overload or disturbances in iron utilisation. Decompensated liver disease. **Precautions:** Hypersensitivity reactions, including serious and potentially fatal anaphylactic/anaphylactoid reactions. Increased risk in patients with known allergies including drug allergies, including patients with a history of severe asthma, eczema or other atopic allergy, and patients with immune inflammatory conditions. Each patient should be observed for adverse effects for at least 30 minutes following each Monofer injection. Avoid in patients with hepatic dysfunction where iron overload is a precipitating factor and patients with ongoing bacteraemia. Absorption of oral iron is reduced when administered concomitantly. **Fertility, Pregnancy, and Lactation:** Pregnancy: Limited data from use in pregnant women. Treatment should be confined to second and third trimester if the benefit outweighs the potential risk for both the mother and the foetus. Breast-feeding: At therapeutic doses, no effects on the breastfeed newborns/infants are anticipated. Fertility: No data on human fertility. **Effects on ability to drive and use machines:** No studies on have been performed. **Undesirable Effects:** Acute severe hypersensitivity reactions may occur. In pregnancy, associated foetal bradycardia may occur. Distant skin discolouration has been reported. Common ADRs (≥1/100 to <1/10): Nausea; Rash; Injection site reactions. Uncommon ADRs (≥1/1000 to <1/100): Hypersensitivity. Headache, paraesthesia, dysgeusia, blurred vision, loss of consciousness, dizziness, fatigue; Tachycardia; Hypotension; hypertension; Chest pain, dyspnea, bronchospasm. Abdominal pain, vomiting, dyspepsia, constipation, diarrhoea; Pruritus, urticaria, flushing, sweating, dermatitis; Hypophosphataemia; Back pain, myalgia, arthralgia, muscle spasms; Pyrexia, chills/shivering, infection, local phlebitis reaction, skin exfoliation; Hepatic enzyme increase. For further information consult full prescribing information. [July 2022]

References. 1. Monofer® Summary of Product Characteristics, Hong Kong, 13.07.2022. **2.** Kalra PA, et al. Port J Nephrol Hypert 2012; 26: 13–24. **3.** Kassianides X, et al. Expert Rev Hematol. 2021 Jan;14(1):7-29. **4.** Kalra PA, Bhandari. Int J Nephrol Renovasc Dis 2016; 9: 53–64. **5.** Jahn MR, et al. Eur J Pharm Biopharm 2011; 78: 480–91. **6.** Zoller H, et al. Gut 2022; 0: 1–10 (doi:10.1136/gutjnl-2022-327897) – including supplementary material.

For healthcare professionals only.

MENARINI

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HK-MNF-032025-055 (Mar 2025)

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INTRODUCING

BESREMi™
(ropeginterferon alfa-2b)
INJECTION

FIRST MEDICATION,
FIRST INTERFERON APPROVED and
PREFERRED FIRST LINE THERAPY
for Adult Polycythemia Vera
regardless of previous
treatment history.

References : 1. U.S. Food and Drug Administration/Press Announcements. (2021) FDA Approves Treatment for Rare Blood Disease 2. NCCN Clinical Practice Guidelines in Oncology (NCCN Guidelines®) for Myeloproliferative Neoplasms V.1.2025. © National Comprehensive Cancer network, Inc., All rights reserved. 3. BESREMi Package insert HK-68864, PharmaEssentia Asia (Hong Kong) Limited.

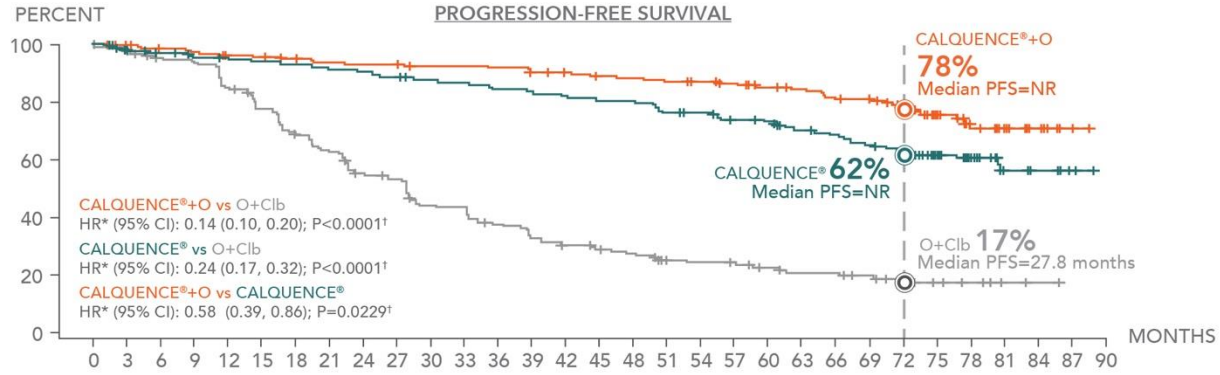
PharmaEssentia Asia (Hong Kong) Limited.
Contact info: Unit 2316, Level 23, Prosperity Tower, No. 39 Queen's Road, Central, Hong Kong
https://hq.pharmaessentia.com/tw

PEC-BES-HK-2601003

MEETING INFORMATION

Venue	Meeting Rooms S221 – S230, 2/F, Hong Kong Convention and Exhibition Centre
Date & Time	14 March 2026 (Saturday) 14:00 – 19:30
Event Secretariat	Event Genie Limited Email: support@eventgenie.com.hk Tel: +852 5930 0700
Entitlement	All participants will be entitled: ▪ Access to all scientific sessions ▪ e-Programme book ▪ Certificate of attendance (subject to certain criteria requested by the respective Colleges)

Superior PFS to Obinutuzumab + Chlorambucil[1]



Consistent Superiority Regardless of del(17p) / TP53m[1]

	CALQUENCE® Regimens vs O + Clb CLL with del(17p)/TP53m		
	PFS at 6 years	HR‡ (95%, CI)	Median PFS (months)
CALQUENCE® + O	56%	0.28 (0.13, 0.59); P=0.0009#	73.1
CALQUENCE®	56%	0.23 (0.10, 0.52); P=0.0002#	NR
O + Clb	18%	-	17.5

Proven Safety Profile[1]

- ELEVATE-TN 6 year follow-up: most common any-grade AEs and grade ≥3 AEs were consistent with earlier analyses.§
- ELEVATE-TN 5 year follow-up: discontinuation rates due to AEs were similar (CALQUENCE® + O, 17% [n=179]; CALQUENCE®, 16% [n=179]; O + Clb, 14% [n=177]).[5]

*HR based on stratified Cox proportional-hazards model. †P-value based on stratified log-rank test. ‡HR based on unstratified Cox proportional-hazards model. #P-value based on unstratified log-rank test. §Most common any-grade AEs and grade ≥3 AEs include: diarrhea, headache, arthralgia, neutropenia, fatigue, cough, COVID-19, thrombocytopenia, pneumonia, hypertension, syncope.

Abbreviations: AE, adverse event; CI, confidence interval; Clb, chlorambucil, CLL, chronic lymphocytic leukemia; del(17p), deletion of the short arm of chromosome 17; ESMO, European society for medical oncology; HR, hazard ratio; iwCLL, international workshop on chronic lymphocytic leukemia; mo, months; NR, not reached; O, obinutuzumab; PFS, progression-free survival; TP53m, TP53 gene mutations.

Reference: 1. Sharman J, et al. Acalabrutinib ± Obinutuzumab vs Obinutuzumab + Chlorambucil in Treatment-naive Chronic Lymphocytic Leukemia: 6-Year Follow-up of ELEVATE-TN. Presented at: American Society of Hematology (ASH) Annual Meeting and Exposition 2023; 9-12 December 2023; San Diego, California. Abs 636. **2.** Eichhorst B, et al. Ann Oncol. 2021 Jan;32(1):23-33. **3.** Hallek M, et al. Am J Hematol. 2021 Dec 1;96(12):1679-1705. **4.** Weirda WG, et al. Chronic Lymphocytic Leukemia/ Small Lymphocytic Lymphoma. NCCN Clinical Practice Guidelines in Oncology (NCCN Guidelines®). Version 1. 2024. Available at: NCCN.org. Accessed 31 Jan 2024. **5.** Sharman JP. Egyed M. Jurczak W, et al. Acalabrutinib ± obinutuzumab vs obinutuzumab + chlorambucil in treatment-naive chronic lymphocytic leukemia: 5-year treatment-naive chronic lymphocytic leukaemia (ELEVATE-TN); a randomised controlled phase 3 trial published correction appears in follow-up of ELEVATE-TN. Poster presented at: American Society of Clinical Oncology (ASCO) Annual Meeting; June 3-7, 2022. Abs 7539.

Presentation: Acalabrutinib capsule 100 mg. Indications: For the treatment of adult patients with: • Mantle cell lymphoma (MCL) who have received at least one prior therapy • Chronic lymphocytic leukemia (CLL). Dosage: 100 mg orally approximately every 12 hours, swallow whole with water and with or without food. Contraindications: None. Precautions: Consider prophylaxis in patients who are at increased risk for opportunistic infections. Monitor patients for signs and symptoms of infection and treat promptly; Monitor patients for signs of bleeding. Consider the benefit-risk of withholding for 3-7 days pre- and post-surgery depending upon the type of surgery and the risk of bleeding; Monitor complete blood counts regularly; Other malignancies have occurred, including skin cancers and other solid tumors. Advise patients to use sun protection; Monitor for symptoms of arrhythmias and manage; Avoid in patients with severe hepatic impairment; May cause fetal harm and dystocia in pregnancy; Advise not to breastfeed while taking and for at least 2 weeks after the final dose. Interactions: Avoid co-administration with strong CYP3A inhibitors, strong CYP3A inducers and proton pump inhibitors. Dose adjustments may be recommended; Stagger dosing with H2-receptor antagonists and antacids. Undesirable effects: Anemia, neutropenia, upper respiratory tract infection, thrombocytopenia, headache, diarrhea, and musculoskeletal pain. Full local prescribing information is available upon request. API.HK.CAL.0620

For Healthcare Professionals Use Only

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AstraZeneca Hong Kong Limited
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Please refer to the full Prescribing Information for further information (including information on warnings and precautions, special populations, hepatic and renal impairment, fertility, pregnancy and lactation) prior to prescribing. Please visit contactazmedical.astrazeneca.com, for (1) enquiring Medical Information (MI), (2) reporting Individual Case Safety Report (ICSR) and/or (3) reporting Product Quality Complaint (PQC) to AstraZeneca Hong Kong Limited.

HK-11026 Nov 2024

MAIN ROOM PROGRAMME

14:00 – 14:05	Opening Remarks *Dr. Vivien MAK Wai Man*
	Chairpersons: Dr. Vivien MAK Wai Man, Dr. LAW Man Fai
14:05 – 14:35	**Raising the Bar for Relapsed/Refractory Follicular Lymphoma: How Can We Go Further?** *Dr. Laurie SEHN (Canada)*
14:40 – 15:10	**Entering A New Era in the Management of Acute Lymphoblastic Leukaemia** *Dr. NG Chin Hin (Singapore)*
15:15 – 15:55	**Presidential Symposium** **Novel CAR-T Cellular Therapy for Hematological Malignancies and Autoimmune Diseases** *Prof. HUANG He (China)*
15:55 – 16:00	Outstanding New Haematology Fellow Award Presentation
16:00 - 16:05	Group Photo
16:05 – 16:35	Break Time (Posters & Exhibits)
	Chairpersons: Dr. LAW Man Fai, Dr. Samantha LUK Yan Yan, Dr. Saliangi WU
16:35 – 17:05	**Sequencing Myeloma Therapy Across the Continuum: 2nd Line to Late Line Perspectives** *Prof. Niels VAN DE DONK (The Netherlands)*
17:10 – 17:40	**Modern Management of Polycythemia Vera –** **Ropeginterferon Alfa-2b Demonstrates Long-Term Disease Improvements** *Prof. Kazuya SHIMODA (Japan)*
17:45 – 18:15	**The Evidence-based Management of CML: An Asian Perspective** *Dr. Takeshi KONDO (Japan)*
18:15 – 18:30	Break Time (Posters & Exhibits)
	Young Fellow and Best Abstract Presentation *Chairpersons: Dr. Vivien MAK Wai Man, Dr. Rosalina IP Ka Ling*
18:30 – 19:30	**Phenotypic and Genotypic Features of von Willebrand Disease in Hong Kong** *Dr. Mathew CHEUNG Tsz Long*
	Real-World Outcome of Eltrombopag-Containing Regimens as Frontline Treatment for Aplastic Anaemia: A Multi-Center Retrospective Study in Hong Kong *Dr. Stephen LAM Sze Yuen*
	Evaluation of Oxford Nanopore Sequencing in Rapid AML Genomic Profiling *Dr. Harry LAU Ka Ngai*
	Clinical Outcome of 4-factor Prothrombin Complex Concentrate (4-F PCC) for Factor Xa-inhibitor Reversal: A Multi-centre Retrospective Review *Dr. Lala SIN Yuen Ting*
	Mycophenolate Mofetil in Chinese Patients with Immune Thrombocytopenia *Dr. Jessica WONG G Kei*
	Best Abstract Presentation **Donor Selection and Clinical Outcomes of Allogeneic Haematopoietic Stem Cell Transplantation in Adults Older than 50 Years** *Dr. Garret LEUNG*
19:30 – 19:35	Closing Remarks *Dr. Vivien MAK Wai Man*
20:00 – 21:30	Dinner *(For HKSH members, HKHNA members and invited guests only)*

CONCURRENT ROOM PROGRAMME

	Chairpersons: Dr. Gloria HWANG Yu Yan, Dr. HA Chung Yin, Dr. Rosalina IP Ka Ling
16:35 – 17:05	**From Trial to Transformation: Five-Year Outcomes of HOPE-B Gene Therapy in Haemophilia B** *Prof. Michiel COPPENS (The Netherlands)*
17:10 – 17:40	**Latest Advances in the Management of Complement-Mediated Disease** *Dr. Talha MUNIR (United Kingdom)*
17:45 – 18:15	**The Ongoing Debate on IV Iron in Iron Deficiency Anaemia** *Dr. James UPRICHARD (United Kingdom)*
18:15 – 18:30	Break Time (Posters & Exhibits)
	Nursing Symposium *Chairpersons: Mr. Simon LEUNG, Mr. LIU Tak Kei* *Associated Organisation: Hong Kong Haematology Nursing Association*
18:30 – 19:30	**When CAR-T Gets Complicated: Nursing Care That Changes Patient Outcomes** *Ms. Katrina DEBOSZ (Australia)*

DARZALEX® SC daratumumab subcutaneous

DARZALEX® daratumumab concentrate for solution for infusion

DARZALEX® – OPEN A NEW DIMENSION OF COMBINATION EFFICACY

Newly diagnosed multiple myeloma

Frontline DARZALEX® + VTd for TE patients*[1]

39%

HR=0.61; 95% CI, 0.52–0.72; p<0.0001

Reduction in risk of disease progression or death (vs VTd)

mPFS 83.7 mo

Frontline DARZALEX® + Rd for TIE patients†[2]

45%

HR=0.55; 95% CI, 0.45–0.67; p<0.0001

Reduction in risk of disease progression or death (vs Rd)

mPFS 61.9 mo

Frontline DARZALEX® + VRd[3–5]

For TE patients‡[3]

58%

HR=0.42; 95% CI, 0.30–0.59; p<0.001

For TIE patients§[4]

43%

HR=0.57; 95% CI, 0.41–0.79; p=0.0005

Reduction in risk of disease progression or death (vs VRd)[3,4]

Estimated 84.3% PFS rate at 4.0 yrs (95% CI, 79.5–88.1)[3,5]

Estimated 68.1% PFS rate at 4.5 yrs (95% CI, 60.8–74.3)[4,5]

Relapsed/refractory multiple myeloma

DARZALEX® + Rd for patients ≥1 PLOT¶[6]

56%

HR=0.44; 95% CI, 0.35–0.54; p<0.0001 (vs Rd)

DARZALEX® + Vd for patients with 1 PLOT#[7]

78%

HR=0.22; 95% CI, 0.15–0.32; p<0.0001 (vs Vd)

Reduction in risk of disease progression or death

mPFS 45.0 mo **mPFS 27.0 mo**

DARZALEX® + Pd for patients ≥1 PLOT containing a PI and lenalidomide and were lenalidomide-refractory**[8]

37%

HR=0.63; 95% CI, 0.47–0.85; two-sided p=0.0018

Reduction in risk of disease progression or death (vs Pd)

mPFS 12.4 mo

*CASSIOPEIA is a multicentre, randomised, open-label, phase III trial evaluating the safety and efficacy of DARZALEX® + VTd vs VTd in TE NDMM patients. Patients (N=1,085) were randomised 1:1 to receive either DARZALEX® + VTd or VTd alone.[1]
†MAIA is a multicentre, randomised, open-label, phase III trial evaluating the safety and efficacy of DARZALEX® + Rd vs Rd in TIE NDMM patients. Patients (N=737) were randomised 1:1 to receive either DARZALEX® + Rd or Rd alone.[2]
‡PERSEUS is a multicentre, randomised, phase III trial evaluating DARZALEX® + VRd vs VRd in TE NDMM patients. Patients (N=709) were randomised 1:1 to receive DARZALEX® + VRd before and after transplantation, followed by lenalidomide maintenance therapy, or VRd alone before and after transplantation, followed by lenalidomide maintenance therapy.[3]
§CEPHEUS is a randomised phase III trial evaluating DARZALEX® + VRd vs VRd in patients with NDMM who were TIE or for whom transplant was not planned as the initial therapy (transplant deferred). Patients (N=395) were randomised 1:1 to receive eight cycles of DARZALEX® + VRd or VRd followed by DARZALEX® + Rd or Rd until progression.[4]
¶mPFS not reached for DARZALEX® + VRd.[5]
#POLLUX was a multicentre, randomised, open-label, phase III trial in RRMM patients with ≥1 PLOT. Patients (N=569) were randomised 1:1 to receive either DARZALEX® + Rd or Rd alone.[6]
#CASTOR was a multicentre, randomised, open-label, phase III trial in RRMM patients with ≥1 PLOT. Patients (N=498) were randomised 1:1 to receive DARZALEX® + Vd or Vd alone. Data presented is for the subgroup of patients with 1 PLOT.[7]
**APOLLO is a multicentre, randomised, open-label, phase III trial evaluating the safety and efficacy of DARZALEX® + Pd vs Pd in patients with RRMM who received ≥1 PLOT containing a PI and lenalidomide, had a partial response or better to one or more previous lines of anti-myeloma therapy and were refractory to lenalidomide if only one previous line of therapy was received. Patients (N=304) were randomised 1:1 to receive either DARZALEX® + Pd or Pd alone.[8]

Abbreviations: CI, confidence interval; HR, hazard ratio; mo, months; mPFS, median progression-free survival; NDMM, newly diagnosed multiple myeloma; Pd, pomalidomide+dexamethasone; PFS, progression-free survival; PI, proteasome inhibitor; PLOT, prior line of treatment; Rd, lenalidomide+dexamethasone; RRMM, relapsed/refractory multiple myeloma; TE, transplant-eligible; TIE, transplant-ineligible; Vd, bortezomib+dexamethasone; VRd, bortezomib+lenalidomide+dexamethasone; vs, versus; VTd, bortezomib+thalidomide+dexamethasone; yrs, years.

References: 1. Moreau P, et al. *Lancet Oncol.* 2024;25(8):1003–14. **2.** Facon T, et al. *Leukemia.* 2025;39(4):942–50. **3.** Sonneveld P, et al. *N Engl J Med.* 2024;390(4):301–13. **4.** Usmani SZ, et al. *Nat Med.* 2025;31(4):1195–202. Erratum in: *Nat Med.* 2025;31(4):1366. **5.** Sonneveld P. Presented at the 6th European Myeloma Network (EMN) meeting, 10–12 April 2025; Athens, Greece. **6.** Dimopoulos MA, et al. *J Clin Oncol.* 2023;41(8):1590–99. **7.** Mateos MV, et al. *Clin Lymphoma Myeloma Leuk.* 2020;20(8):509–18. **8.** Dimopoulos MA, et al. *Lancet Oncol.* 2021;22(6):801–12.

Johnson&Johnson

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CP-568766 Mar 2026

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Scan to read **DARZALEX® IV** Abbreviated Prescribing Information

DO YOU EXPECT MORE FOR YOUR PNH PATIENTS?

FABHALTA® iptacopan

A groundbreaking PNH treatment
The first approved oral monotherapy for PNH[1,2]

PRIMARY END POINTS

Significantly more patients achieved Hb improvements in the absence of RBC transfusions with FABHALTA® vs. C5i

Patients with
Normalised§ Hb
of ≥12 g/dL in the absence of RBC transfusions after 24 weeks**

Response rates[3]

FABHALTA® (n=62)	C5i (n=35)
69% (95% CI: 58-79)	2% (95% CI: 1-4)

vs.

(Difference‡: 67%; 95% CI: 56-77; $p<0.001$)[3]

Patients with
Hb increase
of ≥2 g/dL from baseline in the absence of RBC transfusions after 24 weeks**

Response rates[3]

FABHALTA® (n=62)	C5i (n=35)
82% (95% CI: 73-90)	2% (95% CI: 1-4)

vs.

(Difference‡: 80%; 95% CI: 71-88; $p<0.001$)[3]

SECONDARY END POINTS

Over 90% of patients on FABHALTA® avoid transfusions

Patients with
Transfusion independence rate
after 24 weeks†

Response rates[3]

FABHALTA® (n=62)	C5i (n=35)
95% (95% CI: 88-100)	26% (95% CI: 12-42)

vs.

(Difference‡: 69%; 95% CI: 51-84; $p<0.001$)[3]

Most common adverse reactions: The most common adverse reactions (≥10%) in adults with PNH receiving FABHALTA® were headache, nasopharyngitis, diarrhoea, abdominal pain, bacterial infection, viral infection, nausea, and rash.
APPLY trial design: APPLY was a 24-week, randomised, open-label, controlled, phase 3 trial to assess the efficacy and safety of switching to FABHALTA® compared with continuing on intravenous C5i therapy (US-approved and non–US approved eculizumab or ravulizumab) in adult patients with PNH and residual anaemia (mean Hb of <10 g/dL) despite previous treatment with a stable regimen of C5i treatment for at least 6 months prior to randomisation; 97 patients were randomised to either switch to FABHALTA® 200 mg taken orally twice daily (n=62) or continue their C5i regimen (n=35).

C5i, C5 inhibitor; CI, confidence interval; Hb, haemoglobin; PNH, paroxysmal nocturnal haemoglobinuria; RBC, red blood cell.
†Both transfusion independence and normalisation of Hb is defined as Hb ≥12g/dL with no red-cell transfusions between days 14 and 168 or without meeting the protocol-specified criteria for red-cell transfusion.
**Assessed between Days 126 and 168. Requiring RBC transfusions refers to any patient receiving transfusions or meeting protocol-defined criteria.
†Transfusion independence rate defined as transfusion avoidance, neither receiving nor meeting the criteria for RBC transfusion assessed between Days 14 and 168.
‡Adjusted difference in proportion.
§Normalisation defined as meeting the primary end point of Hb≥12 g/dL. Normal Hb levels vary but generally are between 12-16 g/dL for women and 13-18 g/dL for men.

References: 1. FABHALTA® Hong Kong Prescribing Information. **2.** Risitano AM, et al. *Lancet Haematol.* 2021;8:e344–e354. **3.** Peffault de Latour R, et al. Oral iptacopan monotherapy in paroxysmal nocturnal hemoglobinuria. *New Engl J Med.* 2024;390(11):994-1008.

FABHALTA®
Important note: Before prescribing, consult full prescribing information. **Presentation:** Hard capsules containing 225.8 mg iptacopan hydrochloride monohydrate equivalent to 200 mg of iptacopan. **Indications:** FABHALTA is indicated as monotherapy in the treatment of adult patients with paroxysmal nocturnal haemoglobinuria (PNH) who have haemolytic anaemia. **Dosage and administration:** ♦ 200 mg orally twice daily, may be taken with or without food. ♦ Adherence to dosing schedule: advise patients with PNH about the importance of adherence to the dosing schedule in order to minimize the risk of haemolysis. ♦ Missed dose(s): Take one dose as soon as possible (even if it is shortly before the next scheduled dose) and then resume the regular dosing schedule. ♦ PNH is a disease that requires chronic treatment. Discontinuation of this medicinal product is not recommended unless clinically indicated. ♦ Switching from eculizumab to iptacopan: Iptacopan should be initiated no later than 1 week after the last dose of eculizumab. ♦ Switching from ravulizumab to iptacopan: Iptacopan should be initiated no later than 6 weeks after last dose of ravulizumab. ♦ Switching from other PNH therapies to iptacopan: Not studied. **Special populations:** ♦ **Elderly:** No dose adjustment required. ♦ **Renal impairment:** No dose adjustment for mild or moderate renal impairment. No data currently available in patients with severe renal impairment or on dialysis. ♦ **Hepatic impairment:** Not recommended in patients with severe hepatic impairment. No dose adjustment for mild or moderate, hepatic impairment. **Pediatric population (<18 years of age):** The safety and efficacy of iptacopan have not been established. **Contraindications:** ♦ Hypersensitivity to active substance or to any of the excipients. ♦ Patients not vaccinated against *Neisseria meningitidis* and *Streptococcus pneumoniae* unless the risk of delaying treatment outweighs the risk of developing an infection from these encapsulated bacteria. ♦ Patients with unresolved serious infection caused by encapsulated bacteria, including *Neisseria meningitidis, Streptococcus pneumoniae* or *Haemophilus influenzae* type B, at treatment initiation. **Warnings and precautions: Serious infections caused by encapsulated bacteria:** ♦ Complement inhibitors such as iptacopan, may predispose individuals to serious, life- threatening, or fatal infections. ♦ To reduce the risk of infection, patients must be vaccinated against encapsulated bacteria, including *Neisseria meningitidis* and *Streptococcus pneumoniae*. It is recommended to vaccinate against *Haemophilus influenzae* type B if available. ♦ Vaccines should be administered at least 2 weeks prior to administration of the first dose of iptacopan. ♦ If treatment must be initiated prior to vaccination, patients should be vaccinated as soon as possible and provided with antibacterial prophylaxis until 2 weeks after vaccination. ♦ If necessary, patients may be revaccinated. ♦ Patients should be informed of and monitored for early signs and symptoms of serious infection. ♦ Immediately evaluate and treat patients if infection is suspected. **PNH laboratory monitoring:** ♦ Monitor patients regularly for signs and symptoms of haemolysis, including measuring lactate dehydrogenase (LDH) levels. **Monitoring PNH manifestations after treatment discontinuation:** ♦ Monitor patients for at least two weeks after the last dose for signs and symptoms of haemolysis. ♦ Signs and symptoms include elevated LDH levels, along with sudden decrease in haemoglobin or PNH clone size, fatigue, haemoglobinuria, abdominal pain, dyspnoea, dysphagia, erectile dysfunction or major adverse vascular events (including thrombosis). ♦ Consider alternative therapy if treatment discontinuation is necessary. ♦ Consider restarting treatment if haemolysis occurs. **Pregnancy, lactation and fertility: Pregnancy:** Animal studies do not indicate harmful effects with respect to reproductive toxicity. PNH in pregnancy is associated with adverse maternal and foetal outcomes. May consider use in pregnant women or women planning to become pregnant following a careful assessment of the risks and benefits, if necessary. **Breast-feeding:** Not known if transferred into human milk. No data on the effects on the breast-fed newborn/infant or on milk production. A risk to the newborns/infants cannot be excluded. A decision must be made whether to discontinue breast-feeding or to discontinue/abstain from FABHALTA therapy taking into account the benefit of breast feeding for the child and the benefit of therapy for the woman. **Fertility:** No data on human fertility. Available non-clinical data do not suggest an effect on fertility. **Adverse drug reactions: Very common (≥10%):** Upper respiratory tract infection, headache, diarrhoea. **Common (≥1 to <10%):** Urinary tract infection, bronchitis, platelet count decreased, dizziness, abdominal pain, nausea, arthralgia. **Uncommon (≥0.1 to <1%):** Pneumonia bacterial, urticaria. Interactions: Effects of other medicinal products on iptacopan: Concomitant use of strong inducers of CYP2C8, UGT1A1, PgP, BCRP and OATP1B1/3, such as rifampicin, with iptacopan is not recommended due to the potential for reduced efficacy of iptacopan. **Effects of iptacopan on other medicinal products:** ♦ CYP3A4 substrates: *In vitro* data showed iptacopan has potential for induction of CYP3A4 and may decrease the exposure of sensitive CYP3A4 substrates. Caution if co-administered with sensitive CYP3A4 substrates, especially for those with a narrow therapeutic index (e.g. carbamazepine, ciclosporin, ergotamine, fentanyl, pimozide, quinidine, sirolimus, tacrolimus). ♦ CYP2C8 substrates: In vitro data showed iptacopan has potential for time-dependent inhibition of CYP2C8 and may increase the exposure of sensitive CYP2C8 substrates, such as repaglinide, dasabuvir or paclitaxel. Caution if co-administered with sensitive CYP2C8 substrates. **Packs:** 200mg (56's). **Legal classification:** P1S1S3 Reference: EMA Jun 2024. Last revision: Jul 2024

NOVARTIS
Novartis Pharmaceuticals (HK) Limited
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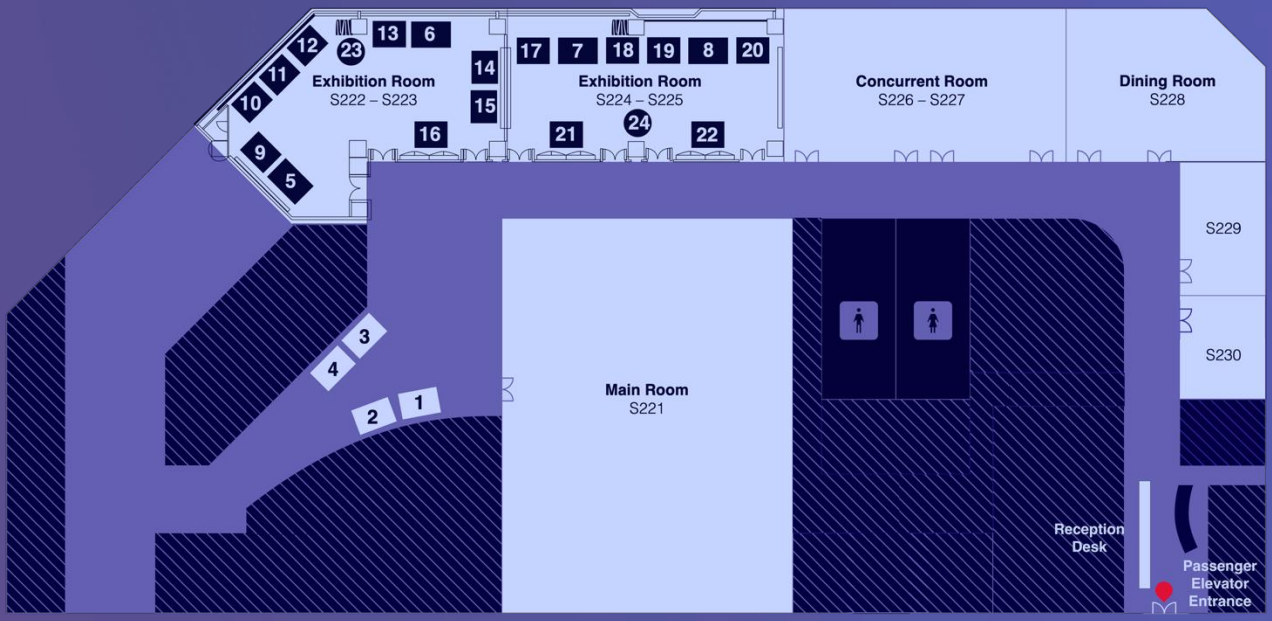
Tel: 2882 5222 Fax: 2577 0274
FA-I1338498 Jan 2025

HOUSE PLAN AND EXHIBITORS

Meeting Rooms S221 – S230



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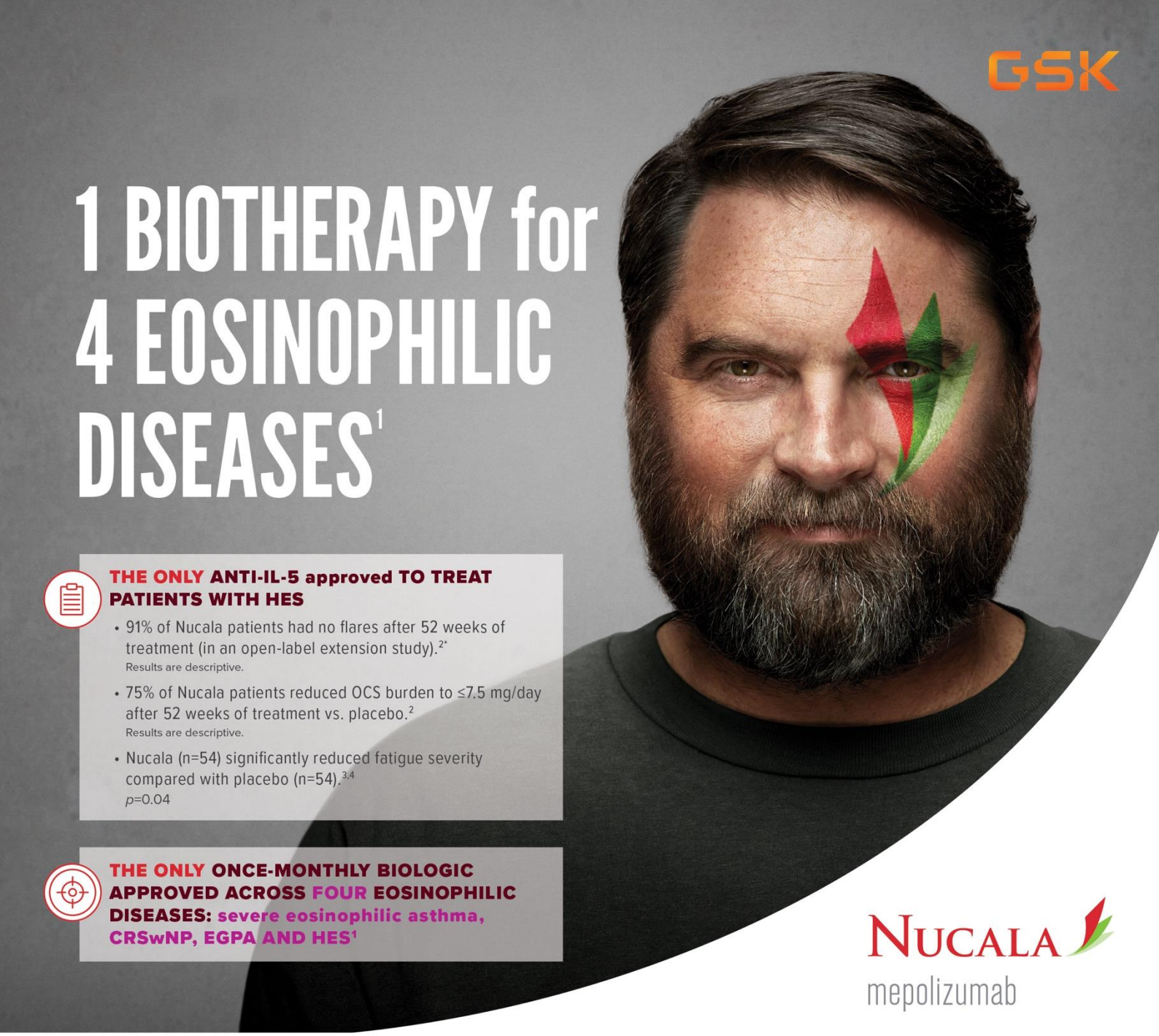
GSK

1 BIOTHERAPY for 4 EOSINOPHILIC DISEASES[1]



THE ONLY ANTI-IL-5 approved TO TREAT PATIENTS WITH HES

- 91% of Nucala patients had no flares after 52 weeks of treatment (in an open-label extension study).[2*]
  Results are descriptive.
- 75% of Nucala patients reduced OCS burden to ≤7.5 mg/day after 52 weeks of treatment vs. placebo.[2]
  Results are descriptive.
- Nucala (n=54) significantly reduced fatigue severity compared with placebo (n=54).[3,4]
  p=0.04

THE ONLY ONCE-MONTHLY BIOLOGIC APPROVED ACROSS FOUR EOSINOPHILIC DISEASES: severe eosinophilic asthma, CRSwNP, EGPA AND HES[1]

NUCALA
mepolizumab

Nucala is indicated as an add-on therapy with intranasal corticosteroids for the treatment of adult patients with severe CRSwNP for whom therapy with systemic corticosteroids and/or surgery do not provide adequate disease control. Nucala is also indicated as an add-on treatment for severe eosinophilic asthma, eosinophilic granulomatosis with polyangiitis (EGPA) and hypereosinophilic syndrome (HES).[1]

CRSwNP, chronic rhinosinusitis with nasal polyps; EGPA, eosinophilic granulomatosis with polyangiitis; HES, hypereosinophilic syndrome; IL, interleukin; mg, milligram; OCS, oral corticosteroids.

*Study 205203 is a multi-centre, open-label extension, 20-week treatment period, safety study of mepolizumab in adolescent and adult participants with HES who took part in the phase 3 study 200622. Subjects from study 200622 will participate in this extension study if they had completed the 32-Week treatment period in study 200622 or if they were withdrawn from the study pre-maturely, but were continued in the study per protocol until 32 Weeks from randomization. Data from this study (205203) and 200622 will be combined to provide up to 52-Week exposure data to further characterize the long-term safety profile of mepolizumab and provide additional data on the clinical benefit in HES subjects beyond 32 Weeks. The duration of the study participation will be 20 Weeks for subjects who continue with mepolizumab treatment via MHE104317/MHE112562 after this open-label extension study; and 28 Weeks for subjects who do not continue with MHE104317/MHE112562.[2]

Important Safety Information[1] • Nucala (mepolizumab) 100 mg solution for injection in pre-filled pen **Contraindications:** • Hypersensitivity to the active substance or to any excipients of Nucala solution for injection. **Warnings and Precautions:** • Not to be used to treat acute asthma exacerbations. Asthma-related adverse symptoms or exacerbations may occur during treatment. • Abrupt discontinuation of corticosteroids after initiation of Nucala therapy is not recommended. • Acute and delayed systemic reactions, including hypersensitivity reactions (e.g. anaphylaxis, urticaria, angioedema, rash, bronchospasm, hypotension), have occurred following administration of Nucala. • In the event of a hypersensitivity reaction, appropriate treatment as clinically indicated should be initiated. • Pre-existing helminth infections should be treated before starting Nucala. • Nucala has not been studied in patients with organ threatening or life-threatening manifestations of EGPA • Nucala has not been studied in patients with life-threatening manifestations of HES. **Adverse Events:** Most commonly reported adverse reactions in: • Severe eosinophilic asthma: headache, injection site reactions and back pain. • CRSwNP: headache and back pain. • EGPA: headache, injection site reactions and back pain • HES: headache, urinary tract infection, injection site reactions and pyrexia.

REFERENCES: 1. Nucala (mepolizumab) 100mg solution for injection in pre-filled pen Hong Kong Full Prescribing Information. Version: HK112021 (GDS14/EMA20211112). **2.** GlaxoSmithKline. Data on File: REF-162727. **3.** Roufosse F, Kahn JE, Rothenberg ME, *et al*. Efficacy and safety of mepolizumab in hypereosinophilic syndrome: A phase III, randomized, placebo-controlled trial. *J Allergy Clin Immunol*. 2020;146(6):1397–1405. 4. GlaxoSmithKline. Data on File: REF-96426.

Please read the full prescribing information prior to administration.
Full prescribing information is available upon request.

GlaxoSmithKline Limited Suites 1004-10, 10/F, Tower 6, The Gateway, 9 Canton Road, Tsimshatsui, Kowloon, Hong Kong Tel: (852) 3189 8989 Fax: (852) 3189 8931

PM-HK-MPL-ADVT-230001 (Dec 2026) Date of preparation: Jan 2025

The first & only JAK inhibitor indicated specifically for myelofibrosis (MF) patients with moderate to severe anaemia[1]

A TREATMENT OPTION FOR

SPLENOMEGALY OR SYMPTOMS

IN PATIENTS WHO HAVE MF

MODERATE TO SEVERE ANAEMIA

MF patients with anaemia (Hb <10 g/dL) were assessed for the following at Week 24[1]:

TOTAL SYMPTOM SCORE REDUCTION

- Rate of total symptom score (TSS)*† reduction ≥50% from baseline met as a co-primary endpoint in MOMENTUM and was assessed in a subgroup analysis in SIMPLIFY-1[1‡]

SPLEEN VOLUME REDUCTION

- Rate of spleen volume reduction (SVR) ≥35% from baseline met as a secondary endpoint in MOMENTUM and was assessed in a subgroup analysis in SIMPLIFY-1[1‡]

In clinical trials, the most common adverse reactions were diarrhoea, thrombocytopenia, nausea, headache, dizziness, fatigue, asthenia, abdominal pain, and cough.[1§]

Omjjara
momelotinib

Hb=haemoglobin; JAK=Janus kinase.

*TSS measured using the Myelofibrosis Symptom Assessment Form (MFSAF) version 4.0 in MOMENTUM.[1]

†TSS measured using the modified Myeloproliferative Neoplasm Symptom Assessment Form (MPN-SAF) v2.0 diary in SIMPLIFY-1.[1]

‡Efficacy was assessed in SIMPLIFY-1 for a subgroup of patients who had anaemia (Hb <10g/dL).[1]

§The safety population included 448 patients from three Phase 3 trials.[1]

References: 1. Omjjara (momelotinib) Hong Kong Prescribing Information. 2025

Safety information for Omjjara (Momelotinib)
CONTRAINDICATIONS: • Hypersensitivity to the active substance or to any excipient of Omjjara. • Pregnancy and breast-feeding. **WARNINGS AND PRECAUTIONS:** Momelotinib may cause serious and fatal bacterial and viral infections and should not be initiated in patients with active infections. Monitoring for hepatitis B reactivation, thrombocytopenia, neutropenia, and liver function is essential. Major adverse cardiovascular events (MACE), venous thromboembolic events, and second primary malignancies have been reported, though causality is unestablished. Prior to initiating or continuing therapy, the benefits and risks should be considered particularly in patients with cardiovascular risk factors. Patients should be monitored for adverse reactions with co-administration of certain medicinal products (e.g., sensitive breast cancer resistance protein [BCRP] substrates). Additional monitoring of the clinical signs and symptoms of myelofibrosis is recommended with concomitant use of Omjjara and strong CYP3A4 inducers. Women of childbearing potential should use additional contraceptive methods during treatment and for at least 1 week after the last dose of Omjjara. Omjjara contains lactose and is essentially sodium-free. **ADVERSE REACTIONS:** • Urinary tract infection • Upper respiratory tract infection • Pneumonia • Nasopharyngitis • COVID-19 • Cystitis • Bronchitis • Oral herpes • Sinusitis • Herpes zoster • Cellulitis • Respiratory tract infection • Sepsis • Lower respiratory tract infection • Oral candidiasis • Skin infection • Gastroenteritis • Thrombocytopenia • Neutropenia • Vitamin B1 deficiency • Dizziness • Headache • Syncope • Peripheral neuropathy • Paraesthesia • Blurred vision • Vertigo • Hypotension • Haematoma • Flushing • Cough • Diarrhoea • Abdominal pain • Nausea • Vomiting • Constipation • Arthralgia • Pain in extremity • Asthenia • Fatigue • Pyrexia • Alanine transaminase (ALT) increased • Aspartate transaminase (AST) increased • Contusion

Please read the full prescribing information prior to administration.
Full prescribing information is available upon request.
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For adverse event reporting, please call GlaxoSmithKline Limited at (852) 3189 8989 (Hong Kong), or send an email to us at **HKAdverseEvent@gsk.com**.
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PM-HK-MML-LBND-250001 Date of preparation: Feb 2025 (Jan 2027)

GSK

CME / CNE ACCREDITATION

CME Accreditation

College	Credits	Group-Category
Hong Kong College of Paediatricians	5	A-PP
Hong Kong College of Physicians	4	PP-PP
Hong Kong College of Radiologists	4.5	B-PP
The Hong Kong College of Pathologists	5	CME-PP

CNE Accreditation

College	Credits
Hong Kong Haematology Nursing Association	3.5

XOSPATA®
gilteritinib

XOSPATA® is indicated as monotherapy for the treatment of adult patients who have relapsed or refractory acute myeloid leukaemia (AML) with a FLT3 mutation.[1]

TAKE A STAND FOR LONGER SURVIVAL

Median Overall Survival[2]

9.3 Months with Xospata **VS** **5.6 Months** with salvage chemotherapy

HR=0.64 (95% CI: 0.49, 0.83); P< 0.001

Percentage of patients underwent transplantation[2]

25.5% with Xospata (n=63/247) **VS** **15.3%** with salvage chemotherapy (n= 19/124)

Recommended by:[3-5]

NCCN ESMO GOOD SCIENCE BETTER MEDICINE BEST PRACTICE NICE National Institute for Health and Care Excellence

Abbreviations:
CI: confidence interval; FLT3: FMS-like tyrosine kinase 3; HR: hazard ratio; OS: overall survival

References:
1. Xospata® Prescribing Information Hong Kong 2. Perl AE, Martinelli G, Cortes JE, etal. Gilteritinib or chemotherapy for relapsed or refractory FLT3-mutated AML. N Engl J Med 2019;381(18):1728-40 3. National Comprehensive Cancer Network (NCCN). NCCN Clinical Practice Guidelines in Oncology: Acute myeloid leukemia (Version 2. 2021 -November) 4. Heuser M, et al. Annals of Oncology. 2020;31(6): 697-712. 5. NICE technology appraisal guidance. Available at: https://www.nice.org.uk/guidance/ta642/resources/gilteritinib-for-treating-relapsed-or-refractory-acute-myeloid-leukaemia-pdf-82609134829765, accessed on 22 Mar 2022.

Please find the abbreviated prescribing information here:

Email us at pv@hk.astellas.com when there is any Adverse Event

Astellas Pharma Hong Kong Company Limited
Unit 1103-08, 11/F, Tower 1, Grand Century Place, 193 Prince Edward Rd. West, Mongkok, Kowloon, Hong Kong
Tel: (852) 2377 9801 | Fax: (852) 2856 1440 | Email: info@hk.astellas.com

MAT-HK-XOS-2026-00001 | Jan 2026

astellas

OUR SPEAKERS

(Alphabetically ordered by last name)

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The University of Amsterdam
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Ms. Katrina DEBOSZ
CAR-T Nurse Practitioner,
Institute of Haematology,
Royal Prince Alfred Hospital
Australia

Prof. HUANG He
Professor,
The First Affiliated Hospital,
Zhejiang University School of Medicine,
China

Dr. Takeshi KONDO
Deputy Director,
Aiiku Hospital,
Sapporo,
Japan

Dr. Talha MUNIR
Consultant Haematologist,
St. James's Hospital,
Leeds,
United Kingdom

THE FIRST AND ONLY BTKi APPROVED IN FOLLICULAR LYMPHOMA

BRUKINSA® + Obinutuzumab reduced the relative risk of progression or death by 50% vs obinutuzumab alone[1]

Approx. 70% of patients had sustained responses at 18 months with BRUKINSA® + Obinutuzumab[1]

Reference

1. Zinzani PL, Mayer J, Flowers CR, et al. ROSEWOOD: A Phase II Randomized Study of Zanubrutinib Plus Obinutuzumab Versus Obinutuzumab Monotherapy in Patients With Relapsed or Refractory Follicular Lymphoma. J Clin Oncol. 2023; 41(33):5107-5117

Abbreviated Prescribing Information

Presentation: BRUKINSA® (zanubrutinib) capsules 80mg. **Indication:** BRUKINSA® as monotherapy is indicated for the treatment of adult patients with Waldenström's macroglobulinaemia (WM) who have received at least one prior therapy, or in first line treatment for patients unsuitable for chemo-immunotherapy. BRUKINSA® as monotherapy is indicated for the treatment of adult patients with marginal zone lymphoma (MZL) who have received at least one prior anti-CD20-based therapy. BRUKINSA® as monotherapy is indicated for the treatment of adult patients with chronic lymphocytic leukemia (CLL). BRUKINSA® in combination with obinutuzumab is indicated for the treatment of adult patients with refractory or relapsed follicular lymphoma (FL) who have received at least two prior systemic therapies. **Dosage & Administration:** The recommended total daily dose of BRUKINSA® is 320 mg. The daily dose may be taken either once daily (four 80 mg capsules) or divided into two doses of 160 mg twice daily (two 80 mg capsules). **Contraindications:** Hypersensitivity to the active substance or to any of the excipients. **Special Warnings & Precautions:** (1) Haemorrhage: Warfarin or other vitamin K antagonists should not be administered concomitantly with BRUKINSA®. Patients should be monitored for signs and symptoms of bleeding and monitor complete blood counts. Consider the risks and benefits of anticoagulant or antiplatelet therapy when co-administered with BRUKINSA®, (2) Infections: Consultation with a liver disease expert physician is recommended for patients who test positive for HBV or have positive hepatitis B serology, before initiating treatment. Patients should be monitored and managed according to the medical standards to prevent hepatitis B reactivation. Consider prophylaxis according to standard of care in patients who are at increased risk for infections. Patients should be monitored for signs and symptoms of infection and treat appropriately, (3) Cytopenia: Monitor complete blood counts monthly during treatment, (4) Second primary malignancies including skin cancer: Advise patients to use sun protection, (5) Atrial fibrillation and flutter: Monitor signs and symptoms for atrial fibrillation and atrial flutter and manage as appropriate, (6) Women of childbearing potential: Women of childbearing potential must use a highly effective method of contraception while taking BRUKINSA®, (7) BRUKINSA® contains sodium: This medicinal product contains less than 1 mmol sodium (23 mg) per dose, that is to say essentially 'sodium-free'. **Undesirable effects:** The most commonly occurring adverse reactions (≥20%) were neutropenia, thrombocytopenia, upper respiratory tract infection, haemorrhage/haematoma, rash, bruising, anaemia, musculoskeletal pain, diarrhoea, pneumonia and cough. Refer to the full prescribing information for other undesirable effects. **Interactions:** If a strong and moderate CYP3A inhibitor must be used, reduce the BRUKINSA® dose for the duration of the inhibitor use. Concomitant use with strong and moderate CYP3A inducers should be avoided. No clinically significant differences in BRUKINSA® pharmacokinetics were observed when co administered with gastric acid reducing agents. **Pregnancy & Lactation:** BRUKINSA® should not be used during pregnancy. Breast-feeding should be discontinued during treatment with BRUKINSA®. **Full prescribing information should be consulted prior to prescribing.**

VV-QDOC-229771 Approved on 28 Jan 2025

BeOne

BeOne Medicines (Hong Kong) Co., Ltd.
Room 1002, Level 10, Two Chinachem Central,
26 Des Voeux Road Central, Central, Hong Kong

Brukinsa® zanubrutinib 80mg capsules

OUR SPEAKERS

(Alphabetically ordered by last name)

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Prof. Niels VAN DE DONK
Professor in Hematology,
Amsterdam University Medical Center,
The Netherlands

Reblozyl®
(luspatercept)

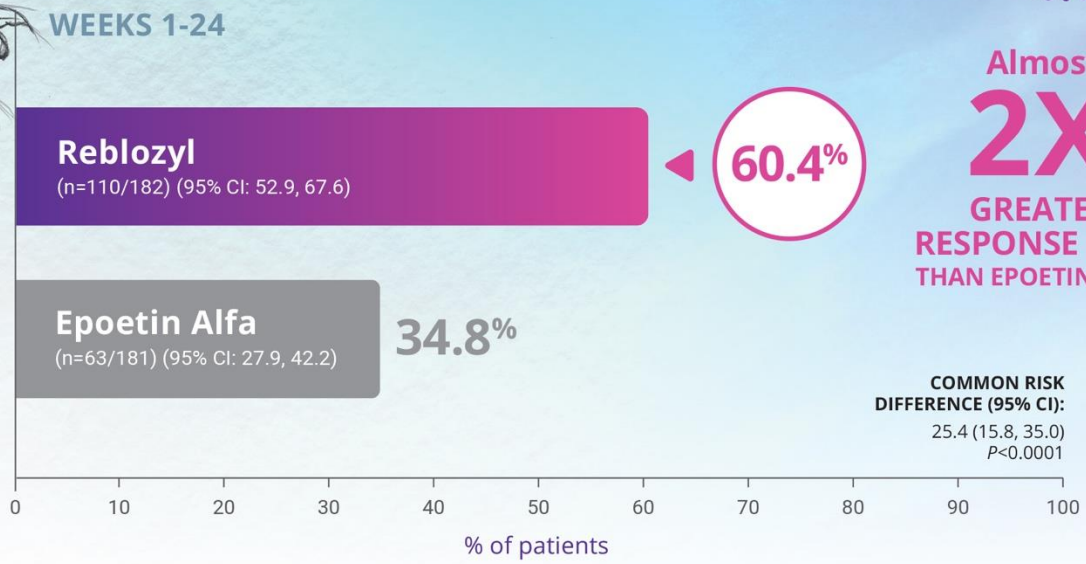
In a **head-to-head** study vs epoetin alfa...

Reblozyl sets the new standard for superior efficacy that lasts

In ESA-naïve patients with anemia due to LR-MDS
Q3W Reblozyl demonstrated superior response rates and durability vs QW epoetin alfa[1]

NOW APPROVED
For your ESA-naïve patients with transfusion-dependent anemia due to LR-MDS

Primary endpoint: RBC-TI for ≥12 weeks with concurrent mean Hb increase ≥1.5 g/dL[1]



Almost 2X GREATER RESPONSE RATE THAN EPOETIN ALFA

Indications[1]:

Reblozyl is indicated in adults for the treatment of transfusion-dependent anaemia due to very low, low and intermediate-risk myelodysplastic syndromes (MDS).
Reblozyl is indicated in adults for the treatment of anaemia associated with transfusion-dependent and non-transfusion-dependent beta-thalassaemia.

Limitations of use[1]

Reblozyl is not indicated for use as a substitute for RBC transfusions in patients who require immediate correction of anaemia.

CI=confidence interval; ESA=erythropoiesis-stimulating agent; LR-MDS=lower-risk myelodysplastic syndromes; Q3W=every 3 weeks; QW=once a week; RBC-TI=red blood cell transfusion independence.

References: 1. Reblozyl Hong Kong Prescribing Information 2024.

REBLOZYL 25 MG AND 75 MG POWDER FOR SOLUTION FOR INJECTION
ABBREVIATED PRESCRIBING INFORMATION

ACTIVE INGREDIENT: Each vial contains 25 mg or 75 mg of luspatercept. After reconstitution, each mL of solution contains 50 mg luspatercept. **INDICATIONS**: Reblozyl is indicated in adults for the treatment of transfusion-dependent anaemia due to very low, low and intermediate-risk myelodysplastic syndromes (MDS). Reblozyl is indicated in adults for the treatment of anaemia associated with transfusion-dependent and nontransfusiondependent betathalassaemia. Reblozyl is not indicated for use as a substitute for RBC transfusions in patients who require immediate correction of anemia. **DOSAGE & ADMINISTRATION:** The recommended starting dose of Reblozyl is 1.0 mg/kg administered once every 3 weeks. *Myelodysplastic syndromes:* If after at least 2 consecutive doses at the 1.0 mg/kg, a patient is not RBC transfusion-free, or does not reach Hb concentration of ≥ 10 g/dL and the Hb increase is < 1 g/dL, the dose should be increased to 1.33 mg/kg. If after at least 2 consecutive doses at the 1.33 mg/kg, a patient is not RBC transfusion-free, or does not reach Hb concentration of ≥ 10 g/dL and the Hb increase is < 1 g/dL, the dose should be increased to 1.75 mg/kg. The dose increase should not occur more frequently than every 6 weeks (2 administrations) and should not exceed the maximum dose of 1.75 mg/kg every 3 weeks. *Transfusion-dependent b-thalassaemia:* In patients who do not achieve a response, defined as a reduction in RBC transfusion burden of at least a third after ≥ 2 consecutive doses (6 weeks), at the 1.0 mg/kg starting dose, the dose should be increased to 1.25 mg/kg. The dose should not be increased beyond the maximum dose of 1.25 mg/kg every 3 weeks. *Non-transfusion-dependent b-thalassaemia:* In patients who do not achieve or maintain a response, defined as an increase from baseline in predose Hb of ≥1 g/dL, after ≥ 2 consecutive doses (6 weeks) at the same dose level (in absence of transfusions, i.e. at least 3 weeks after the last transfusion), the dose should be increased by one dose level (current dose 0.6 mg/kg to increased dose 0.8 mg/kg; current dose 0.8 mg/kg to increased dose 1 mg/kg; current dose 1 mg/kg to increased dose 1.25 mg/kg;). The dose should not exceed the maximum dose of 1.25 mg/kg every 3 weeks. Method of administration: For subcutaneous use. After reconstitution, Reblozyl solution should be injected subcutaneously into the upper arm, thigh or abdomen. **CONTRAINDICATIONS:** Hypersensitivity to the active substance or to any of the excipients. Pregnancy. Patients requiring treatment to control the growth of Extramedullary haemopoiesis masses (EMH) masses. **SPECIAL WARNINGS AND PRECAUTIONS FOR USE:** Traceability: In order to improve the traceability of biological medicinal products, the name and the batch number of the administered product should be clearly recorded. Thromboembolic events: In bthalassaemia patients, thromboembolic events (TEEs) were reported in 3.6% (8/223) of patients treated with luspatercept in the double-blind phase of the pivotal study in transfusiondependent patients and in 0.7% (1/134) of patients during the open-label phase of the pivotal study in non-transfusiondependent patients. Reported TEEs included deep vein thrombosis (DVT), portal vein thrombosis, pulmonary emboli, ischaemic stroke and superficial thrombophlebitis. All patients with TEEs were splenectomised and had at least one other risk factor for developing TEE (e.g. history of thrombocytosis or concomitant use of hormone replacement therapy). The occurrence of TEE was not correlated with elevated Hb levels. In MDS patients, TEEs were reported in 3.9% (13/335) of patients treated with luspatercept. Reported TEEs included cerebral ischemia and cerebrovascular accident in 1.2% (4/335) of patients. All TEEs occurred in patients with significant risk factors (atrial fibrillation, stroke or heart failure and peripheral vascular disease) and were not correlated with elevated Hb, platelet levels or hypertension. Extramedullary haemopoiesis masses (EMH): Patients with EMH masses may experience worsening of these masses and complications during treatment. Signs and symptoms may vary depending on anatomical location. Patients should be monitored at initiation and during treatment for symptoms and signs or complications resulting from the EMH masses, and be treated according to clinical guidelines. Treatment with luspatercept must be discontinued in case of serious complications due to EMH masses. Increased blood pressure: In MDS and bthalassaemia pivotal studies, patients treated with luspatercept had an average increase in systolic and diastolic blood pressure up to 5 mmHg from baseline. An increased incidence of hypertension was observed in the first 12 months of treatment in nontransfusiondependent bthalassaemia patients treated with luspatercept. The treatment must be started only if the blood pressure is adequately controlled. Blood pressure should be monitored prior to each luspatercept administration. Luspatercept dose may require adjustment or may be delayed, and patients should be treated for hypertension as per current clinical guidelines. The potential benefit of treatment with Reblozyl should be reevaluated in case of persistent hypertension or exacerbations of preexisting hypertension. Traumatic fracture: **Patients should be informed of the risk of traumatic fracture.** Sodium content: This medicinal product contains less than 1 mmol sodium (23 mg) per dose, that is to say essentially 'sodiumfree'. **INTERACTIONS:** No formal clinical interaction studies have been performed. **FERTILITY, PREGNANCY & LACTATION:** The effect of luspatercept on fertility in humans is unknown. Women of childbearing potential have to use effective contraception during treatment with Reblozyl and for at least 3 months after the last dose. Prior to starting treatment with Reblozyl, a pregnancy test has to be performed for women of childbearing potential and the patient card has to be provided. Treatment with Reblozyl should not be started if the woman is pregnant. Because of the unknown adverse effects of luspatercept in newborns/infants, a decision must be made whether to discontinue breast-feeding during therapy with Reblozyl and for 3 months after the last dose or to discontinue Reblozyl therapy, taking into account the benefit of breast-feeding for the child and the benefit of therapy for the woman. **ADVERSE REACTIONS:** *Myelodysplastic syndromes:* The most frequently reported adverse drug reactions in patients receiving Reblozyl were fatigue, diarrhoea, nausea,asthenia, dizziness, oedema peripheral and back pain . The most commonly reported Grade ≥ 3 adverse drug reactions included hypertension events, syncope, dyspnoea, fatigue and thrombocytopenia. The most commonly reported serious adverse drug reactions were urinary tract infection, dyspnoea and back pain. *Transfusion-dependent b-thalassaemia:* The most frequently reported adverse drug reactions in patients receiving Reblozyl were headache, bone pain and arthralgia. The most commonly reported Grade ≥ 3 adverse drug reaction was hyperuricaemia. The most serious adverse reactions reported included thromboembolic events of deep vein thrombosis, ischaemic stroke portal vein thrombosis and pulmonary embolism. *Non-transfusion-dependent b-thalassaemia:*The most frequently reported adverse drug reactions in patients receiving Reblozyl were bone pain, headache, arthralgia, back pain, prehypertension and hypertension. The most commonly reported Grade ≥ 3 and most serious adverse reaction reported was traumatic fracture. Spinal cord compression due to EMH masses occurred in 1% of patients.

Please refer to the full prescribing information before prescribing. Prescribing information is available on request.
Date of revision of the text: August 2024

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Bristol Myers Squibb™

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Causeway Bay, Hong Kong
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2007-HK-2500005 20250328

ABSTRACTS

Lecture 1

Raising the Bar for Relapsed / Refractory Follicular Lymphoma: How Can We Go Further?

Dr. Laurie SEHN
Clinical Professor,
BC Cancer Centre for Lymphoid Cancer and University of British Columbia,
Canada

Abstract

Despite effective first-line regimens, many patients with follicular lymphoma eventually relapse, and outcomes are particularly poor for those with early treatment failure or refractory disease. In recent years, treatment options for relapsed/refractory follicular lymphoma have expanded to include bispecific antibodies, CAR T-cell therapy, and other novel agents that can induce deep and durable remissions in patients who have exhausted conventional therapies.

This lecture will explore evolving strategies aimed at improving patient outcomes in relapsed or refractory follicular lymphoma and review pivotal clinical trial data of emerging combination approaches that seek to deepen responses and extend remission duration. Real-world considerations—including trial eligibility versus everyday practice, access, logistics, and shared decision-making—will be emphasized to help physicians individualize treatment plans and improve long-term outcomes for patients with relapsed or refractory follicular lymphoma.

Truxima®
Rituximab

THE FIRST RITUXIMAB BIOSIMILAR APPROVED BY THE EMA AND FDA[1-4]

TRUXIMA® offers potential pharmacoeconomic benefits for healthcare systems, which translate to improved patient access to biologic treatments and combination therapies and facilitate treatment innovations[4]

79+ countries

Truxima® is approved in more than 79 countries and is available in 59 countries[5]

More than **1,600 Truxima®** prescription cases have been reported[§4]

Truxima® has received authorisation for use in below indications:

- **Non-Hodgkin's Lymphoma**[*†6,7]
- **Diffuse Large B Cell Lymphoma**[‡8]
- **Chronic Lymphocytic Leukamia**[†7]

[*] A phase III, randomized, and double-blind study in 140 patients with previously untreated advanced follicular lymphoma. Patients treated with Truxima® (n=70) showed comparable PFS, TTP and OS compared to originator rituximab (n=70). Truxima® was consistently well tolerated and showed comparable safety profiles, including immunogenicity, over the entire study period without any unexpected toxicities.
[†] A study collecting real-world safety and effectiveness data on rapid infusion (RI)-Truxima® from the medical records of 196 patients with non-Hodgekin's lymphoma or chronic lymphocytic leukemia in 10 European centers, 6 months after the date of the first RI (index date); the infusion-related reaction rate was compared to previously published data. Ten percent of patients experienced an infusion-related reaction on day 1-2 post-index, which was not significantly different to the rate for rituximab described previously (8.8%). Complete response and partial response to Truxima® was observed in 74% and 22% of patients, respectively. This real-world study demonstrates that the safety and effectiveness profile of RI-Truxima® is similar to RI of reference rituximab.
[‡] A study collecting real-world data relating to the effectiveness and safety of Truxima® treatment from the medical records of 389 patients with diffuse large B cell lymphoma (24 centers, five European countries). For the primary outcome (clinical effectiveness), OS, PFS and best response (BR) were assessed. The percentage (95% confidence interval [95% CI]) of patients alive at 12-, 18-, and 30 months postindex (initiation of Truxima®) was 86% (82.4%–89.4%), 81% (76.9%–84.9%), and 76% (71.2%–80.1%), respectively. The PFS rate (percent, [95% CI]) at 12-, 18-, and 30 months postindex was 78% (74.2%–82.5%), 72% (67.9%–76.9%), and 67% (61.9%–71.7%), respectively. For 82% (n = 312) of patients, the BR to Truxima® was a complete response. Adverse events were consistent with known effects of chemotherapy.
[§] Estimated total number of patients receiving Truxima® in real-world evidence studies listed in Choi D, et al. 2022.[4]

EMA=European Medicine Agency; FDA=Food and Drug Administration; OS=overall survival; PFS=progression-free survival; RI=rapid infusion; TTP=time to progression.

Package leaflet: Information for the patient
Truxima® 100mg / 500 mg concentrate for solution for infusion
Read this leaflet carefully before you start taking this medicine because it contains important information for you. • Keep this leaflet. You may need to read it again. • If you have any further questions, ask your doctor, pharmacist or nurse. • If you get any side effects talk to your doctor, pharmacist or nurse. This includes any possible side effects not listed in this leaflet. See section 4. **What is in this leaflet:** 1. What Truxima® is and what it is used for 2. What you need to know before you use Truxima® 3. How to use Truxima® 4. Possible side effects 5. How to store Truxima® 6. Contents of the pack and other information **1. What Truxima® is and what it is used for What Truxima® is** Truxima® contains the active substance "rituximab". This is a type of protein called a "monoclonal antibody". It is designed to stick to a type of white blood cell called "B-Lymphocyte". When sticking to the surface of this cell, rituximab causes the cell to die.**What Truxima® is used for** Truxima® may be used for the treatment of several different conditions in adults. Your doctor may prescribe Truxima® for the treatment of: **a) Non-Hodgkin's Lymphoma** This is a disease of the lymph tissue (part of the immune system) that affects a type of white blood cell called B-Lymphocytes. In adults, Truxima® can be given alone or with other medicines called "chemotherapy". In adult patients where the treatment is working, Truxima® may be used as a maintenance treatment for 2 years after completing the initial treatment. **b) Chronic lymphocytic leukaemia** Chronic lymphocytic leukaemia (CLL) is the most common form of adult leukaemia. CLL affects a particular lymphocyte, the B cell, which originates from the bone marrow and develop in the lymph nodes. Patients with CLL have too many abnormal lymphocytes, which accumulate mainly in the bone marrow and blood. The proliferation of these abnormal B-lymphocytes is the cause of symptoms you may have. Truxima® in combination with chemotherapy destroys these cells which are gradually removed from the body by biological processes.**2. What you need to know before you use Truxima® Do not take Truxima® if:** • you are allergic to rituximab, other proteins which are like rituximab, or any of the other ingredients of this medicine (listed in section 6) • you have a severe active infection at the moment • you have a weak immune system • you have severe heart failure or severe uncontrolled heart disease and have rheumatoid arthritis, granulomatosis with polyangiitis or microscopic polyangiitis. Do not have Truxima® if any of the above apply to you. If you are not sure, talk to your doctor, pharmacist or nurse before you are given Truxima®. **Warnings and precautions** Talk to your doctor, pharmacist or nurse before you are given Truxima® if: • you have ever had or might now have a hepatitis infection. This is because in a few cases, Truxima® could cause hepatitis B to become active again, which can be fatal in very rare cases. Patients who have ever had hepatitis B infection will be carefully checked by their doctor for signs of this infection. • you have ever had heart problems (such as angina, palpitations or heart failure) or breathing problems. If any of the above apply to you (or you are not sure), talk to your doctor, pharmacist or nurse before you are given Truxima®. Your doctor may need to take special care during your treatment with Truxima®. **Children and adolescents** Talk to your doctor, pharmacist or nurse before you are given this medicine if you, or your child, are under 18 years of age. This is because there is not much information about the use of Truxima® in children and young people. **Other medicines and Truxima®** Tell your doctor, pharmacist or nurse if you are taking, have recently taken or might take any other medicines. This includes medicines obtained without a prescription and herbal medicines. This is because Truxima® can affect the way some other medicines work. Also some other medicines can affect the way Truxima® works. **In particular, tell your doctor:** • if you are taking medicines for high blood pressure. You may be asked not to take these other medicines 12 hours before you are given Truxima®. This is because some people have a fall in their blood pressure while they are being given Truxima®. • if you have ever taken medicines which affect your immune system – such as chemotherapy or immune-suppressive medicines. If any of the above apply to you (or you are not sure), talk to your doctor, pharmacist or nurse before you are given Truxima®. **Pregnancy and breast-feeding** You must tell your doctor or nurse if you are pregnant, think that you might be pregnant or are planning to become pregnant. This is because Truxima® can transfer across the placenta and may affect your baby. If you can get pregnant, you and your partner must use an effective method of contraception while using Truxima®. You must also do this for 12 months after your last treatment with Truxima®. Do not breast-feed while you are being treated with Truxima®. Also do not breast-feed for 12 months after your last treatment with Truxima®. This is because Truxima®may pass into breast milk. **Driving and using machines** It is not known whether Truxima® has an effect on you being able to drive or use any tools or machines. **Truxima® contains sodium** This medicine contains 52.6 mg sodium in each 10 mL vial and 263.2 mg in each 50 mL vial. This is equivalent to 2.6% (for 10ml vial) and 13.2% (for 50ml vial) of the recommended maximum daily dietary intake of sodium for an adult. **3. How to use Truxima® How it is given** Truxima® will be given to you by a doctor or nurse who is experienced in the use of this treatment. They will watch you closely while you are being given this medicine. This is in case you get any side effects. You will always be given Truxima® as a drip (intravenous infusion). **Medicines given before each Truxima® administration** Before you are given Truxima®, you will be given other medicines (pre-medication) to prevent or reduce possible side effects. **How much and how often you will receive your treatment a) If you are being treated for non-Hodgkin's Lymphoma** - *If you are having Truxima® alone* Truxima® will be given to you once a week for 4 weeks. Repeated treatment courses with Truxima® are possible. - *If you are having Truxima® with chemotherapy* Truxima® will be given to you on the same day as your chemotherapy. This is usually given every 3 weeks up to 8 times. • If you respond well to treatment, you may be given Truxima® as a maintenance treatment every 2 or 3 months for two years. Your doctor may change this, depending on how you respond to the medicine. **b) If you are being treated for chronic lymphocytic leukaemia** When you are treated with Truxima® in combination with chemotherapy, you will receive Truxima® infusions on day 0 cycle 1 then day 1 of each cycle for 6 cycles in total. Each cycle has a duration of 28 days. The chemotherapy should be given after the Truxima® infusion. Your doctor will decide if you should receive concomitant supportive therapy. If you have any further questions on the use of this medicine, ask your doctor, pharmacist or nurse. **4. Possible side effects** Like all medicines, this medicine can cause side effects, although not everybody gets them. Most side effects are mild to moderate but some may be serious and require treatment. Rarely, some of these reactions have been fatal. **Infusion reactions** During or within the first 24 hours of the first infusion you may develop fever, chills and shivering. Less frequently, some patients may experience pain at the infusion site, blisters, itching, sickness (nausea), tiredness, headache, breathing difficulties, blood pressure raised, wheezing, throat discomfort, tongue or throat swelling, itchy or runny nose, vomiting, flushing or palpitations, heart attack or low number of platelets. If you have heart disease or angina, these infusion reactions might get worse. **Tell the person giving you the infusion immediately** if you develop any of these symptoms, as the infusion may need to be slowed down or stopped. You may require additional treatment such as an antihistamine or paracetamol. When these symptoms go away, or improve, the infusion can be continued. These reactions are less likely to happen after the second infusion. Your doctor may decide to stop your Truxima® treatment if these reactions are serious. **Infections Tell your doctor immediately if you get signs of an infection including:** • fever, cough, sore throat, burning pain when passing urine or feeling weak or generally unwell • memory loss, trouble thinking, difficulty walking or sight loss – these may be due to a very rare, serious brain infection, which has been fatal (progressive multifocal leukoencephalopathy or PML). You might get infections more easily during your treatment with Truxima®. These are often colds, but there have been cases of pneumonia or urinary infections. These are listed below under "Other side effects". **Skin reactions** Very rarely, severe blistering skin conditions that can be life-threatening may occur. Redness, often associated with blisters, may appear on the skin or on mucous membranes, such as inside the mouth, the genital areas or the eyelids, and fever may be present. **Tell your doctor immediately if you have any of these symptoms. Other side effects include: a) If you are being treated for non-Hodgkin's Lymphoma or chronic lymphocytic leukaemia** Very common side effects (may affect more than 1 in 10 people): • bacterial or viral infections, bronchitis • low number of white blood cells, with or without fever or blood cells called "platelets" • feeling sick (nausea) • bald spots on the scalp, chills, headache • lower immunity – because of lower levels of anti-bodies called "immunoglobulins" (IgG) in the blood which help protect against infection Common side effects (may affect up to 1 in 10 people): • infections of the blood (sepsis), pneumonia, shingles, cold, bronchial tube infections, fungal infections, infections of unknown origin, sinus inflammation, hepatitis B • low number of red blood cells (anaemia), low number of all blood cells • allergic reactions (hypersensitivity) • high blood sugar level, weight loss, swelling in the face and body, high levels of the enzyme "lactate dehydrogenase (LDH)" in the blood, low calcium levels in the blood • unusual feelings of the skin – such as numbness, tingling, pricking, burning, a creeping skin feeling, reduced sense of touch • feeling restless, problems falling asleep, • becoming very red in the face and other areas of the skin as a consequence of dilation of the blood vessels • feeling dizzy or anxious • producing more tears, tear duct problems, inflamed eye (conjunctivitis) • ringing sound in the ears, ear pain • heart problems – such as heart attack and uneven or fast heart rate • high or low blood pressure (low blood pressure especially when standing upright) • tightening of the muscles in the airways which causes wheezing (bronchospasm), inflammation, irritation in the lungs, throat or sinuses, being short of breath, runny nose • being sick (vomiting), diarrhoea, pain in the stomach, irritation or ulcers in the throat and mouth, problems swallowing, constipation, indigestion • eating disorders: not eating enough, leading to weight loss • hives, increased sweating, night sweats • muscle problems – such as tight muscles, joint or muscle pain, back and neck pain • general discomfort or feeling uneasy or tired, shaking, signs of flu • multiple-organ failure. Uncommon side effects (may affect up to 1 in 100 people): • blood clotting problems, decrease of red blood cell production and increase of red blood cell destruction (aplastic haemolytic anaemia), swollen or enlarged lymph nodes • low mood and loss of interest or enjoyment in doing things, feeling nervous • taste problems – such as changes in the way things taste • heart problems – such as reduced heart rate or chest pain (angina) • asthma, too little oxygen reaching the body organs • swelling of the stomach. Very rare side effects (may affect up to 1 in 10,000 people): • short term increase in the amount of some types of anti-bodies in the blood (called immunoglobulins – IgM), chemical disturbances in the blood caused by break-down of dying cancer cells • nerve damage in arms and legs, paralysed face • heart failure • inflammation of blood vessels including those leading to skin symptoms • respiratory failure • damage to the intestinal wall (perforation) • severe skin problems causing blisters that can be life-threatening. Redness, often associated with blisters, may appear on the skin or on mucous membranes, such as inside the mouth, the genital areas or the eyelids, and fever may be present. • kidney failure • severe vision loss Not known (it is not known how often these side effects happen): • a reduction in white blood cells which does not happen straight away • reduced platelets number just after the infusion – this can be reversed, but can be fatal in rare cases • hearing loss, loss of other senses Truxima® may also cause changes in laboratory tests carried out by your doctor. If you are having Truxima® with other medicines, some of the side effects you may get may be due to the other medicines. Reporting of side effects If you get any side effects talk to your doctor, pharmacist or nurse. This includes any side effects not listed in this leaflet. By reporting side effects you can help provide more information on the safety of this medicine. **5. How to store Truxima®** Keep this medicine out of the sight and reach of children. Do not use this medicine after the expiry date which is stated on the carton and the vial after EXP. The expiry date refers to the last day of that month. Store in a refrigerator (2 °C – 8 °C). Keep the container in the outer carton in order to protect from light. Do not throw away any medicines via wastewater or household waste. Ask your pharmacist how to throw away medicines that you no longer use. These measures will help protect the environment.**6. Contents of the pack and other information What Truxima® contains** • The active ingredient in Truxima® is called rituximab. The 10 mL vial contains 100 mg of rituximab (10 mg/mL). The 50 mL vial contains 500 mg of rituximab (10 mg/mL). • The other ingredients are sodium chloride, tri-sodium citrate dihydrate, polysorbate 80 and water for injections. **What Truxima® looks like and contents of the pack** Truxima® is a clear, colourless solution, supplied as a concentrate for solution for infusion.10mL vial – Pack of 2 vials. 50mL vial – Pack of 1 vial. **Marketing Authorisation Holder** Celltrion Healthcare Hong Kong Limited, Suite 3305, 33rd Floor, Tower 5, The Gateway, Harbour City, 15 Canton Road, Tsim Sha Tsui, Kowloon. **Manufacturer,** CELLTRION, Inc.20, Academy-ro 51 beon-gil, Yeonsu-gu, Incheon, 22014, Korea **This leaflet was last revised in July 2022.**

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This material is for healthcare professionals only.

HEALTHCARE CELLTRION
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ABSTRACTS

Lecture 2

Entering A New Era in the Management of Acute Lymphoblastic Leukaemia

Dr. NG Chin Hin
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Singapore

Abstract

Background:
Acute Lymphoblastic Leukaemia (ALL) is an aggressive haematologic malignancy characterized by the uncontrolled proliferation of immature lymphoid cells. Despite significant progress with chemotherapy-based regimens, frontline treatment remains challenging due to relapse risk, toxicity.

Novel immunotherapies, such as bispecific T-cell engagers, are reshaping the treatment landscape by harnessing the immune system to achieve deeper, more durable remissions. Incorporating these agents early in therapy offers the potential to improve survival outcomes, reduce reliance on intensive chemotherapy, and minimize long-term treatment-related complications.

Objectives:
This presentation will explore how novel immunotherapy, such as bispecific T-cell engager addresses unmet needs by maximizing survival opportunities for MRD-negative patients, advocate for a MRD-centric treatment paradigm, and discuss how immunotherapies' benefits against transplant-related toxicity and immune system burden.

- **Specifically targets** the aggressive *FLT3*-ITD mutation[1-4]
- Studied in a wide range of ages, **up to 75 years of age**[1,5]
- Additional OS benefit in patients **post-allo-HSCT**[6]

Superior overall survival with

22%

in risk of death*[1,5]

HR=0.78; 95% CI: 0.62-0.98 P=0.032

Median duration of complete response

38.6 months

with VANFLYTA® + standard chemo†[7]

95% CI: 21.9-NE; N=147

12.4 months

with placebo + standard chemo†[7]

95% CI: 8.8-22.7; N=150

Study design: QuANTUM-First is a randomised, double-blind, placebo-controlled, global phase 3 study which enrolled patients aged 18-75 with newly diagnosed AML with a *FLT3*-ITD mutation. Patients were randomly assigned (1:1) to VANFLYTA® or placebo group. During the first induction cycle, all patients received standard 7+3 induction regimen with IV cytarabine from days 1-7 and IV anthracycline (daunorubicin or idarubicin) on days 1-3. Patients were randomly assigned on day 7 to receive VANFLYTA® 35.4 mg or placebo orally once daily for 14 days. Patients with persistent leukemia after the first cycle could receive a second cycle of induction chemotherapy with either 7+3 or 5+2 regimen plus VANFLYTA® or placebo. Patients with CR or with CRi could proceed to consolidation, consisting of high-dose cytarabine (on days 1, 3 and 5) plus VANFLYTA® 35.4 mg or placebo and/or allo-HSCT, for up to 4 cycles. During maintenance, patients received VANFLYTA® (26.5 mg once daily for days 1-14, then 53 mg once daily thereafter) or placebo. Primary efficacy outcome was OS. Exploratory outcomes included RFS and DoCR by IRC.[1,5,7]

Indication: VANFLYTA® is indicated in combination with standard cytarabine and anthracycline induction and standard cytarabine consolidation chemotherapy, followed by VANFLYTA® single-agent maintenance therapy for adult patients with newly diagnosed acute myeloid leukaemia (AML) that is *FLT3*-ITD positive.[1]

*Median OS of VANFLYTA® plus standard chemotherapy (31.9 months; 95% CI: 21.0-NE) versus placebo plus standard chemotherapy (15.1 months; 95% CI: 13.2-26.2).[5]

†Patients who achieved CR during induction per IRC assessment.[7]

allo-HSCT=allogeneic haematopoietic stem cell transplantation; AML=acute myeloid leukemia; CI=confidence interval; CR=complete remission; CRi=complete remission with incomplete neutrophil or platelet recovery; DoCR=duration of complete remission; *FLT3*=FMS (feline McDonough sarcoma)-like tyrosine kinase 3; HR=hazard ratio; IRC=independent review committee; ITD=internal tandem duplication; IV=intravenous; NE=not estimable; OS=overall survival; RFS=relapse-free survival.

References: 1. VANFLYTA® Hong Kong Prescribing Information. (July 2024v01) **2.** Daver N, et al. Leukemia 2019;33:299-312. **3.** Mead AJ, et al. Blood 2007;110:1262-1270. **4.** Aikawa T, et al. Oncotarget 2020;11:943-955. **5.** Erba H, et al. Lancet 2023;401:1571-1583. **6.** Schlenk R, et al. Presented at: EHA2023 Hybrid Congress. S137. **7.** Supplementary appendix. Erba H, et al. Lancet 2023;401:1571-1583.

Presentation: VANFLYTA 17.7 mg film-coated tablets: Each tablet contains 17.7 mg quizartinib (as dihydrochloride). VANFLYTA 26.5 mg film-coated tablets: Each tablet contains 26.5 mg quizartinib (as dihydrochloride). **Active Ingredient:** Quizartinib (as dihydrochloride). **Indications:** In combination with standard cytarabine and anthracycline induction and standard cytarabine consolidation chemotherapy, followed by VANFLYTA single-agent maintenance therapy for adult patients with newly diagnosed acute myeloid leukaemia (AML) that is FLT3-ITD positive. **Dosage:** Induction: 35.4 mg (2 × 17.7 mg) once daily for two weeks in each cycle (28-day cycle). Patients can receive up to 2 cycles of induction. Consolidation: 35.4 mg (2 × 17.7 mg) once daily for two weeks in each cycle (28-day cycle). Patients can receive up to 4 cycles of consolidation. Maintenance: Starting dose of 26.5 mg once daily for two weeks if QTcF is ≤ 450 ms, then increased to 53 mg (2 × 26.5 mg) once daily if QTcF is ≤ 450 ms. Maintenance therapy may continue for up to 36 cycles (28-day cycle). **Contraindications:** Hypersensitivity to quizartinib or any excipients. Congenital long QT syndrome. Breast-feeding. **Precautions:** QT Interval Prolongation: Monitor ECG and electrolytes regularly. Do not start treatment if QTcF > 450 ms. Infections in Elderly Patients: Monitor closely for severe infections during induction. Contraception: Effective contraception is required for women of childbearing potential and male patients with female partners of childbearing potential. **Undesirable Effects:** Very Common: Upper respiratory tract infections, fungal infections, herpes infections, bacteraemia, thrombocytopenia, anaemia, neutropenia, decreased appetite, headache, epistaxis, diarrhoea, nausea, abdominal pain, vomiting, dyspepsia, increased alanine aminotransferase, oedema, prolonged electrocardiogram QT. Common: Pancytopenia. Uncommon: Cardiac arrest, ventricular fibrillation. Frequencies of undesirable effects vary with dosage. For further detail, please refer to full prescribing information.

Full local prescribing information is available upon request.

Please report Individual Case Safety Report (ICSR)/Adverse Event (AE) to Daiichi Sankyo via pv_hk@daiichisankyo.com

For healthcare professionals only.

HK-DAI-VAN-2504005 Approval date: May 2025

Daiichi-Sankyo

Daiichi Sankyo Hong Kong Limited
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ABSTRACTS

Presidential Symposium

Novel CAR-T Cellular Therapy for Hematological Malignancies and Autoimmune Diseases

Prof. HUANG He
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Abstract

Since the concept of CAR-T therapy was introduced in 1993, entered clinical practice in 2010, and the first acute lymphoblastic leukemia patient, Emily, received CAR-T treatment in 2012, this therapy has undergone over 12 years of development and refinement. To date, the application of CAR-T therapy has primarily focused on hematological malignancies, with B-lineage tumor-associated targets at its core. Among these, 15 commercial products targeting CD19 and BCMA have been developed, providing significant benefits to numerous patients. However, CAR-T therapy still faces numerous challenges that urgently need to be addressed. First, the scope of target coverage remains limited. Currently, there are no mature treatment options for T-lineage tumors, myeloid tumors, or solid tumors, highlighting the urgent need for the development of more novel targets. Second, the treatment model has its limitations. The current personalized preparation approach leads to high treatment costs and long waiting times for patients, making it difficult to meet clinical demands.

To address these challenges, researchers both domestically and internationally have conducted extensive explorations in recent years. Chinese research teams have emerged at the forefront of related fields globally. In the development of novel targets, significant progress has been made in CD7-targeted CAR-T research for T-lineage tumors. In the field of dual-target designs, multiple clinical research achievements related to CD19/CD22 and CD19/CD20 dual-target CAR-T therapies have been published in academic papers. In the development of universal CAR-T therapies, the technology has evolved from first-generation CD52 knockout to the current stage of achieving universality through multi-gene editing. Although these novel-target, dual-target, and universal CAR-T products have not yet been officially marketed, several have entered the IND (Investigational New Drug) stage with the National Medical Products Administration.

Overall, these functionally enhanced, novel-target, and universal CAR-T products represent important supplements and upgrades to existing commercial CAR-T therapies. With the continued advancement of related research, it is anticipated that more innovative CAR-T products will emerge in the coming years, further optimizing treatment efficacy and safety, and driving new breakthroughs and developments in the field of CAR-T therapy. Accelerate the clinical research and application of in vivo delivered CAR-T technology. Since its conceptual inception to clinical practice, this technology has advanced rapidly. Current clinical research evidence suggests that in vivo delivered CAR-T demonstrates promising therapeutic effects in hematological malignancies and autoimmune diseases.

These advancements are expected not only to further optimize the treatment efficacy for hematological diseases but also to expand into areas such as solid tumors and autoimmune diseases . Moreover, they hold potential for application in the treatment of aging-related conditions and fibrotic diseases, warranting continued attention and anticipation from both clinical and research communities.

FOSUN KAIROS
复星凯瑞

Leader in Cell and Gene Therapies

成为最有价值的细胞和基因疗法引领者

我们的愿景

ABSTRACTS

Lecture 4

Sequencing Myeloma Therapy Across the Continuum: 2nd Line to Late Line Perspectives

Prof. Niels VAN DE DONK
Professor in Hematology,
Amsterdam University Medical Center,
The Netherlands

Abstract

While the armamentaria for multiple myeloma treatment continues to expand, optimizing treatment sequencing to maximize patient survival remains a key challenge. This session will explore practical approaches to tailoring therapy from second-line through advanced relapse stages, with a focus on the unique considerations that influence decision-making.

The lecture will start by discussing how treatment-related and patient-related factors—including prior drug exposure, refractoriness to lenalidomide, comorbidities, disease biology, and access constraints—impact the choice of therapy at first relapse. Special emphasis will be placed on strategies for managing lenalidomide-refractory patients when access to novel agents is limited.

The discussion will then transition to later-line settings, highlighting real-world clinical experience in the use of T-cell redirecting therapies—such as bispecific antibodies and CAR-T cells—particularly in cases of aggressive or high-risk relapse.

Attendees would gain practical insights into balancing evidence-based best practices with on-the-ground realities, enabling them to make informed, context-specific sequencing decisions across the myeloma treatment continuum.

IASO BIOTECHNOLOGY

FUCASO
Equecabtagene Autoleucel Injection

The World's First Approved Fully Human anti-BCMA CAR-T Therapy[a]

DEEP RESPONSE DURABLE SURVIVAL

Treatment of R/R MM after ≥3 prior lines of therapy

Single Infusion

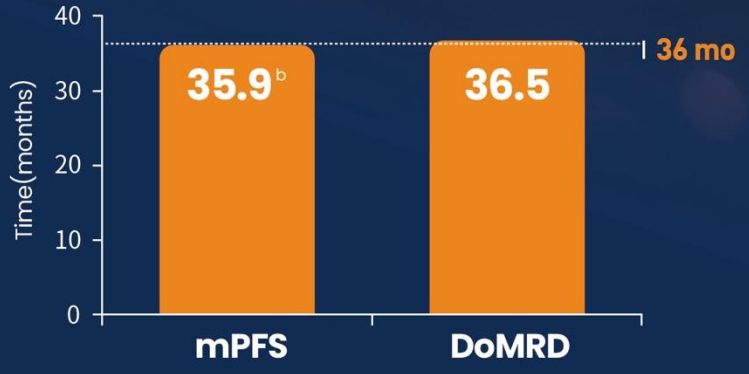
PIC/S Certification

PIC/S is an international organization that brings together over 50 regulatory authorities for medicinal products, including the **FDA**, **MHRA**, and **PMDA**, and works closely with the **EMA** and other key bodies. The GMP standards established by PIC/S are widely regarded as the global "**Gold Standard**" for pharmaceutical manufacturing.

Effective Disease Control & Prolonged Survival[2]

ORR	≥CR	MRD-NR
98.9%[b]	88.4%[b]	98.9%[b]

Median follow-up: 36 months



Favorable Safety Profile[2]

0.9%	0%
≥ Grade 3 CRS	≥ Grade 3 ICANS

The FUMANBA-1 study was a single-arm, multicenter, open-label, phase 1b/2 trial that enrolled patients with R/R MM who had received at least three prior lines of therapy. Its primary objectives were to evaluate the efficacy and safety of equecabtagene autoleucel. The primary endpoint for phase 1 was safety and tolerability; for phase 2, it was the ORR at 3 months after infusion. A total of 109 patients were enrolled, and 107 comprised the efficacy-evaluable population.

Deep response defined as ≥CR[1]; [a]patients without prior CAR-T treatment; [b]In June 2023, NMPA approved Fucaso® for the treatment of adult patients with R/R MM after three or more prior lines of therapy[3]. Prior to this, a total of two BCMA-targeted CAR-T cell therapies had been approved for the treatment of R/R MM worldwide, namely Idecabtagene vicleucel (murine scFV) and Ciltacabtagene autoleucel (llama scFV)[4].
R/R MM: Relapsed/Refractory Multiple Myeloma; BCMA: B Cell Maturation Antigen; CAR-T cell: Chimeric Antigen Receptor T Cell; PIC/S: Pharmaceutical Inspection Co-operation Scheme; FDA: Food and Drug Administration; EMA: European Medicines Agency; PMDA: Pharmaceuticals and Medical Devices Agency; GMP: Good Manufacturing Practice; ; CR: Complete Response; ORR: Overall Response Rate; MRD-NR: Minimal Residual Disease Negativity Rate; mPFS: Median Progression-Free Survival; CRS: Cytokine Release Syndrome; ICANS: Immune Effector Cell-Associated Neurotoxicity Syndrome ; MHRA: Medicines and Healthcare Products Regulatory Agency; NMPA: National Medical Products Administration; DoMRD: Duration of MRD Negativity; scFV: Single Chain Variable Fragment

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Approval code: XLMA20260203-1, Expiration date 2026.12.31

ABSTRACTS

Lecture 5

From Trial to Transformation: Five-Year Outcomes of HOPE-B Gene Therapy in Haemophilia B

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The University of Amsterdam
The Netherlands

Abstract

Haemophilia B is an X-linked coagulation disorder characterized by Factor IX deficiency, typically managed with lifelong intravenous factor replacement therapy. Etranacogene dezaparvovec (HEMGENIX®) is a one-time adeno-associated viral (AAV) vector-based therapy that enables sustained endogenous FIX expression. The HOPE-B phase 3 clinical trial evaluates this therapy in adults with severe or moderately severe Haemophilia B.

This presentation will review five-year follow-up data from HOPE-B, focusing on the durability of FIX activity, sustained hemostatic efficacy, and discontinuation of prophylactic therapy in 94% of participants. Key endpoints include annualized bleeding rate (ABR) reduction, factor consumption, and long-term safety signals. Additionally, post-therapy monitoring, patient-reported outcomes, and the evolving concept of a “haemophilia-free mindset” will be discussed. The session will conclude by examining implications for clinical practice in Asia and considering future directions for gene therapy.

ELREXFIO®
(elranatamab)
INJECTION FOR SUBCUTANEOUS USE | 44 mg/1.1 mL 76 mg/1.9 mL

In the treatment of relapsed and refractory multiple myeloma (RRMM)

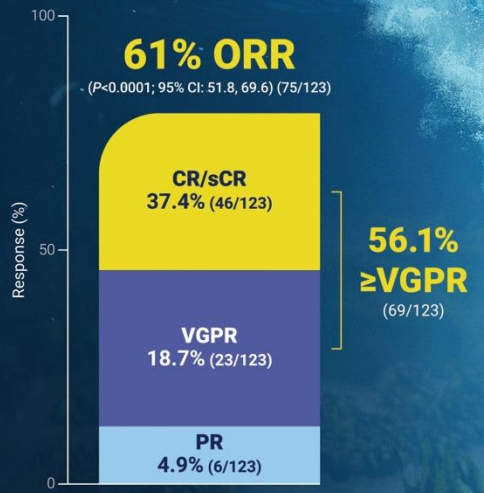
DISCOVER ACCESSIBLE DEEP RESPONSE

Deep response defined as ≥CR.[1]

ELREXFIO (elranatamab) is an off-the-shelf **BCMA-directed bispecific immunotherapy** that demonstrated **deep responses** in a range of patients with triple-class exposed MM.[2,*]

Delivered powerful and deepening responses[3-5]

- As of the 17.6-month median follow up, the majority of patients in the BCMA-naïve cohort (75/123) achieved an objective response (primary endpoint) and reached the median PFS in 17.2 months.[3]



Median PFS = 17.2 months[4]

- The most frequent adverse reactions are CRS (57.9%), anaemia (54.1%), neutropenia (44.8%), fatigue (44.3%), upper respiratory tract infection (38.8%), injection site reaction (38.3%), diarrhoea (37.7%), pneumonia (37.2%), thrombocytopenia (36.1%), lymphopenia (30.1%), decreased appetite (26.8%), pyrexia (27.3%), rash (26.2%), arthralgia (25.1%), hypokalaemia (23.0%), nausea (21.3%), and dry skin (21.3%).[2]

Designed for off-the-shelf subcutaneous administration[2]

- ELREXFIO offers the convenience of a ready-to-use, single-dose vial and no weight-based calculations[2]

After Week 24
QW to Q2W DOSING
in responding patients[2,‡]

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https://www.pfi.sr/TB2

Study design: MagnetisMM-3 was an open-label, multicenter, nonrandomized, Phase 2 study in 187 adult patients with RRMM refractory to at least 1 PI, 1 IMiD, and 1 anti-CD38 mAb. After 2 step-up doses of ELREXFIO, patients received 76 mg once weekly for Weeks 2 through 24, reduced to once every 2 weeks if a partial response or better was achieved and persisted for ≥2 months. The primary endpoint was ORR as assessed by BICR per IMWG criteria.[6]

* Patients previously treated with at least 1 PI, 1 IMiD, and 1 anti-CD38 mAb.

† CRS data based on the 183 patients who received the recommended step-up dosing regimen of 12 mg/32 mg.

‡ For patients who have received at least 24 weeks of treatment with ELREXFIO and have achieved a response

BCMA=B-cell maturation antigen; BICR=Blinded Independent Central Review; CD=cluster of differentiation; CR=complete response; CRS=cytokine release syndrome; IMiD=immunomodulatory drug; IMWG=International Myeloma Working Group; mAb=monoclonal antibody; MM=multiple myeloma; ORR=objective response rate; PFS=progression-free survival; PI=proteasome inhibitor; PR=partial response; QW=once weekly; Q2W=once every 2 weeks; SARS-CoV-2=severe acute respiratory syndrome coronavirus 2; sCR=stringent complete response; VGPR=very good partial response.

References: 1. Kumar S, Paiva B, Anderson KC, et al. International Myeloma Working Group consensus criteria for response and minimal residual disease assessment in multiple myeloma. *Lancet Oncol.* 2016;17:e328-e346. **2.** ELREXFIO (elranatamab) Hong Kong Prescribing Information (ver Mar 2024). **3.** Pfizer Data on File, REF-L1A0405. **4.** Pfizer Data on File, REF-L1A0406. **5.** Pfizer Data on File, REF-L1A0404. **6.** Lesokhin A. et al. Elranatamab in *Nature Medicine.* 2023;29:2259-2267

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ABSTRACTS

Lecture 6

Modern Management of Polycythemia Vera – Ropeginterferon Alfa-2b Demonstrates Long-Term Disease Improvements

Prof. Kazuya SHIMODA
Professor & Chairman,
Division of Hematology, Diabetes, and Endocrinology,
Department of Internal Medicine,Faculty of Medicine,
University of Miyazaki
Japan

Abstract

Polycythemia vera (PV) is a subgroup of myeloproliferative neoplasms characterized by uncontrolled malignant proliferation of hematopoietic cells, leading to an increased number of red blood cells, often accompanied by elevated leukocyte and platelet counts. The oncogenic driver mutation JAK2Val617Phe is observed in approximately 95% of patients.

The therapeutic goals of PV include managing PV-related symptoms, preventing thrombotic and hemorrhagic events, and reducing the risk of disease progression to myelofibrosis (MF) and acute myeloid leukemia (AML). Historically, many PV treatment guidelines have placed greater emphasis on thrombotic risk, categorizing patients into low- and high-risk groups based on age and prior thrombotic events. For all patients with PV, current guidelines recommend phlebotomy and low-dose aspirin as standard therapy. In addition, cytoreductive therapy is indicated for high-risk patients. In patients with PV requiring cytoreductive therapy, ropeg-IFN-α demonstrated a higher complete hematological response rate than HU. Furthermore, the median JAK2VF allele burden continuously declined during treatment with ropeg-IFN-α. Accordingly, ropeg-IFN-α is now considered a first-line therapy for patients with high-risk PV. The efficacy of ropeg-IFN-α was also reported in low-risk PV patients. The maintenance of a median HCT value of 45% or less for 12 months without disease progression was achieved in 81% of patients in the ropeg-IFN-α group, compared to 51% in the phlebotomy group. The JAK2VF allele burden decreased by 11.9% in the ropeg-IFN-α group, while remaining largely unchanged in the phlebotomy group.

Nearly 30% of patients with PV have a risk of progression to myelofibrosis regardless of their thrombotic risk. According to the long-term observation study, the probability of event-free survival, defined as the absence of thromboembolic events, disease progression, or death, was significantly higher in the ropeg-IFN-α group compared with the control group. The Cox proportional hazards ratio was 0.34, indicating a substantial reduction in risk with ropeg-IFN-α. These fewer clinical events seemed to be associated with a reduction in JAK2VF allele burden. Patients who achieved a molecular response (an undetectable JAK2VF allele burden, or a reduction of 50% or more in those with a baseline burden less than 50%, or a reduction of 25% or more in those with a baseline burden greater than 50%) experienced fewer events than those who did not.

Ropeg-IFN-α is the treatment of choice for high-risk PV and for low-risk PV patients requiring cytoreductive therapy to reduce thromboembolic risk. Some patients achieve a significant reduction in JAK2VF allele burden with ropeg-IFN-α, and this molecular response may correlate with fewer events, including progression to MF or AML, or death. We may now be at a turning point, shifting the therapeutic goal for PV from thrombosis prevention to event reduction, including disease progression and mortality.

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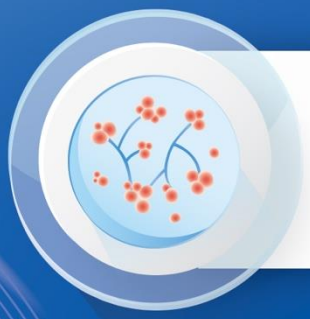
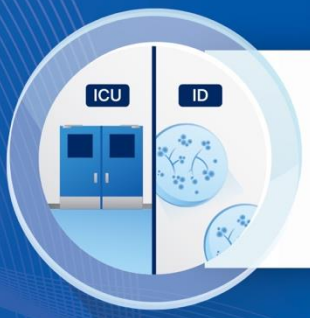
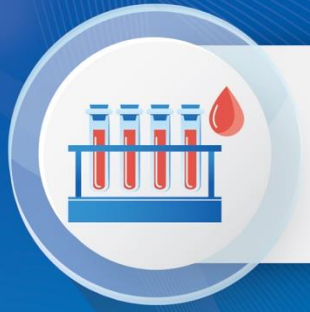


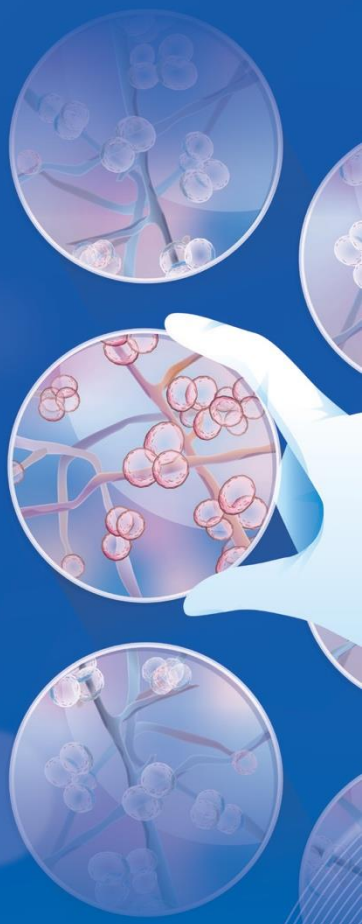
Exhibits excellent antifungal activities against *Candida* species[3]



Proven efficacy and tolerability against invasive fungal infections in ICU/ ID patients[4]



Proven efficacy in the treatment of invasive candidiasis and antifungal prophylaxis in patients with haematologic disorders[5]



ICU=intensive care unit; ID=infectious diseases.

References: 1. MYCAMINE® Hong Kong prescribing information. Oct 2022. **2.** Chen Q, et al. China Med J 2012;125:345-51. **3.** Pfaller MA, et al. J Antibiotics 2015;68:556-561 **4.** Tismit J-F, et al. Mycoses 2020;63:443-451. **5.** Kotsopoulou M, et al. Infect Dis Ther 2019;8:255-268.

MYCAMINE For Injection 50 mg (micafungin)
Indications Treatment of Patients with Candidemia, Acute Disseminated Candidiasis, *Candida* Peritonitis and Abscesses. Treatment of Patients with Esophageal Candidiasis. Prophylaxis of *Candida* Infections in Patients Undergoing Hematopoietic Stem Cell Transplantation. **Dosage and administration** Dose in adult patients Treatment of Candidemia, Acute Disseminated Candidiasis, *Candida* Peritonitis and Abscesses: 100 mg once daily. Treatment of Esophageal Candidiasis: 150 mg once daily. Prophylaxis of *Candida* Infections in HSCT Recipients: 50 mg once daily. Dose in pediatric patients 4 months or older Treatment of Candidemia, Acute Disseminated Candidiasis, *Candida* Peritonitis and Abscesses: 2 mg/kg once daily (maximum daily dose 100 mg). Treatment of Esophageal Candidiasis: 3 mg once daily for patients ≤30 kg; 2.5 mg/kg once daily (maximum daily dose 150 mg) for patients >30 kg. Prophylaxis of *Candida* Infections in HSCT Recipients: 1 mg/kg once daily (maximum daily dose 50 mg). Administer by intravenous infusion only. Infuse over 1 hour. **Contraindications** Known hypersensitivity to micafungin, any component of Mycamine, or other echinocandins. **Warnings and precautions** Hypersensitivity reactions. Haematological effects. Hepatic effects. Renal effects. **Drug Interactions** Sirolimus, nifedipine, itraconazole. **Use in specific populations** Pregnancy Pregnancy Category C. Mycamine should be used during pregnancy only if the potential benefit justifies the potential risk to the fetus. Nursing Mothers Caution should be exercised when Mycamine is administered to a nursing woman. Pediatric Use Safety and effectiveness in pediatric patients younger than 4 months of age have not been established. Geriatric Use No dose adjustment is necessary. Renal Impairment No dose adjustment required. Hepatic Impairment Dose adjustment is not required in patients with mild, moderate, or severe hepatic impairment. **Adverse reactions** Occurring ≥5% in adult patients with Candidemia and other *Candida* infections: Diarrhea, nausea, vomiting, hypoglycemia, hypernatremia, hyperkalemia, pyrexia, blood alkaline phosphatase increased, atrial fibrillation. Occurring ≥5% in adult patients with Esophageal Candidiasis: Diarrhea, nausea, vomiting, pyrexia, headache, phlebitis, rash. Occurring ≥5% in adult patients during prophylaxis of *Candida* infections in HSCT recipients: Diarrhea, nausea, vomiting, abdominal pain, neutropenia, thrombocytopenia, rash, headache, insomnia, anxiety, tachycardia.
Refer to full package insert (Oct 2022) for complete information.

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01-2025-HK-MYCA-PRL-992-V1

ABSTRACTS

Lecture 7

Latest Advances in the Management of Complement-Mediated Disease

Dr. Talha MUNIR
Consultant Haematologist,
St. James's Hospital,
Leeds,
United Kingdom

Abstract

Complement-mediated diseases comprise a diverse group of disorders caused by dysregulation of the complement system, a key component of innate immunity. Recent advances have transformed their management, shifting from non-specific immunosuppression to targeted complement inhibition, leading to improved disease control and reduced morbidity. Novel complement inhibitors now target multiple points in the cascade, including C5, C3, factor B, factor D, and the lectin pathway, enabling more precise and individualized therapy across conditions such as paroxysmal nocturnal haemoglobinuria (PNH), atypical haemolytic uremic syndrome, C3 glomerulopathy, and neuromyelitis optica spectrum disorder. Long-acting agents and subcutaneous formulations have further enhanced treatment convenience and adherence.

This talk will focus on the role of complement inhibitors in the management of these disorders, with particular emphasis on PNH. It will review the fundamentals of PNH management and highlight key considerations in selecting the most appropriate complement inhibitor for different clinical scenarios.

ALTUVIIIO®

Antihemophilic Factor (Recombinant),
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MORE DAYS NEAR NORMAL

FACTOR VIII ACTIVITY LEVELS

Once-weekly ALTUVIIIO is a first-in-class, high-sustained Factor VIII replacement therapy that provides normal to near-normal levels (>40%) for most of the week in adults.[1]

Presentation: ALTUVIIIO (efanesoctocog alfa) powder and solvent for solution for injection. **Indications:** For use in adults and children with hemophilia A for routine prophylaxis to reduce frequency of bleeding episodes, or on-demand treatment and control of bleeding episodes, or perioperative management of bleeding. **Dosage and Administration:** For intravenous use after reconstitution only. Routine Prophylaxis: 50 IU/kg administered once weekly. On-demand Treatment and Control of Bleeding Episodes: Single dose of 50IU/kg. For minor and moderate bleeding episodes occurring within 2 to 3 days after a prophylactic dose, a lower dose of 30 IU/kg dose may be used. Additional doses of 30 or 50 IU/kg every 2 to 3 days may be considered. For major bleeding episodes, additional doses of 30 or 50 IU/kg every 2 to 3 days can be considered. Recommend to allow at least 72 hours between the last 50 IU/kg dose for treatment of a bleed and resuming prophylaxis dosing. Prophylaxis can be continued as usual thereafter. Perioperative Management: Single dose of 50IU/kg. For minor surgery, an additional dose of 30 or 50 IU/kg after 2 to 3 days may be considered. For major surgery, additional doses of 30 or 50 IU/kg every 2 to 3 days may be administered as clinically needed. *For full dosage information, please refer to the full prescribing information.* **Contraindications:** Severe hypersensitivity reactions, including anaphylaxis, to the product or its excipients. **Precautions:** Inform patients of signs of hypersensitivity reactions that may progress to anaphylaxis. Advise patients to discontinue use of ALTUVIIIO if hypersensitivity symptoms occur and contact a physician and/or seek immediate emergency care. Monitor all patients for development of Factor VIII inhibitors by appropriate clinical observations and laboratory tests. Perform appropriate testing if the patient's plasma Factor VIII level fails to increase as expected or if bleeding is not controlled after ALTUVIIIO administration. Recommend to use a validated one-stage clotting assay if assessment of plasma Factor VIII activity is needed. In patients with existing cardiovascular risk factors, substitution therapy with Factor VIII may increase cardiovascular risk. If a central venous access device (CVAD) is required, risk of CVAD-related complications should be considered. **Drug Interactions:** None. **Pregnancy and lactation:** Not known whether ALTUVIIIO can affect reproductive capacity or cause fetal harm when given to pregnant women. Consider the developmental and health benefits of breastfeeding along with the mother's clinical need for ALTUVIIIO and any potential adverse effects on the breastfed infant from ALTUVIIIO or from the underlying maternal condition. **Undesirable Effects:** Headache, arthralgia, pain in extremity, back pain, pyrexia, vomiting. *For other undesirable effects, please refer to the full prescribing information.* **Preparation:** 1 x 500IU or 3000IU ALTUVIIIO vial, with 1 pre-filled syringe with solvent and 1 vial adapter. **Legal Classification:** Part 1, Schedule 1 & Schedule 3 Poison

Full prescribing information is available upon request.

Reference: 1. Drygalski AV, et al. N Engl J Med. 2023;388(4):310-318.

API-HK-ATV-24.10

MAT-HK-2500524-1.0-10/2025

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ABSTRACTS

Lecture 8

The Evidence-based Management of CML: An Asian Perspective

Dr. Takeshi KONDO
Deputy Director,
Aiiku Hospital,
Sapporo,
Japan

Abstract

In this lecture, Dr. Kondo will present a comprehensive Asian-perspective analysis of the real-world challenges and emerging strategies in the treatment of frontline chronic myeloid leukemia (CML), drawing on his clinical experience in Japan. Despite significant advancements in the management of CML with tyrosine kinase inhibitors (TKIs), there remain substantial unmet needs in achieving optimal patient outcomes. One critical issue is the tolerability of current treatment options, which can prevent patients from achieving long-term efficacy due to emergence of adverse events over time.

Dr. Kondo will discuss the latest evidence from regional studies and clinical trials, highlighting the disparities in treatment outcomes compared to Western populations. The evolving treatment landscape, including the potential role of allosteric TKIs or STAMP inhibitors, combination therapies, and personalized medicine approaches, will be explored. Special attention will be given to the management of adverse effects and strategies to improve patient adherence and quality of life.

This lecture aims to equip haematologists with a deeper understanding of the current gaps in CML management within the Asian context and to provide insights into innovative approaches that could be translated into clinical practice to enhance patient care.

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Active Ingredient: Brentuximab vedotin **Indication:** Treatment of adult patients with previously untreated CD30+ Stage III or IV Hodgkin lymphoma (HL) in combination with doxorubicin, vinblastine and dacarbazine (AVD); Treatment of adult patients with CD30+ HL at increased risk of relapse or progression following ASCT; Treatment of adult patients with relapsed or refractory CD30+ Hodgkin lymphoma (HL) following autologous stem cell transplant (ASCT) or at least 2 prior therapies when ASCT or multi-agent chemotherapy is not a treatment option; In combination with cyclophosphamide, doxorubicin and prednisone (CHP) for the treatment of adult patients with previously untreated systemic anaplastic large cell lymphoma (sALCL); Treatment of adult patients with relapsed or refractory sALCL; Treatment of adult patients with CD30+ cutaneous T-cell lymphoma (CTCL) after at least 1 prior systemic therapy. **Dose & Administration:** Previously untreated HL: In combination with chemotherapy (doxorubicin [A], vinblastine [V] and dacarbazine [D] [AVD]), 1.2mg/kg IV infusion over 30 min on days 1 and 15 of each 28-day cycle for 6 cycles. HL at increased risk of relapse or progression following ASCT & CTCL after at least 1 prior systemic therapy: 1.8 mg/kg IV infusion over 30 min every 3 wk up to max of 16 cycles. Previously untreated sALCL: In combination with chemotherapy (cyclophosphamide [C], doxorubicin [H] and prednisone [P] [CHP]), 1.8 mg/kg IV infusion over 30 minutes every 3 weeks for 6 to 8 cycles. Relapsed or refractory HL & relapsed or refractory sALCL: 1.8 mg/kg IV infusion over 30 min every 3 wk, patients who achieve stable disease or better should receive a minimum of 8 cycles and up to a max of 16 cycles. **Contraindications:** Hypersensitivity to brentuximab vedotin or the excipients. Combined use of brentuximab & bleomycin. **Special Population:** Closely monitor for new or worsening neurological, cognitive or behavioural signs or symptoms suggestive of progressive multifocal leukoencephalopathy (PML); new or worsening abdominal pain suggestive of acute pancreatitis; new or worsening pulmonary symptoms; emergence of serious & opportunistic infections; immediate & delayed infusion-related reactions. Discontinue use if anaphylaxis & Stevens-Johnson syndrome occurs. Patient w/rapidly proliferating tumour & high tumour burden at risk of tumour lysis syndrome. Monitor for symptoms of neuropathy. Patient experiencing new or worsening peripheral neuropathy may require delay & dose reduction or discontinuation of treatment. Monitor CBC prior to therapy; serum glucose. Patient w/ an elevated BMI w/ or w/o history of DM; renal & hepatic impairment; on controlled Na-diet. Women of childbearing potential should use 2 methods of contraception during & until 6 months after therapy. Men should not father a child during therapy & for up to 6 mth after last dose. May affect ability to drive or operate machinery. Childn & elderly. **Adverse Reactions:** Infection, sepsis/septic shock, upper resp tract infection, herpes zoster, pneumonia, herpes simplex, oral candidiasis; neutropenia, anaemia, febrile neutropenia, thrombocytopenia; Decreased appetite, hyperglycaemia; peripheral sensory neuropathy, peripheral motor neuropathy, dizziness; cough, dyspnoea; diarrhoea, nausea, vomiting, constipation, abdominal pain, stomatitis; elevation of ALT/AST; alopecia, pruritus, rash; myalgia, arthralgia, back pain, bone pain; fatigue, pyrexia, infusion-related reactions, chills.

For detailed information, please consult full prescribing information.
For reporting suspected side effects for Takeda products at AE.HongKong@takeda.com
For asking medical information and other inquiries for Takeda products at medinfohk@takeda.com

Reference: 1* Adcetris Package Insert, EU-OCT2023 - HK-JAN2024

C-APROM/HK/ ADCE/0054 (10/2025)

ABSTRACTS

Lecture 9

The Ongoing Debate on IV Iron in Iron Deficiency Anaemia

Dr. James UPRICHARD
Consultant Haematologist,
St George's Hospital,
London
United Kingdom

Abstract

The ongoing debate regarding IV iron centers on its transition from a "last resort" to a primary intervention in ID management. Modern IV formulations effectively bypass hepcidin-mediated gut blockade, which frequently limits oral iron efficiency in inflammatory conditions like IBD, CKD, and Heart Failure. Unlike oral therapy, which suffers from non-compliance due to GI distress and slow response, IV iron provides rapid, highly effective iron repletion in a single session. While historical fears of anaphylaxis have been mitigated by the favourable safety profiles of newer IV Irons, current clinical focus has shifted to managing specific side effects like FGF23-mediated hypophosphatemia seen in certain IV iron formulations. Ultimately, the debate is increasingly settled by value-based economics from both a healthcare resource and patient perspective. The higher upfront cost of IV iron is offset by reducing blood transfusions, minimizing infusion frequency through high-dose single-delivery systems, reducing burden of monitoring and treating hypophosphatemia and lowering hospital readmissions.

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COLUMVI® as monotherapy is indicated for the treatment of adult patients with R/R DLBCL, after two or more lines of systemic therapy[1]

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STRONG response rates achieved in 3L+ DLBCL population[3]

- 40% CR and 52% ORR

RAPID and DURABLE remissions after treatment completion[3]

- Median time to first CR of 42 days
- 29.8 months mDoCR

FIXED DURATION treatment[3]

- Convenient Q3W dosing intervals
- A maximum of 12 cycles administered over ~8.3 months*

*Treatment with COLUMVI is recommended for a maximum of 12 cycles, until disease progression or unmanageable toxicity. Each cycle is 21 days.

Abbreviations
3L+, third line or later; **R/R DLBCL,** relapsed or refractory diffuse large B-cell lymphoma; **CR,** complete response; **ORR,** overall response rate; **mDoCR,** median duration of complete response; **Q3W,** every 3 weeks

References
1. COLUMVI® Hong Kong Product Information.
2. Dickinson MJ, et al. N Engl J Med. 2022. 387(24):2220-2231.
3. Dickinson MJ, et al. Oral presentation presented at the 2024 ASH Annual Meeting; San Diego, CA. 7-10 December 2024.

Disclaimer: For Healthcare Professionals Only.

The above information may cover experimental or other uses of drugs that are not currently approved in Hong Kong, which is intended for scientific knowledge exchange only.

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ABSTRACTS

Young Fellow Presentation

Phenotypic and Genotypic Features of von Willebrand Disease in Hong Kong

Dr. Mathew CHEUNG Tsz Long
Department of Pathology,
United Christian Hospital

Abstract

Background: While the molecular pathogenesis of von Willebrand disease (VWD) is well-studied, the genetic landscape of VWD in the Hong Kong population is less clear.

Methods: 71 patients from 64 families were enrolled. A multitude of VWF assays were performed. While whole exome sequencing was performed for the newly recruited patients, previous genetic data generated by targeted NGS gene panels or Sanger sequencing were also included for analysis.

Results: Majority of the patients had either type 1 VWD / low VWF (41%), or type 2 VWD (52%). 45 VWF variants, including seven novel variants, were detected in 41 patients. Three novel variants, namely VWF p.C1165Y, p.L1384P and p.A1461T, were classified as likely pathogenic for type 2 VWD. Type 2 VWD showed good genotype-phenotype correlation, but the correlation in type 1 VWD was less clear. Negative VWF genotyping results provided diagnostic clues to alternative diagnoses including haemophilia A and acquired von Willebrand syndrome. Some issues regarding the phenotypic and genotypic diagnosis of VWD were observed.

Conclusion: The phenotypes of the Hong Kong patients with novel variants may provide insights into the complex pathogenesis of VWD. Moreover, the role of genetic tests in enabling correct diagnosis in a simplified manner is highlighted.

ABSTRACTS

Young Fellow Presentation – Outstanding New Fellow

Real-World Outcome of Eltrombopag-Containing Regimens as Frontline Treatment for Aplastic Anaemia: A Multi-Center Retrospective Study in Hong Kong

Dr. Stephen LAM Sze Yuen
School of Clinical Medicine,
The University of Hong Kong

Abstract

Background: Eltrombopag (EPAG), an oral thrombopoietin receptor agonist, has emerged as a promising frontline treatment for aplastic anemia (AA). It improves the overall response rate and shortens the time to response with a favorable safety profile. Combined with immunosuppressive therapy, EPAG has become a standard first-line treatment. However, real-world data on its efficacy and long-term outcomes are limited. This study represents one of the largest real-world datasets to date on long-term outcomes of Asian AA patients treated with EPAG-based regimens.

Methods: A multi-center retrospective study of adult patients with therapy-naïve acquired AA treated with EPAG-containing regimens in 7 hospitals in Hong Kong over a 10.5-year period (January 2014 to July 2024) was conducted. The Camitta criteria[1] for defining disease severity were adopted. Hematologic response, failure-free survival (FFS), and overall survival (OS) were evaluated. Partial response (PR) was defined as recovery of blood parameters above and beyond MAA/SAA, and complete response (CR) was defined by neutrophil count ≥1.0 x 109/L, platelet count ≥100 x 109/L, and hemoglobin ≥10 g/dL. Overall response (OR) was the composite of PR and CR. The outcome was correlated with clinicopathologic characteristics, including disease severity, EPAG dosing, and time to treatment initiation.

Results: There were 118 patients (44 men and 74 women) with AA (moderate, MAA, N=18; severe, SAA, N=66; very severe, vSAA, N=34), at a median age of 57 (18-87) years. A paroxysmal nocturnal hemoglobinuria clone was detected in 40% of patients with available information (N=107). Treatment regimens included EPAG (N=8), EPAG+cyclosporin A (CsA) (N=58) and EPAG+CsA+antithymocyte globulin (ATG) (N=52).

For MAA patients, most were treated with EPAG+CsA (N=14). The OR rate was 93% (CR, N=57%; PR, N=36%). At a median follow-up of 67 (5-121) months, the 5-year FFS and OS were 65% and 80% respectively. For SAA/vSAA, most were treated with EPAG+CsA (N=44) and EPAG+CsA+ATG (N=50). The OR was 66% (CR, N=48%; PR, N=18%). At a median follow up of 57 (1-132) months, The 5-year FFS and OS were 70% (95% confidence interval, CI: 58-84%) and 67% (95% CI: 57-78%). EPAG+CsA+ATG, compared with EPAG+CsA, resulted in higher CR (60% versus 34%, P=0.014); higher OR (76% versus52%, P=0.019); shorter time to CR (6.5 versus 11.2 months, P=0.011); comparable time to first response (2.7 versus 2.6 months, P=0.2); comparable 5-year FFS (58% versus 78%, P=0.2); and superior 5-year OS (not reached versus 5.5 years, P=0.0006). 5 patients received allogeneic haematopoietic stem cell transplantation (HSCT) at partial response (N=1), or due to no response (N=2) or relapse (N=2). For SAA/vSAA, delay in treatment (time from diagnosis to treatment initiation ≥25 days) was associated with inferior OS (Hazard ratio, HR 2.37, P=0.03). For patients achieving CR, the OS was >90% at 7 years. Multivariate analyses showed inferior OS to be associated with age at diagnosis >65 years (HR 7.0, P=0.03); achieving PR only (HR 8.6, P=0.01); and no response to treatment (HR 24, P<0.001).

As a lower EPAG dose is recommended for Asian patients, the dosage of EPAG used and its impact were investigated. The median duration of treatment with EPAG-containing regimen was 25 months. The dosage of EPAG ranged from 25 mg to 200 mg daily. The mean daily dose of EPAG in the first month of treatment was 50 mg and 75 mg in 36% and 29% of patients respectively. There was no dose-dependent improvement of CR or OR observed in MAA/SAA/vSAA patients.

Conclusions: EPAG-containing regimens, particularly EPAG+CsA+ATG, demonstrated high efficacy in treatment-naïve AA patients. Achieving CR predicted superior survival outcomes. Early treatment initiation might benefit AA patients. These findings highlight the need for prospective trials to optimise EPAG-based strategies. Finally, prospective comparison with frontline allogeneic HSCT is also warranted. Genomic studies are ongoing to investigate biomarkers that are predictive of disease or treatment outcome.

ABSTRACTS

Young Fellow Presentation

Evaluation of Oxford Nanopore Sequencing in Rapid AML Genomic Profiling

Dr. Harry LAU Ka Ngai
Department of Clinical Pathology,
Tuen Mun Hospital

Abstract

Background: Risk stratification and clinical management for acute myeloid leukemia (AML) relies heavily on its genomic profiling. Conventional cytogenetic study is currently the cornerstone in AML genomic profiling but has limitations. Whole genome sequencing (WGS) has been shown to provide greater diagnostic yield and more efficient risk stratification. Oxford Nanopore Sequencing has the advantage of identifying structural variations (SVs) while providing copy number variations (CNVs), single nucleotide variants (SNVs), indels and methylation detection simultaneously. Recently, a methylation-based classification of acute leukemia by sparse sequencing data (MARLIN) was developed by the Dana-Farber Cancer Institute which provides rapid acute leukemia classification within 2 hours of nanopore sequencing.

Aims: To evaluate the clinical utility of Nanopore Sequencing with adaptive sampling and methylation-based classification in providing rapid epigenomic profiling for AML patients.

Methods: DNA was extracted from bone marrow samples from 11 patients with AML and a patient with mixed phenotype acute leukemia (MPAL), B/myeloid. Nanopore sequencing with adaptive sampling targeting 800 regions was performed with Promethion P2 Solo device using FLO-PRO114M flow cells. SVs, CNVs and myeloid panel SNVs and indels were detected by SVIM, EPI2ME and DeepVariant tools respectively. The results were integrated and compared with the cytogenetic and myeloid NGS results and targeted RNA-sequencing in selected cases at diagnosis. Modified basecalling with detection of 5mC and 5hmC bases at CpG sites was performed retrospectively by Dorado. Methylation calls at the 357,340 CpG sites from the MARLIN reference cohort were extracted using modkit. The MARLIN classifier with a prediction threshold of 80% was utilized to predict their respective methylation classes.

Results: The fastest time from DNA extraction to reporting of genomic profile took 24 hours. Mean coverage at the target regions was 23.3X. All 5 cases with clinically significant chromosomal translocations, such as CBFB::MYH11, RUNX1::RUNX1T1, NUP98::NSD1, MECOM rearrangement and KMT2A-partial tandem duplication (PTD) were detected. All CNVs events (>5Mbs) detected were concordant. A clonal evolution event del(13)(q12q14) was missed. For myeloid panel SNVs and indels detection, sensitivity and specificity were 91.7% (22/24) and 100% respectively. The two variants not detected were CEBPA b-ZIP domain in-frame deletion variant and subclonal PTPN11 missense variant. For FLT3-ITD, 2/4 variants were detected. Additional clinically significant findings were detected by nanopore sequencing in 3 cases, namely NUP98::NSD1 fusion, Xp11.4 microdeletion and KMT2A-PTD. MARLIN successfully predicted methylation classes in 10/12 cases. The predicted methylation classes in 6 cases were highly concordant to the genetic driver alterations. In case AML2, the predicted CEBPA methylation class complemented the missed CEBPA b-ZIP variant by nanopore sequencing. The HOX Group 4 was genetically heterogeneous and required further genomic profiling to identify any HOX-activating driver mutations such as NUP98::NSD1. MARLIN failed to predict methylation class in two cases, namely AML with atypical MECOM rearrangement and MAPL, B/myeloid with complex karyotype and KMT2A amplification.

Summary / Conclusion: Nanopore sequencing with adaptive sampling complemented with MARLIN methylation class prediction can rapidly provide comprehensive epigenomic profiling for AML patients.

ABSTRACTS

Young Fellow Presentation

Clinical Outcome of 4-factor Prothrombin Complex Concentrate (4-F PCC) for Factor Xa-inhibitor Reversal: A Multi-centre Retrospective Review

Dr. Lala SIN Yuen Ting
Department of Medicine & Geriatrics,
Tuen Mun Hospital

Abstract

Background – Acute major bleeding is a severe and potentially fatal complication in patients receiving factor-Xa inhibitors (FXaI) therapy. Andexanet alfa was approved by the Food and Drug Administration (FDA) as specific reversal agent for managing such condition, yet the cost per reversal is expensive and it is not yet available in Hong Kong. Off-label use of 4-factor prothrombinex complex concentrate (4-F PCC) is still our current practice for managing FXaI-associated major bleeding. This retrospective multi-centre review aims to evaluate the efficacy and safety outcomes of 4-F PCC in FXaI reversal for acute major bleeding.

Methods – Adult patients aged 18 or above admitted to 3 hospitals in the New Territories West Cluster (NTWC) during 9/2020 to 8/2023 who were on FXaI and received 4-F PCC (i.e. Beriplex) for anticoagulation reversal due to acute major bleeding were reviewed. Primary outcome is all-cause 30-day mortality. Secondary outcomes include hemostatic effectiveness, all-cause inpatient mortality, 30-day incidence of thrombosis, length of stay (LOS) in the index admission and change to patients' anticoagulation regimen after index bleeding episode. Effect of different clinical factors in predicting primary outcomes would be evaluated.

Results – 168 patients taking FXaI suffered from acute major bleeding requiring Beriplex reversal were included. The median age of the patient population was 80.5 years and 64.3% were male patients. 86.9% patients were anticoagulated for atrial fibrillation or flutter. 83.3% of them were taking apixaban. The most common type of acute major bleeding in this cohort was intracranial hemorrhage (ICH) (74.4%).

The hemostatic effectiveness, 30-day mortality and inpatient mortality of patients after Beriplex were 66.7%, 32.7% and 39.3% respectively. Beriplex showed consistent hemostatic efficacy across subgroups of different bleeding sites. Hemostatic effectiveness of Beriplex was lower in male patients (61.0% VS 79.1%, OR 0.3, p = 0.0141) and those presented with spontaneous ICH (58.1% VS 87.2%, OR 3.28, 95% CI 1.03 – 10.43, p = 0.0445). Achieving hemostatic effectiveness strongly predicted better prognosis in all mortality outcomes. Patient with ICH had higher 30-day mortality from acute major bleeding (24.8% VS 9.3%, OR 9.16, p = 0.0405). Use of additional hemostatic agents predicted higher inpatient mortality in overall population (66.7% VS 36.6%, OR 3.28, p = 0.0445). Comparing to the worst initial GCS category (scored 3 – 8), GCS scored 9 – 12 predicted a lower 30-day mortality (40.0% VS 75.0%, OR 0.18, p = 0.0315) and the effect was most evident in the subset scored 13 – 15 (15.6% VS 75.0%, OR 0.03, p <0.001).

30-day incidence of arterial/venous thrombosis was 4.8% with all events occurred before resumption of anticoagulation. The median LOS among overall population was 12 days whereas 28.0% of patients were hospitalized for >30 days. Only 33.9% patients were resumed on anticoagulation in either inpatient or outpatient setting after index bleeding episode.

Conclusion – Beriplex demonstrated similar hemostatic effectiveness in FXaI-associated major bleeding as showed by other foreign cohorts, with significantly higher 30-day and inpatient mortalities partly accounted by a very high-risk patient population in our cohort. Direct comparison with clinical outcomes of Andexanet alfa is unlikely meaningful due to heterogenicity in study design and patient population. Future large-scale prospective studies are needed to further compare the performance of these 2 reversal agents. An ultimate cost-effective analysis would need development of more complex decision models.

ABSTRACTS

Young Fellow Presentation

Mycophenolate Mofetil in Chinese Patients with Immune Thrombocytopenia

Dr. Jessica WONG G Kei
Department of Medicine and Therapeutics,
Prince of Wales Hospital

Abstract

Background
Immune thrombocytopenia (ITP) is an acquired immune mediated disease characterized by isolated thrombocytopenia. Patients with suboptimal response to first-line therapy require prompt initiation of second-line treatment to prevent severe bleeding manifestations. Mycophenolate mofetil (MMF), an immunosuppressant used in a wide range of autoimmune conditions has a reasonable safety profile.

Methods
In this single center, retrospective study, Chinese ITP patients treated with MMF in the Prince of Wales Hospital from January 2019 to December 2023 were analyzed. Patients' demographics, response rate, response kinetics, bleeding severity and adverse events were recorded.

Results
A total of 102 patients with ITP were evaluated. Sixty-one (60%) patients achieved response (platelet $\geq 30 \times 10^9$/L and at least 2-fold increase from baseline without bleeding symptoms) with MMF, of whom 41 (67%) patients achieved sustained treatment response without the use of rescue medications. Median time to first response was 6 weeks (range 2-40 weeks) and median duration of response was 68 weeks (range 6-354 weeks). Twelve patients tapered off MMF after achieving complete response and remained treatment-free with MMF. Subgroup analysis showed that a shorter period of time from ITP diagnosis to MMF use was associated with response to MMF ($p < 0.05$). Bleeding events at grade 3 or above were infrequent. Most common adverse events were gastrointestinal discomfort and infection. Neutropenia was reported but was not associated with febrile neutropenia.

Conclusion
This is the largest study providing real-world experience on the use of MMF in Chinese ITP patients. MMF is an effective, safe and readily available medication for ITP patients.

ABSTRACTS

Nursing Symposium

When CAR-T Gets Complicated: Nursing Care That Changes Patient Outcomes

Ms. Katrina DEBOSZ
CAR-T Nurse Practitioner,
Institute of Haematology,
Royal Prince Alfred Hospital
Australia

Abstract

Chimeric antigen receptor T-cell (CAR-T) therapy has transformed outcomes for patients with relapsed and refractory haematological malignancies, but its safety and effectiveness depend on the timely recognition and management of treatment-related toxicities. Cytokine release syndrome (CRS), immune effector cell-associated neurotoxicity syndrome (ICANS), and prolonged cytopenias represent predictable consequences of immune activation, and their severity is closely linked to the timing of clinical recognition and intervention.

Nurses play a critical role across the CAR-T pathway, including baseline assessment, early detection of physiological and neurological change, and coordination of care beyond the treating centre. This lecture will examine the CAR-T patient journey to demonstrate how nursing recognition influences toxicity progression, intervention timing, and recovery, and highlight nursing surveillance as essential to safe and effective CAR-T therapy delivery.

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II	**Uplifting Pre-donation Haemoglobin Cutoff from 11.5 to 12 g/dL for Female Blood Donors in Hong Kong – A Tectonic Change in Practice with over 50 Years of History** *Kong Shun Yin; Chu Chui Yee; Lee Cheuk Kwong*
III	**Chronic Graft-Versus-Host Disease: Real-World Outcomes and Efficacy of Ruxolitinib Compared with Conventional Treatments** *Leung Garret; Sim Joycelyn; Lie Albert; Kwong Yok-Lam; Gill Harinder*
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VI	**A Paradigm Shift in Thalassaemia Screening through the Development of the α-thalassaemia Early Eluting Peak** *Lam Wing Kit; Yuen Carmen Michelle; Tsui Lawrence Lap Chi; Li Ting Hon Stanford; Yeung Vivian Ka Pik; Sin Albert Chun Fung; Wong Tsz Fung; Law Winnie Yim Fong; Fan Christina Pui Ying; Ko Lok Nga; Woo Vivian Hoi Kei; Chan Kit Yu; Chan Tsz Ning; Fan Tze Wing; Too Lok Han; Cheng Chi Keung; Wong Man Ling; Wu Aves Hui Hsuan; Lit Benny Man Wai; Wong Yu Fong; Chan Man Wai; Ip Chun Him; Leung Julia Cheuk Ting; Wong Po Chun; Yuen Kei Ching; Yuen Wang Ho; Wong Hoi Ching; Li Jamilla Wai Yan; Leung Anskar Yu Hung; Cheung Joyce Sin; Chan Natalie Pui Ha; Ng Margaret Heung Ling; Kwong Joyce Hoi Yi; Chow Eudora Yu De; Wong Wai Shan; Leung Kate Fung Shan; Yip Sze Fai*
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FREE PAPERS

Poster Presentation – Abstract I

Reduced Compared with Standard Dose Direct Oral Anticoagulant for Extended Treatment of Venous Thromboembolism: A Systematic Review and Meta-analysis

Cheung, Ka Man Carmen[1]; Carlin, Stephanie[2-3]; Scheier, C. Thomas[2-3]; Yi, Qi-Long[4]; Chan, Noel[2-3]; Bhagirath, Vinai[2-3]; Petropoulos, Jo-Anne[5]; Wong, Siu Ming Raymond[1]; Weitz, I Jeffrey[2,6]; Eikelboom, W John[2-3]

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[6] Thrombosis and Atherosclerosis Research Institute and Hamilton Health Sciences, Hamilton, Ontario, Canada

Abstract

Introduction: Randomized studies suggest reduced-dose direct oral anticoagulants (DOACs) offer similar efficacy to standard-dose regimens for extended venous thromboembolism (VTE) treatment and may reduce bleeding risk. It is unclear whether standard-dose are preferable in certain subgroups.

Methods: We systematically searched MEDLINE, EMBASE, EMCARE, and CENTRAL for randomized trials comparing reduced- with standard-dose DOACs for extended VTE treatment. (INPLASY202550061) Outcomes included recurrent VTE, major bleeding, clinically relevant non-major bleeding (CRNMB), and mortality, analysed in overall populations and key subgroups.

Results: Five trials with a total of 8,781 patients were included: 4,395 received reduced-dose and 4,386 standard-dose DOAC for at least 12 months. Pooled data demonstrated that a reduced dose apixaban (2.5 mg twice daily) or rivaroxaban (10 mg once daily), compared to a standard dose (5 mg twice daily for apixaban, 20 mg once daily for rivaroxaban), resulted in similar rates of recurrent VTE (1.66% vs 1.78%; risk ratio [RR] 0.94, 95% confidence interval [CI] 0.68–1.29). Major bleeding was less frequent with reduced doses (1.16% vs 1.96%; RR 0.62, 95% CI 0.42–0.92), as was CRNMB (5.16% vs 7.00%; RR 0.75, 95% CI 0.63–0.88). Mortality rates were comparable (4.91% vs 5.81%; RR 0.86, 95% CI 0.63–1.17). These results held for high-risk subgroups, including patients with recurrent VTE or active cancer, except that reduced-dose DOACs appeared to lower recurrent VTE risk in males but increase risk in females (p=0.04). Risk of bias was rated “low” in four studies and “some concerns” in one study using the RoB2 tool. Certainty of evidence was moderate for three outcomes and low for one outcome.

Conclusion: In extended VTE treatment, reduced-dose versus standard-dose DOAC therapy is associated with a similar risk of recurrent VTE and lower risk of major and CRNMB, including in high-risk subgroups. A potential interaction with sex warrants further investigation.

FREE PAPERS

Poster Presentation – Abstract II

Uplifting Pre-donation Haemoglobin Cutoff from 11.5 to 12 g/dL for Female Blood Donors in Hong Kong – A Tectonic Change in Practice with over 50 Years of History

Kong Shun Yin[1]; Chu Chui Yee[1]; Lee Cheuk Kwong[1]

[1] Hong Kong Red Cross Blood Transfusion Service, 15 King's Park Rise, Kowloon, Hong Kong

Abstract

Introduction:
Hong Kong Red Cross Blood Transfusion Service (BTS) has adopted 11.5 g/dL as the pre-donation haemoglobin (Hb) cutoff for female donors for over 50 years. Though thalassaemia trait is not uncommon locally, BTS prepared to uplift the pre-donation Hb for female donors from 11.5 to 12 g/dL as a worldwide trend and mitigation measure of iron deficiency related to blood donation.

Aim:
This article aims to discuss the strategies for implementation and review the impact to the whole blood (WB) collection of the change in pre-donation Hb cutoff for female donors.

Method:
WB collection statistics, deferral rate and donation history were retrieved from the blood bank data management information system and reviewed. The total number of WB donations from male and female and the deferral rate of low Hb for female WB donors were compared between Jun to Sep 2024 (11.5 g/dL as cutoff) and Jun to Sep 2025 (12 g/dL as cutoff). Details of female WB donors with Hb 11.5-11.9 g/dL from 1 Jun to 30 Sep 2025 were analysed.

Result:
Preparation phase: Years of effort was paid at multiple levels of governance and operation to overcome the barriers of uplifting pre-donation Hb threshold for female donors

- Various governing bodies of HKRCBTS endorsed the initiative to raise the pre-donation Hb cutoff for female donors but the blood inventory was deeply affected by the local societal change since 2010s and global COVID-pandemics.
- The dilemma was tackled with the iron program for blood donors. Universal oral iron supplementation and ferritin testing in selected risk groups were implemented since 2010s. Territory-wide promotional campaigns to enhance dietary iron intake and iron health awareness were conducted in Hong Kong.
- A gradual improvement in deferral rate of low Hb for female donors (12.0% in 2019 to 9.1% in 2021 to 7.2% in 2023) was observed. Together with a manageable daily loss of 20 donors estimated from previous collection data, it paved the way for HKRCBTS to raise the Hb cutoff for female donor from 11.5 to 12 g/dL on 27 May 2025 while maintain a stable blood supply.

Post-implementation review: From 1 Jun to 30 Sep 2025, there were 29606 female donors and 1985 (6.7%) of them were deferred for Hb level of 11.5-11.9 g/dL. The mean deferral rate of low Hb for female WB donors from Jun to Sep 2024 and from Jun to Sep 2025 were 8% and 11.47% respectively ($p < 0.05$). Although there was a daily loss of 16 donors on average due to uplift of pre-donation Hb cutoff for female donors, the total number of WB collection was not significantly affected (16095-18910 units from Jun to Sep 2024 vs. 14437-17018 units from Jun to Sep 2025, $p = 0.61$).

Among the 1985 deferred female WB donors, 1215 donors (61.2%) had successful WB donation previously and 213/1215 donors had a pre-donation Hb level of 11.5-11.9 g/dL at the time of prior donation. The proportion of donors having successful WB donation within past 6 and 12 months was 65.8% and 88.7% respectively. With a mean time of 42 days from deferral of low Hb to next successful WB donation, 506/1985 female WB donors fulfilled the pre-donation Hb cutoff of 12 g/dL at next WB donation.

Conclusion:
The uplift of pre-donation Hb cutoff for female donors from 11.5 to 12 g/dL did not affect the WB collection by BTS but could prevent female donors with Hb 11.5-11.9 g/dL from iron deficiency related to blood donation.

FREE PAPERS

Poster Presentation – Abstract III

Chronic Graft-Versus-Host Disease: Real-World Outcomes and Efficacy of Ruxolitinib Compared with Conventional Treatments

Leung Garret; Sim Joycelyn; Lie Albert, Kwong Yok-Lam; Gill Harinder

Queen Mary Hospital

Abstract

Introduction
Chronic graft-versus-host disease (cGVHD) remains a major challenge in allogeneic hematopoietic stem cell transplantation (HSCT), affecting 30–50% of recipients and leading to significant morbidity through multi-organ involvement. Approximately 50% of patients develop steroid-refractory disease requiring second-line treatment, but no standard therapy existed until the advent of ruxolitinib, a Janus kinase (JAK) inhibitor. While ruxolitinib demonstrated superior efficacy in the phase 3 REACH3 trial, real-world data comparing its effectiveness with conventional therapies in routine clinical practice settings remain limited.

Methods
We performed a single-centre retrospective analysis of 1,026 adult allogeneic HSCT recipients transplanted between January 2014 and December 2023. Among 374 patients who developed cGVHD requiring systemic treatment, 229 required second-line therapy. Response was assessed using National Institutes of Health criteria. To evaluate the real-world efficacy of ruxolitinib and account for baseline differences between treatment groups, we conducted a propensity score-matched (PSM) analysis comparing ruxolitinib with conventional second-line agents. Covariates matched included donor age, disease-risk index, graft source, conditioning intensity, GVHD prophylaxis, cGVHD severity, and prior glucocorticoid use in first-line therapy.

Results
Among 229 cGVHD patients requiring second-line treatment, 125 (55%) received ruxolitinib and 104 (45%) received conventional agents, primarily thalidomide (54%) and sirolimus (14%). For the whole cohort, severe cGVHD was associated with significantly inferior ORR (odds ratio, OR: 0.13; 95% confidence interval, CI: 0.05–0.30; P<0.001), while ruxolitinib treatment was associated with significantly superior ORR (OR: 4.09; 95% CI 2.26–7.52; P<0.001) (Figure 1A).

In the balanced PSM cohort, 66 patients were included in each of the conventional and ruxolitinib group (Table 1). Ruxolitinib, compared with conventional treatment, led to significantly higher ORR (68% versus 39%, P<0.001), superior failure-free survival (hazard ratio, HR: 0.37; 95% CI: 0.22–0.61; P<0.001) (Figure 1C), higher probability of achieving immunosuppression-free remission (HR: 1.81; 95% CI: 1.04–3.16; P=0.036) (Figure 1D), and decreased need of more than three sequential treatment lines (6% versus 20%, P<0.001).

Furthermore, ruxolitinib compared with conventional treatment showed better response in liver (77% versus 47%; P=0.013) and ocular involvement (68% versus 32%; P=0.021). However, however, pulmonary cGVHD remained poorly responsive (ruxolitinib: 22%; conventional: 13%) (Figure 1E). Grade 3 or higher infection rates were comparable and acceptable between ruxolitinib and conventional treatment (24% versus 23%; P=0.84) (Figure 1F). Overall survival was similar in both groups (HR: 0.90; P=0.78).

Conclusion
Our results showed that ruxolitinib was superior to conventional second-line agents for steroid-refractory cGVHD in ORR, failure-free survival, and successful immunosuppression tapering. These findings support ruxolitinib as the preferred second-line treatment for most patients with cGVHD. However, pulmonary GVHD remains an unmet clinical need, representing an important area for future therapeutic development and investigation.

FREE PAPERS

Poster Presentation – Abstract III

Chronic Graft-Versus-Host Disease: Real-World Outcomes and Efficacy of Ruxolitinib Compared with Conventional Treatments

Leung Garret; Sim Joycelyn; Lie Albert, Kwong Yok-Lam; Gill Harinder

Queen Mary Hospital

Abstract

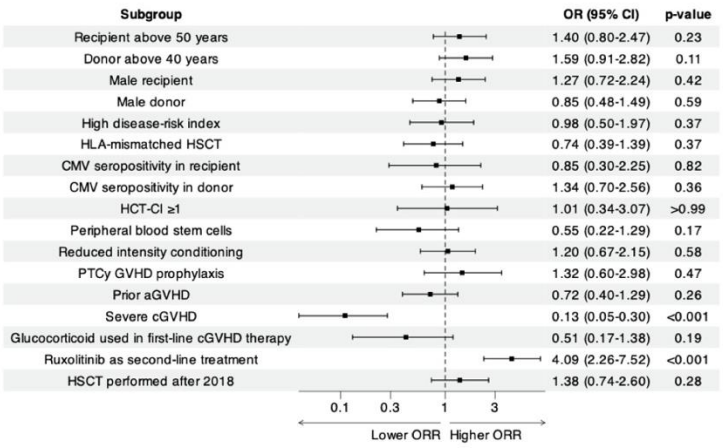
Table 1. Characteristics of Patients Receiving Second-line Chronic GVHD Treatment.

	Unmatched Cohort				Matched Cohort			
	Conventional (N=104)	Ruxolitinib (N=125)	SMD	P-value	Conventional (N=66)	Ruxolitinib (N=66)	SMD	P-value
Age – no. (%)								
Recipient above 50 years	47 (45)	64 (51)	0.120	0.37	31 (47)	34 (52)	0.091	0.60
Donor above 40 years	45 (43)	73 (58)	0.306	0.02	28 (42)	31 (47)	0.092	0.60
Sex – no. (%)								
Male recipient	60 (58)	70 (56)	0.034	0.80	37 (56)	31 (47)	0.183	0.30
Male donor	55 (53)	64 (51)	0.034	0.80	34 (52)	36 (55)	0.061	0.73
High disease-risk index	15 (15)	36 (29)	0.328	0.02	11 (17)	12 (18)	0.040	0.82
HLA-mismatched HSCT	29 (28)	38 (30)	0.055	0.68	19 (29)	12 (18)	0.252	0.15
CMV seropositivity								
Recipient	96 (92)	110 (88)	0.145	0.28	61 (92)	56 (85)	0.240	0.17
Donor	77 (74)	94 (75)	0.027	0.84	47 (71)	49 (74)	0.068	0.70
HCT-CI ≥1	11 (11)	8 (6)	0.150	0.25	6 (9)	2 (3)	0.256	0.27
Peripheral blood stem cells	84 (81)	114 (91)	0.304	0.02	59 (89)	58 (88)	0.048	0.78
Reduced intensity conditioning	31 (30)	57 (46)	0.330	0.01	24 (36)	24 (36)	0.000	>0.99
PTCy GVHD prophylaxis	8 (8)	33 (26)	0.514	<0.001	7 (11)	7 (11)	0.000	>0.99
Prior grade II-IV aGVHD	42 (40)	39 (31)	0.192	0.15	27 (41)	22 (33)	0.157	0.37
Severe cGVHD	35 (34)	8 (6)	0.724	<0.001	7 (11)	7 (11)	0.000	>0.99
Glucocorticoid used in first-line therapy	100 (96)	103 (82)	0.456	0.001	62 (94)	63 (95)	0.040	>0.99

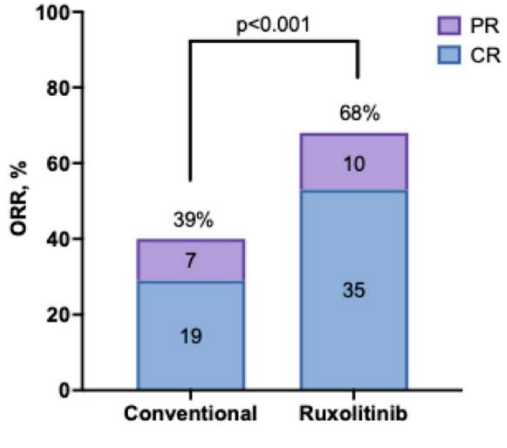
*SMD denotes standardized mean difference.

Figure 1.

A. Odds Ratios of Overall Response in 2L cGVHD Treatment.



B. Overall Response Rates



FREE PAPERS

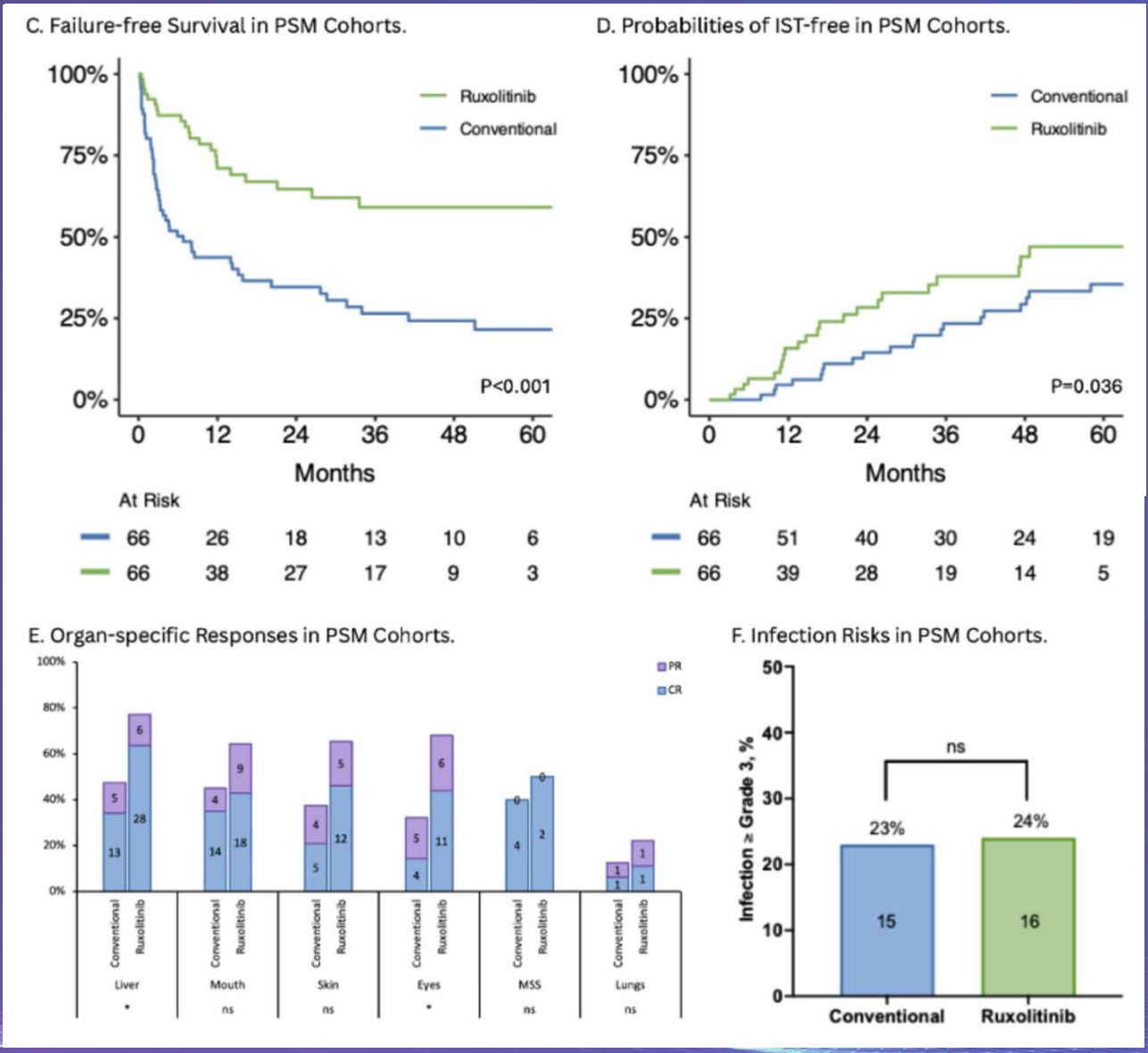
Poster Presentation – Abstract III

Chronic Graft-Versus-Host Disease: Real-World Outcomes and Efficacy of Ruxolitinib Compared with Conventional Treatments

Leung Garret; Sim Joycelyn; Lie Albert, Kwong Yok-Lam; Gill Harinder

Queen Mary Hospital

Abstract



FREE PAPERS

Best Abstract – Abstract IV

Donor Selection and Clinical Outcomes of Allogeneic Haematopoietic Stem Cell Transplantation in Adults Older than 50 Years

Leung Garret; Sim Joycelyn; Lie Albert, Kwong Yok-Lam; Gill Harinder

Queen Mary Hospital

Abstract

Introduction
Optimal donor selection for older patients undergoing allogeneic haematopoietic stem cell transplantation (HSCT) remains controversial. While matched sibling donors (MSD) have traditionally been considered superior, the benefit of younger alternative donors and the impact of post-transplantation cyclophosphamide (PTCy) prophylaxis remain unclear in this population. This study compared clinical outcomes across four donor types in patients older than 50 years.

Methods
We conducted a retrospective analysis of 493 consecutive patients >50 years with malignant haematological diseases undergoing MSD, matched unrelated donor (MUD), mismatched unrelated donor (MMUD), or haploidentical HSCT between January 2014 and December 2023. Patients received either PTCy-based (40mg/kg/day on days +3 to +4 with cyclosporine A and mycophenolate mofetil) or conventional calcineurin inhibitor-based GVHD prophylaxis. Outcome measures included engraftment, graft-versus-host disease (GVHD), non-relapse mortality (NRM), relapse-free survival (RFS), overall survival (OS), and GVHD-free, relapse-free survival (GRFS). Univariate and multivariate analyses identified significant prognostic factors using Cox proportional hazards regression and Fine-Gray models.

Results
Among the 493 patients (median age 57 years, 51% male), 198 received MSD, 101 received MUD, 58 received MMUD, and 136 received haploidentical transplants (Table 1). Patient characteristics differed significantly across groups: haploidentical recipients and MSD donors were older, while MMUD and haploidentical groups had higher disease risk indices. All haploidentical recipients received PTCy prophylaxis and 99% received peripheral blood grafts. Engraftment rates were similar across all donor types (94–99% at day 28). The 180-day incidence of acute GVHD grade II–IV was significantly higher in MMUD (42%) compared with MSD (22%), MUD (31%), and haploidentical (16%) groups (Figure 1A). In multivariate analysis, MMUD and non-PTCy prophylaxis were associated with increased risk of acute GVHD. At 2 years, moderate to severe chronic GVHD incidence was highest in MUD (49%) and MSD (43%) versus MMUD (25%) and haploidentical (17%) (Figure 1B). Risk factors for chronic GVHD included donor age >50 years, female-to-male donation, peripheral blood grafts, and conventional GVHD prophylaxis. NRM was comparable across all groups (9–13% at 2 years). Importantly, haploidentical transplantation showed superior RFS (72% at 2 years, HR 0.62, P=0.016) compared with MSD. Haploidentical also demonstrated significantly better GRFS (58% at 2 years) compared with other groups (23–30%) (Figure 1C). However, this advantage was primarily attributable to PTCy prophylaxis in multivariate analysis. OS was similar across all donor types (62–74% at 2 years). In the high disease risk subgroup, haploidentical showed improved RFS compared with MSD (62% vs 36%, P=0.051) (Figure 1D). Male sex and high disease risk index independently predicted worse survival outcomes.

Conclusion
In patients older than 50 years, MUD transplants with younger donors and conventional GVHD prophylaxis achieved comparable outcomes to older MSD transplants. Notably, haploidentical transplantation with PTCy demonstrated superior RFS and GRFS compared with MSD with conventional prophylaxis, suggesting that alternative donor approaches with optimized GVHD prophylaxis may offer advantages in this older population. Disease risk index remained an important independent predictor of outcomes across all donor types.

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Best Abstract – Abstract IV

Donor Selection and Clinical Outcomes of Allogeneic Haematopoietic Stem Cell Transplantation in Adults Older than 50 Years

Leung Garret; Sim Joycelyn; Lie Albert, Kwong Yok-Lam; Gill Harinder

Queen Mary Hospital

Abstract

Table 1. Demographic and Clinical Characteristic at Baseline.

Characteristic	MSD (N=198)	MUD (N=101)	MMUD (N=58)	Haplo (N=136)
Recipient age – mean ± SD, yr	57 ± 4	57 ± 4	57 ± 4	59 ± 5
Donor age – mean ± SD, yr	55 ± 6	32 ± 7	36 ± 8	34 ± 11
Median follow-up – mo (95% CI)	61 (57-71)	66 (59-82)	65 (42-99)	25 (22-31)
Male sex – no. (%)	101 (51)	49 (49)	29 (50)	73 (54)
Disease – no. (%)				
AML	89 (45)	45 (45)	31 (53)	68 (50)
ALL	31 (16)	15 (15)	13 (22)	18 (13)
MDS	25 (13)	15 (15)	4 (7)	18 (13)
MPN	14 (7)	6 (6)	3 (5)	7 (5)
NHL	16 (8)	9 (9)	6 (10)	14 (10)
Others*	23 (12)	11 (11)	1 (2)	11 (8)
AML – no. (%)				
Favorable	16 (18)	8 (18)	7 (23)	5 (7)
Intermediate	56 (63)	26 (58)	10 (32)	34 (50)
Adverse	17 (19)	11 (24)	14 (45)	29 (43)
ALL – no. (%)				
Ph +ve	9 (29)	7 (47)	5 (39)	10 (56)
Ph -ve	16 (52)	7 (47)	6 (46)	4 (22)
T-ALL	6 (19)	1 (7)	2 (15)	4 (22)
Disease risk index				
Low	32 (16)	15 (15)	9 (16)	12 (9)
Intermediate	138 (70)	65 (64)	29 (50)	89 (65)
High	27 (14)	21 (21)	19 (33)	35 (26)
Very high	1 (1)	0	1 (2)	0
HCT-CI				
≥1 – no. (%)	43 (22)	33 (33)	10 (17)	31 (23)
≥2 – no. (%)	16 (8)	13 (13)	5 (9)	10 (7)
Gender match (donor / recipient) – no. (%)				
Female / Female	46 (23)	25 (25)	12 (21)	24 (18)
Female / Male	46 (23)	11 (11)	8 (14)	27 (20)
Male / Male	55 (28)	38 (38)	21 (36)	46 (34)
Male / Female	51 (26)	27 (27)	17 (29)	39 (29)
CMV status (donor / recipient) – no. (%)				
Pos / Pos	173 (87)	58 (58)	37 (64)	81 (60)
Neg / Pos	11 (6)	39 (39)	14 (24)	49 (36)
Pos / Neg	11 (6)	2 (2)	3 (5)	2 (2)
Neg / Neg	3 (2)	2 (2)	4 (7)	4 (3)
Blood group match – no. (%)				
Bidirectional	16 (8)	17 (17)	10 (17)	7 (5)
Major mismatch	34 (17)	23 (23)	18 (31)	25 (18)
Minor mismatch	31 (16)	30 (30)	17 (29)	22 (16)
No mismatch	117 (59)	31 (31)	13 (22)	82 (60)
Stem cell source – no (%)				
Peripheral blood (PB)	154 (78)	69 (68)	41 (71)	135 (99%)
Bone marrow (BM)	42 (21)	32 (32)	17 (29)	1 (1)
BM + PB	2 (1)	0	0	0
Conditioning intensity – no. (%)				
MAC	100 (51)	39 (39)	25 (43)	27 (20)
RIC	98 (50)	62 (61)	33 (57)	109 (80)
Prior transplantation – no. (%)				
Autologous	3 (2)	4 (4)	0	2 (2)
Allogeneic	1 (1)	4 (4)	2 (3)	6 (4)
GVHD prophylaxis – no. (%)				
PTCy + CsA + MMF	0	0	23 (40)	136 (100)
ShMTX + CsA	198 (100)	0	0	0
ShMTX + CsA + MMF	0	101 (100)	35 (60)	0

*Chronic myeloid leukemia, chronic lymphocytic leukemia, blastic plasmacytoid dendritic cell neoplasm, multiple myeloma, Hodgkin lymphoma and mixed phenotypic acute leukemia.

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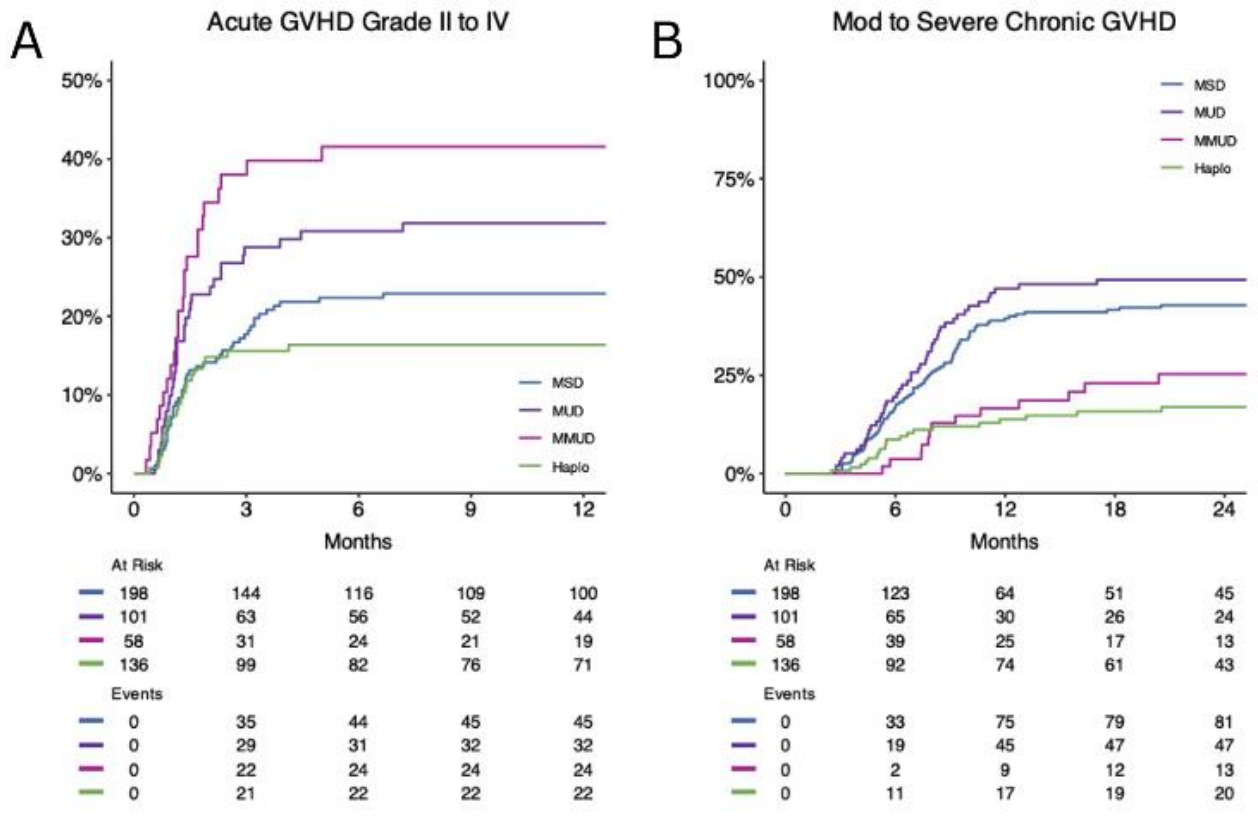
Donor Selection and Clinical Outcomes of Allogeneic Haematopoietic Stem Cell Transplantation in Adults Older than 50 Years

Leung Garret; Sim Joycelyn; Lie Albert, Kwong Yok-Lam; Gill Harinder

Queen Mary Hospital

Abstract

Figure 1. Kaplan-Meier curves of survival by donor types. A. Acute GVHD grade II to IV. B. Moderate to severe chronic GVHD requiring systemic treatments. C. GVHD-free, relapse-free survival. D. RFS of MSD and haplo-HSCT in patients with high or very high DRI.



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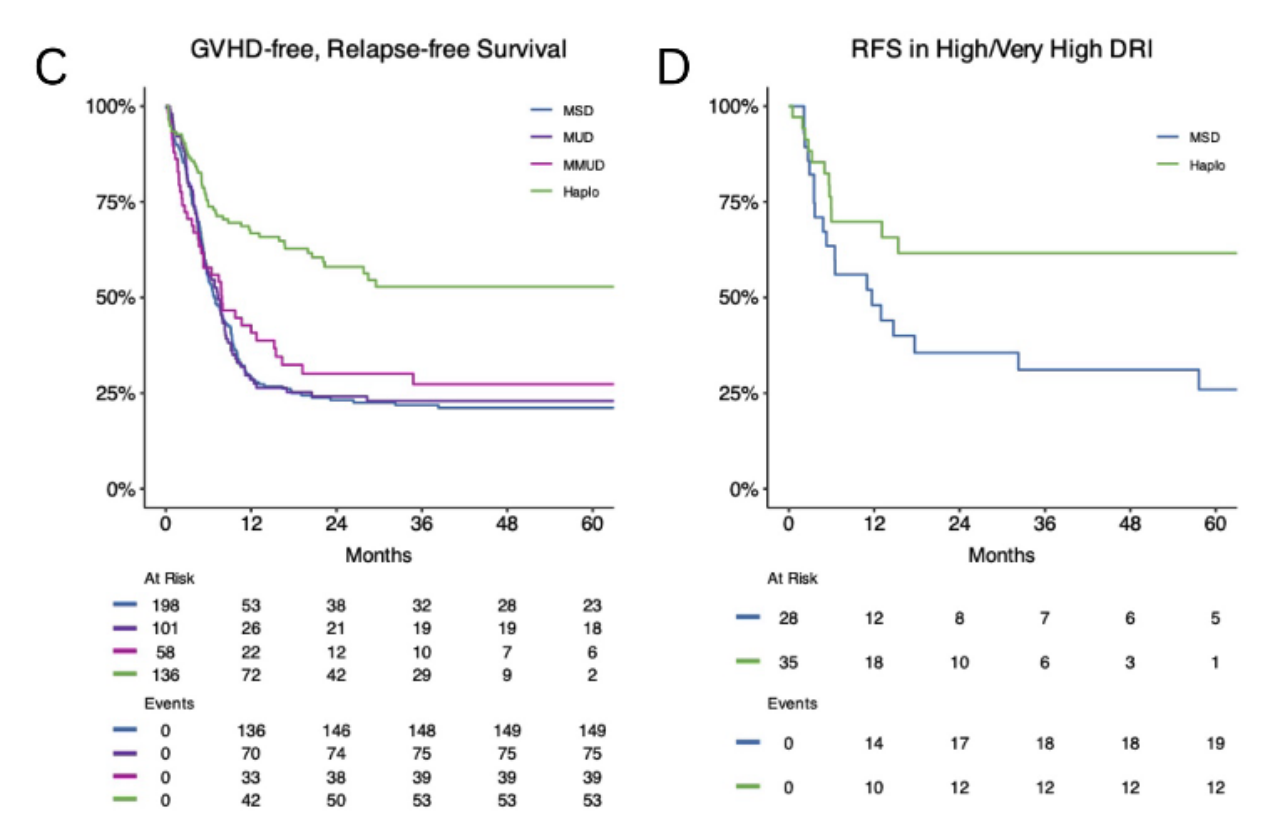
Queen Mary Hospital

Abstract



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Poster Presentation – Abstract V

A Simple Long Polymerase Chain Reaction Third-generation Sequencing Approach for β-globin Genotyping

Lam Wing Kit[1]; Wong Tsz Fung[1]; Ko Lok Nga[1]; Chan Tsz Ning[1]; Fan Tze Wing[1];Yip Sze Fai[1]

[1] Department of Clinical Pathology, Tuen Mun Hospital

Abstract

Introduction: In Hong Kong, the prevalence of β-thalassaemia is about 3%. While phenotypic testing such as high-performance liquid chromatography is standard for detecting β-thalassaemia or haemoglobin (Hb) variants, genotyping is essential for complex cases, rare variants, and prenatal diagnosis.

Objectives: This study aimed to evaluate a long polymerase chain reaction-based Oxford Nanopore Technologies (ONT) third-generation sequencing (TGS) approach for β-globin genotyping.

Methods: For the initial validation, seven peripheral EDTA blood with normal mean corpuscular volume samples were used for assessing the sequencing quality of the ONT TGS, and four cases with known β-thalassaemia mutations for concordance with existing methods. After the initial validation, seven archived cases with diagnostic difficulties or unresolved β-globin chain variants were investigated using the ONT TGS platform. Cost comparisons between ONT TGS and Sanger sequencing were performed.

Results: ONT TGS detected all point mutations and small indels in the cases with known β-thalassaemia mutation status, and potential large deletions were identified by absence-of-heterozygosity (AOH) (Table 1). In the seven archived cases, it identified uncommon variants accounting for the phenotypic findings (Table 2). ONT TGS can offer a 19.3% cost reduction compared to Sanger sequencing with single case per run, and 56.5% cost reduction with batch of five samples per run.

Conclusion: This study showed that the ONT TGS is a promising approach to detect various β-thalassaemia mutations and β-globin variants. The ONT TGS was able to detect uncommon β-thalassaemia mutations and resolve unknown Hb variants. The ONT TGS could also detect AOH through single nucleotide polymorphism or indel analysis. The ONT TGS can be performed using a single long amplicon, which is more cost-effective than Sanger sequencing for covering the same region of interest.

Table 1. The β-thalassaemia mutation status of the known cases and the corresponding ONT TGS results.

Sample	Previously determined mutations	ONT TGS results
Known 1	c.316-197C>T c.85dup	c.316-197C>T c.85dup
Known 2	c.316-197C>T Heterozygous β^0 SEA deletion (by MLPA)	c.316-197C>T Absence-of-heterzygosity
Known 3	c.126_129delCTTT c.-78A>G	c.126_129delCTTT c.-78A>G
Known 4	Negative for β-thalassaemia mutations and β-globin MLPA	No mutation

Abbreviations: MLPA, multiplex probe-dependent ligation amplification; SEA, Southeast Asian; ONT, Oxford Nanopore Technologies; TGS, third-generation sequencing.

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Poster Presentation – Abstract V

A Simple Long Polymerase Chain Reaction Third-generation Sequencing Approach for β-globin Genotyping

Lam Wing Kit[1]; Wong Tsz Fung[1]; Ko Lok Nga[1]; Chan Tsz Ning[1]; Fan Tze Wing[1];Yip Sze Fai[1]

[1] Department of Clinical Pathology, Tuen Mun Hospital

Abstract

Table 2. The HBB genotyping result of the seven archived cases.	
Case 1	Heterozygous β^+ mutation (c.-78A>C)
Case 2	Heterozygous β^0 mutation (c.85dup)
Case 3	Hb G-Taipei (c.68A>G)
Case 4	Hb G-Siriraj (c.22G>A)
Case 5	Homozygous Hb S (c.20A>T)
Case 6	Homozygous β^0 mutation (c.27dup): absence-of-heterozygosity due to consanguinity
Case 7	Heterozygous Hb D-Punjab (c.364G>C)

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Poster Presentation – Abstract VI

A Paradigm Shift in Thalassaemia Screening through the Development of the α-thalassaemia Early Eluting Peak

Lam Wing Kit[1]; Yuen Carmen Michelle[2]; Tsui Lawrence Lap Chi[3]; Li Ting Hon Stanford[4]; Yeung Vivian Ka Pik[5]; Sin Albert Chun Fung[6]; Wong Tsz Fung[1]; Law Winnie Yim Fong[1]; Fan Christina Pui Ying[1]; Ko Lok Nga[1]; Woo Vivian Hoi Kei[1]; Chan Kit Yu[1]; Chan Tsz Ning[1]; Fan Tze Wing[1]; Too Lok Han[1]; Cheng Chi Keung[2]; Wong Man Ling[7]; Wu Aves Hui Hsuan[3]; Lit Benny Man Wai[4]; Wong Yu Fong[4]; Chan Man Wai[4]; Ip Chun Him[5]; Leung Julia Cheuk Ting[5]; Wong Po Chun[6]; Yuen Kei Ching[6]; Yuen Wang Ho[8]; Wong Hoi Ching[8]; Li Jamilla Wai Yan[8]; Leung Anskar Yu Hung[9]; Cheung Joyce Sin[2]; Chan Natalie Pui Ha[2]; Ng Margaret Heung Ling[2]; Kwong Joyce Hoi Yi[3]; Chow Eudora Yu De[3]; Wong Wai Shan[4]; Leung Kate Fung Shan[5]; Yip Sze Fai[1]

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Abstract

Introduction: Although phenotypic screening for β-thalassaemia and other haemoglobinopathies using high-performance liquid chromatography (HPLC) is well-established, phenotypic screening for α0-thalassaemia, especially the locally important Southeast Asian type deletion (--^{SEA}), has been limited by the lack of a reliable test. To address this, we pioneered the discovery of the α-thalassaemia early eluting peak (αEEP) in HPLC for --^{SEA} carrier detection.

Objective: This study aimed to translate the αEEP into clinical practice by confirming its biochemical nature and comparing its diagnostic performance with other existing methods including microscopy (HbHi) and immunochromatographic strip test (ICT) in a multi-centre evaluation.

Methods: A multi-centre diagnostic comparison study was performed across six tertiary referral hospitals in Hong Kong. The αEEP, HbHi and ICT were assessed by blinded observers against genotyping as the gold standard. Diagnostic performance was assessed in clinically relevant subgroups including β-thalassaemia carriers and elevated Hb F levels. The nature of the αEEP was investigated using liquid chromatography-tandem mass spectrometry (LC-MS/MS). Total manpower and reagent costs of each method were compared.

Results: Among 820 patients, the αEEP showed superior diagnostic performance for detecting --^{SEA} mutation (sensitivity 99.6%, specificity 100%) compared with HbHi (sensitivity 95.8%, P=0.006; specificity 97.3%, P<0.001) and ICT (sensitivity 95.8%, P=0.006; specificity 75.4%, P<0.001). The sensitivity of both HbHi and ICT was significantly lower in β-thalassemia carriers than in non-carriers (both tests: 54.6% vs. 97.6%, P<0.001). The specificity of ICT was reduced when Hb F ≥1% compared with <1% (68.6% vs. 78.0%, P=0.02). The αEEP remained robust across all subgroups. LC-MS/MS analysis revealed a strong association between the αEEP and ζ-globin chain (P<0.001). Overall, the αEEP offered cost reductions of 98.6% relative to HbHi and 97.3% relative to ICT.

Conclusion: The αEEP corresponds to embryonic ζ-globin chains and serves as a highly reliable, cost-effective marker for --^{SEA} carrier detection in Hong Kong and potentially other prevalent regions. This finding enables a novel "all-in-one" HPLC screening strategy for --^{SEA}, β-thalassaemia and other haemoglobinopathies.

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Poster Presentation – Abstract VII

Clinical Relevance of Multiparametric Flow Cytometry Measurable Residual Disease Monitoring in Adult Acute Myeloid Leukaemia Patients: A Pilot Evaluation in Hong Kong

Lam Wing Kit[1]; Wong Tsz Fung[1]; Lai Lucy Man Chi[1]; Law Winnie Yim Fong[1]; Tang Mary-Jayne Pui Si[1]; Fan Tze Wing[1]; Sin Yuen Ting[2]; Li Ka Shu[2]; Wong Keith Ka Wai[3]; Ha Chung Yin[2]; Yip Sze Fai[1]

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Abstract

Introduction: Measurable residual disease (MRD) monitoring in acute myeloid leukaemia (AML) is crucial for prognostication and guiding treatment decisions, including allogeneic haematopoietic stem cell transplantation (allo-HSCT). Traditional MRD monitoring in AML relies on molecular methods targeting mutations (e.g. NPM1 mutation) and recurrent gene fusions, but these are applicable in only approximately 35% of patients. A significant unmet clinical need exists for MRD monitoring in the remaining 65% of AML patients lacking suitable molecular targets. Multiparametric flow cytometry MRD (MFC-MRD) monitoring offers a potential solution by providing prognostic information without requiring a specific molecular marker. This pilot study aimed to develop a local MFC-MRD assay using a clinically validated panel in adult AML.

Objectives: This study has the following objectives:
1. To develop a local MFC-MRD assay using a clinically validated panel in adult AML.
2. To compare the applicability of the MFC-MRD assay with the molecular approach in real-world clinical samples.
3. To correlate the MFC-MRD results with patient characteristics and outcome.

Methods: This was a single center, prospective diagnostic test study evaluating MFC-MRD in adult AML. Patients aged 18 years or older receiving intensive chemotherapy during the period 1/6/2023 to 31/3/2025 were enrolled with informed consent. Bone marrow aspirates obtained at diagnosis and during follow-up assessments were analysed by MFC-MRD using the clinically validated ELN panel on a flow cytometer. A leukaemia-associated immunophenotype (LAIP)-based different-from-normal (DfN) approach was employed for data analysis. MFC-MRD results up to the third chemotherapy cycle were analysed, with MFC-MRD positivity defined as ≥0.1% residual disease. Importantly, MFC-MRD results were not disclosed to the clinicians or patients. Patient risk stratification (2022 ELN risk classification), treatment response and clinical progress (censored at 30/9/2025 or date of allogeneic haematopoietic stem cell transplantation [allo-HSCT]) were correlated with MFC-MRD results.

Results: Twenty AML patients (median age, 59 years; male:female ratio, 1:1) were enrolled. 17 (85%) had de novo AML while three (15%) had secondary AML. The median follow-up period was 9.9 months. MFC-MRD was applicable to all 20 patients (100%), compared to only 25% for molecular methods ($p<0.001$). Rates of achieving MFC-MRD negativity by the third treatment cycle (MRD-neg3) by 2022 ELN risk were: favourable-risk 5/5 (100%), intermediate-risk 3/8 (37.5%), and adverse-risk 2/7 (28.6%). Patients who remained MFC-MRD positive after the third cycle had more relapsed/refractory diseases compared to those achieving MRD-neg3 (80% vs 30%). Of the ten MRD-neg3 patients, one (10%) relapsed at 25.1 months after diagnosis, while the remaining patients had no relapse or underwent allo-HSCT with a median follow-up of 9.9 months.

Conclusion: This pilot study successfully developed MFC-MRD monitoring in adult AML using real-world clinical samples. MFC-MRD demonstrated broad applicability, overcoming limitations of molecular monitoring. Overall, MFC-MRD shows promise for refining AML prognostication and treatment.

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Poster Presentation – Abstract VIII

Distinct Clinical Patterns of Oxidative Haemolysis: A Retrospective Study

Lam Wing Kit[1]; Law Winnie Yim Fong[1]; Yip Sze Fai[1]

[1] Department of Clinical Pathology, Tuen Mun Hospital

Abstract

Introduction: Oxidative haemolysis is typically considered to be due to oxidative stress from exposure to xenobiotics and is seen mostly in patients with glucose-6-phosphate dehydrogenase (G6PD) deficiency following exposure to an offending agent. However, the clinical patterns of the oxidative haemolysis were hitherto not systematically studied and the existing literature about cases of oxidative haemolysis are mostly in the form of case reports.

Objectives: This study aimed to investigate the clinical patterns and characteristics of oxidative hemolysis.

Methods: Data were obtained from oxidative haemolysis cases identified from Heinz body test requests from 1st January 2012 to 31st December 2022 in Tuen Mun Hospital, Hong Kong. Oxidative haemolysis was defined as the presence of both morphological evidence of oxidative haemolysis (bite cells and/or blister cells) and increased polychromasia, along with at least one of the following biochemical markers of haemolysis: 1) increased indirect bilirubin level (> 12 μmol/L); 2) increased lactate dehydrogenase level (>220 U/L); or 3) reduced haptoglobin level (<0.30 g/L). The clinical history, drug history, and laboratory results were reviewed.

Results: Among the 27 patients with oxidative haemolysis, three distinctive clinical patterns were identified: 1) zopiclone-induced oxidative haemolysis (Group 1, 11 of 27 cases); 2) other drug-induced oxidative haemolysis (Group 2, 4 of 27 cases); and 3) oxidative haemolysis without potential offending drug identified (Group 3, 12 of 27 cases). Zopiclone-induced oxidative haemolysis showed a female preponderance (Group 1 vs Group 2 vs Group 3: 81.8% vs 50% vs 25%, p=0.017) and higher frequency of depression and/or substance use disorders (Group 1 vs Group 2 vs Group 3: 81.8% vs 0% vs 16.7%, p=0.001). All groups showed a low frequency of G6PD deficiency (Group 1 vs Group 2 vs Group 3: 9.1% vs 25% vs 16.7%, p=0.793). Among the Group 1 patients, four cases (36.3%) presented with acute zopiclone overdose, three of whom had methaemoglobinaemia. The remaining seven patients (63.6%) had delayed or missed diagnoses of zopiclone as the cause of oxidative haemolysis, whom we observed an additional pattern of chronic zopiclone overdose (daily or intermittent consumption of approximately 3–30 times the daily dose of 7.5 mg). Oxidative haemolysis was the dominant feature, and methemoglobinemia and psychomotor symptoms were characteristically absent. Group 3 patients were enriched with cases with critical illnesses and had higher total red cell transfusion requirement (Group 1 vs Group 2 vs Group 3: 1 unit vs 0 unit vs 3 units, p=0.022).

Conclusion: In this study, three distinctive clinical patterns of oxidative haemolysis were identified, with zopiclone being the most frequent cause of drug-induced oxidative haemolysis and is highly associated with history of depression and/or substance use disorders. Oxidative haemolysis without apparent offending agents is more frequently seen in patients with critical illnesses. Investigation of oxidative haemolysis should include detailed clinical assessment with assessment of patient status, organ functions, drug history and toxicology investigations. Zopiclone overdose should be considered a potential cause of oxidative haemolysis.

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Abstract IX

Critical Thrombocytopenia Masked by Leukaemic Cytoplasmic Fragment in Hypercellular Acute Myeloid Leukemia

Lam Wing Kit[1]; Wong Ka Wai Keith[2]

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[2] Department of Medicine and Geriatrics, Tuen Mun Hospital

Abstract

Introduction: Automated completed blood count are widely used and often assumed to be accurate. However, exceptions exist, as demonstrated in our case of acute myeloid leukaemia (AML) with hyperleucocytosis showing inaccurate platelet counts by all automated methods.

Case presentation: A 69-year-old man was admitted for fever, epigastric pain, confusion, multiple bruises and gum swelling with bleeding. Complete blood count showed hyperleucocytosis (white cell count of 152.73 $\times 10^9$/L), hemoglobin of 12.8 g/dL and an impedance platelet count of 53 $\times 10^9$/L. Coagulation profile showed a prolonged PT of 19.8 seconds (reference interval: 10.5-13.0 seconds), a normal APTT, a low fibrinogen level of 0.9 g/L and an elevated D-dimer level of 8571.2 ng/ml. Computed tomography of the brain showed no intracranial hemorrhage.

Peripheral blood smear showed many blasts and promonocytes (83% of leucocytes) with frequent leukaemic cytoplasmic fragments and a paucity of genuine platelets. Repeated testing of the same specimen using optical and fluorescence platelet counting methods showed platelet counts of 46 $\times 10^9$/L and 39 $\times 10^9$/L, respectively.

Despite the apparently reassuring platelet counts, a critical thrombocytopenia was still strongly suspected. The issue was communicated to the clinician in-charge, and immediate platelet transfusion and cryoprecipitate were given. Subsequent flow cytometry platelet counting by flow cytometry confirmed a platelet count of 1.4 $\times 10^9$/L.

The patient received supportive platelet transfusions guided by flow cytometry platelet count monitoring until the hyperleucocytosis and platelet count interference resolved with cytoreductive therapy. He was successfully transitioned from cytoreductive therapy to definitive treatment for AML with azacitidine plus venetoclax, and achieved complete remission without fatal bleeding complications. Unfortunately, the patient had an early relapse and succumbed three months after the initial diagnosis.

Conclusion: Masked critical thrombocytopenia can expose the patient to significant bleeding risk of bleeding contributing to the early mortality in these patients. Timely recognition is crucial to guide transfusions and prevent early fatal complications.

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Abstract X

Acquired Haemophilia A Presenting with Post-Phlebotomy Compartment Syndrome

Lam Wing Kit[1]; Sin Yuen Ting[2]; Wong Ka Wai Keith[2]

[1] Department of Clinical Pathology, Tuen Mun Hospital
[2] Department of Medicine and Geriatrics, Tuen Mun Hospital

Abstract

Introduction: Acquired Haemophilia A (AHA) is a rare bleeding disorder caused by autoantibodies against Factor VIII, most commonly presenting with mucocutaneous bleeding. We present a case with an atypical presentation of compartment syndrome post-phlebotomy.

Case presentation: A 63-year-old man presented to the emergency department with progressive left upper limb swelling and pain four days after phlebotomy at the left cubital fossa for investigation of intermittent bruising over jaw, right wrist and left groin. Physical examination showed tenderness in his left forearm with tense skin, bruises, and multiple blisters over the volar side of left forearm. Left radial pulse is still present with distal sensation intact. Blood tests showed a haemoglobin level of 8.8 g/dL, a normal platelet count and an isolated prolongation of activated partial thromboplastin time (APTT) at 51.5 seconds (reference interval: 23.0-34.7 seconds).

Given the clinical suspicion of compartment syndrome, the patient underwent emergency fasciotomy. Intraoperatively, diffuse bleeding was observed from the subcutaneous tissues, dermis and muscles. Although initial haemostasis was achieved after platelet and plasma transfusions, diffuse wound bleeding recurred on postoperative day 3. The APTT remained prolonged despite repeated plasma transfusions.

This persistent, unexplained bleeding prompted an urgent haematology consultation. Empirical factor eight inhibitor bypassing agent (FEIBA) was initiated for bleeding control while a thorough investigation for an underlying bleeding disorder was undertaken. Factor inhibitor screening showed a time- and temperature-dependent inhibition of the APTT. Factor VIII activity was markedly reduced at 1.3%, and a Bethesda assay confirmed the presence of a factor VIII inhibitor at a level of 14.8 Bethesda units per milliliter, establishing the diagnosis of acquired Haemophilia A. The patient was subsequently started on immunosuppressive therapy, resulting in resolution of the bleeding and normalization of APTT within 10 days.

Conclusion: Diagnosis of AHA can be challenging and often delayed due to a lack of awareness. This case showed an atypical presentation with compartment syndrome after phlebotomy, a seemingly innocuous procedure. The only initial laboratory abnormality was a modestly prolonged APTT, which could easily be overlooked or attributed to other causes. This case highlights the importance of considering AHA in the differential diagnosis of patients with unexplained or excessive bleeding even when the APTT prolongation is only modest. A high index of suspicion is important for prompt diagnosis and appropriate management.

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Poster Presentation – Abstract XI

A Rare Inflammatory Syndrome Managed by a Common Monoclonal Antibody

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Abstract

Background:
TAFRO Syndrome is a rare systemic inflammatory disorder characterized by thrombocytopenia, anasarca, fever, reticulin fibrosis/renal failure and organomegaly, after excluding mimickers including malignancy, infections and autoimmune diseases as required by the Masaki's criteria in 2019. Though considered a subtype of idiopathic multicentric Castleman disease (iMCD) due to resemblance of its lymph node histology to that of Castleman disease, TAFRO Syndrome leads a more rapid and aggressive clinical course than iMCD and can progress to multi-organ failure if not treated promptly. Being a relatively new diagnostic entity, its optimal treatment is yet uncertain, thus challenging.

Objective:
This report shares our experience of successfully managing a 45-year-old woman with TAFRO Syndrome with Rituximab and Cyclosporin A.

Case presentation:
She had history of nasopharyngeal cancer treated by chemo-radiotherapy at 41 years old. She presented with persistent fever and abdominal pain. CT whole body showed generalized oedema and hepatomegaly. Septic workup and autoimmune panel were unremarkable. She developed progressive anaemia and thrombocytopenia. Bone marrow examination demonstrated increased reticulin fibrosis without haemophagocytic activities and was not suggestive of primary myeloproliferative neoplasm. ADAMTS13 activity was not diagnostic of thrombotic thrombocytopenic purpura. Her thrombocytopenia did not respond to intravenous immunoglobulin and steroid. She developed cardiac arrest with asystole for 4 minutes and was successfully resuscitated, subsequently requiring intensive life-supporting care including temporary continuous renal replacement therapy for acute kidney injury.

Considering her thrombocytopenia, anasarca, hepatomegaly, systemic inflammation and the absence of evidence of infections nor malignancy, our team diagnosed her with TAFRO Syndrome. She was given 4 weekly doses of intravenous Rituximab 375mg/m2 and oral Cyclosporin A (gradually escalated from 25mg to 75mg twice daily). Her anasarca and hepatitis gradually resolved. Also, her cell counts and renal function recovered, and she successfully weaned off the ventilator, inotrope and dialysis support. After discharge, PET-CT showed no evidence of occult malignancy. She has been put on maintenance oral Cyclosporin A in our clinic.

Discussion:
Interleukin-6 (IL-6) plays a core role in the pathophysiology of TAFRO Syndrome by stimulating multiple intracellular signaling pathways that transcribe various pro-inflammatory cytokines and vascular endothelial growth factor (VEGF), which promotes endothelial damage and vascular leakage.

There has not been standardized treatment guideline for TAFRO Syndrome. Corticosteroid alone is usually inadequate to completely control the disease. Second-line therapies include anti-IL6 agents (Siltuximab and Tocilizumab), calcineurin inhibitors and Rituximab.

Anti-IL-6 agents are not readily available locally due to their high cost. Also, there were reports of reduced response of Tocilizumab in TAFRO patients with normal serum IL-6 levels. Meanwhile, there have been reports of Rituximab, a far more accessible option, leading to a longer time to next treatment than Tocilizumab and a higher complete response rate than Tocilizumab among TAFRO patients. The efficacy of B-cell depleting Rituximab in such cases is potentially attributable to the underestimated role of plasma cells in the inflammation of TAFRO Syndrome.

Conclusion:
This case motivates evaluation of potential underuse of Rituximab in a more upfront manner for TAFRO patients.

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Poster Presentation – Abstract XII

Arsenic Trioxide Disrupts HSP90α–Client Protein Interactions to Promote Protein Degradation and Suppress Diffuse Large B-Cell Lymphoma

Yue LM; Chau DHW; Tse E; Kwong YL

The University of Hong Kong

Abstract

Introduction: Arsenic trioxide (As_2O_3) is a potent anticancer agent that has been successfully used to treat various malignancies, particularly acute promyelocytic leukaemia. Our previous studies demonstrated that As_2O_3 promotes the degradation of NPM-ALK and cyclin D1 via the ubiquitin–proteasome pathway in anaplastic large cell lymphoma (ALCL) and mantle cell lymphoma (MCL), respectively, leading to growth inhibition and apoptosis in these lymphoma cells. Heat shock protein 90 alpha (HSP90α) is a highly conserved molecular chaperone that plays a central role in the folding, stabilization, and activation of client proteins, including numerous oncoproteins. Notably, both NPM-ALK and cyclin D1 have been identified as HSP90α client proteins and undergo degradation following treatment with HSP90 inhibitors in ALCL and MCL, respectively. The present study aims to elucidate whether HSP90α serves as a molecular target of As_2O_3 in diffuse large B-cell lymphoma (DLBCL) cells in vitro.

Methods: Arsenic-induced cytotoxicity and the expression of HSP90α client proteins (BCL6 and Akt) were assessed in DLBCL cell lines following As_2O_3 treatment. The effect of As_2O3 on BCL6–HSP90α complex formation was examined using co-immunoprecipitation (co-IP) and GST pull-down assays. Rescue experiments were performed by infecting DLBCL cells with lentiviral particles encoding HSP90α cDNA to enforce its overexpression. The impact of HSP90α overexpression on As_2O_3 -induced growth inhibition and apoptosis was further evaluated.

Results: As_2O_3 effectively degraded HSP90α client oncoproteins (BCL6 and Akt) at the post-transcriptional level, induced growth inhibition and apoptosis in DLBCL cells, with effects comparable to or even surpassing those of ganetespib, a Hsp90 inhibitor. Enforced expression of HSP90α preserved a residual level of BCL6 comparable to the basal amount, even after As_2O_3 treatment. This remaining BCL6 was sufficient to protect the cells from As_2O_3 -induced apoptosis, demonstrating that HSP90α is a biological target of As_2O_3. Co-IP and GST pull-down assays further showed that As_2O_3 disrupted the formation of the BCL6–HSP90α complex in DLBCL cells, suggesting a mechanism for targeted client protein degradation.

Conclusions: As_2O_3 inhibits the chaperone function of HSP90α, thereby promoting the degradation of its client oncoproteins in DLBCL. Our findings provide a strong scientific rationale for extending the therapeutic application of As_2O_3, an inexpensive agent with an excellent safety profile, to the treatment of various malignancies, particularly DLBCL.

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Poster Presentation – Abstract XIII

PIN1 Inhibition with Selective Covalent Inhibitors (KPT-6566 and Sulfopin) as a Potential Therapeutic Strategy to Overcome Ibrutinib Resistance in Activated B Cell-like Subtype of Diffuse Large B-cell Lymphoma

Cheng CW; Chau D; Yue LM; Tse E

The University of Hong Kong

Abstract

Objective: Diffuse large B-cell lymphoma (DLBCL) is the most common non-Hodgkin lymphoma, with the activated B-cell-like (ABC) subtype associated with the poorest prognosis due to constitutive NF-κB pathway activation that drives tumor cell survival and proliferation. Although ibrutinib, a Bruton's tyrosine kinase (BTK) inhibitor, blocks NF-κB signaling and offers initial clinical benefit in ABC-DLBCL, responses are often partial and acquired resistance frequently develops. The peptidyl-prolyl isomerase PIN1, overexpressed in various cancers including DLBCL, specifically catalyzes the isomerization of phosphorylated Ser/Thr-Pro motifs in substrates like NF-κB. This enhances NF-κB oncogenic activity by promoting its nuclear accumulation and protein stability, thereby amplifying pro-survival and proliferative signals in cancer cells. We hypothesized that inhibition of PIN1 would suppress NF-κB-mediated survival in ABC-DLBCL cells, providing a therapeutic approach to overcome ibrutinib resistance in this aggressive subtype.

Methods: PIN1 knockdown was achieved in ABC-DLBCL cell lines via lentiviral transduction with shRNA targeting PIN1. Cell proliferation was assessed by MTT assay. Expression of PIN1-interacting oncogenic proteins (including β-catenin, cyclin D1, and NF-κB components) was evaluated by Western blotting, with nuclear NF-κB levels determined in fractionated lysates. The effects of selective covalent PIN1 inhibitors (KPT-6566 and Sulfopin) on cell viability were tested in a dose-dependent manner across multiple ABC-DLBCL lines and compared to ibrutinib and an IRAK1/4 inhibitor treatment.

Results: PIN1 depletion significantly inhibited proliferation in ABC-DLBCL cells and downregulated key oncogenic proteins (β-catenin, cyclin D1, NF-κB), with reduced nuclear NF-κB accumulation. PIN1 knockdown exerted a stronger anti-proliferative effect than ibrutinib, particularly in ibrutinib-resistant ABC-DLBCL lines. Pharmacological inhibition with KPT-6566 and Sulfopin decreased cell viability in a dose-dependent manner across tested ABC-DLBCL lines.

Conclusion: Genetic and pharmacological inhibition of PIN1 suppresses proliferation in ABC-DLBCL cells by attenuating PIN1-mediated NF-κB activation. These findings highlight PIN1 as a promising therapeutic target, especially for overcoming ibrutinib resistance in ABC-DLBCL, and support further development of PIN1 inhibitors for this aggressive subtype.

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Poster Presentation – Abstract XIV

Arsenic Trioxide as a Novel Therapeutic Agent for ALK-Driven Malignancies: Overcoming Resistance in Chimeric ALK Fusion Proteins

CHAU D; YUE LM; PIAO WY; KWONG YL; TSE E

Department of Medicine, The University of Hong Kong

Abstract

Introduction: ALK-driven malignancies present a significant challenge in the field of oncology, primarily due to the complex nature of their biology and the limitations of current treatment options. The *ALK* gene, when mutated or rearranged, leads to the formation of fusion proteins that drive uncontrolled cell proliferation and survival across various malignancies, including Anaplastic Large Cell Lymphoma (ALCL), diffuse large B-cell lymphoma (DLBCL), and Non-Small Cell Lung Cancer (NSCLC). Although tyrosine kinase inhibitors (TKIs) have improved patient outcomes, the emergence of resistance to these therapies poses a substantial hurdle, limiting their long-term efficacy. This resistance underscores the urgent need for alternative therapeutic strategies that can effectively target ALK-driven cancers.

Objective: In this study, we investigate the potential of arsenic trioxide (As_2O_3) as a novel therapeutic agent against the chimeric ALK fusion-driven malignancies.

Methods: We evaluated the effects of As_2O_3 on the aforementioned malignancy models using proliferation assays, apoptosis induction studies, and signalling pathway analyses. The mechanisms of action were further clarified through western blotting and immunoprecipitation.

Results: As_2O_3 exhibited a dose-dependent inhibition of cell proliferation across all chimeric ALK fusion-positive cell lines, with a notable induction of apoptosis. It effectively downregulated NPM-ALK (in ALCL), CLTC-ALK (in DLBCL), and EML4-ALK (in NSCLC) through post-translational modifications that increased their ubiquitination and promoted proteasomal degradation. This downregulation disrupted downstream signalling pathways, leading to a significant reduction in the phosphorylation levels of AKT and STAT3. Additionally, treatment with As_2O_3 significantly inhibited tumour growth in xenograft models, supporting its anti-tumoral properties. Importantly, the effects of As_2O_3 were also observed in Ba/F3 cells expressing TKI-resistant mutants, indicating its potential as a therapeutic option for patients with TKI-resistant forms of the disease.

Conclusion: These results suggest that As_2O_3 is a promising therapeutic candidate for malignancies driven by chimeric ALK fusions, particularly in cases resistant to current ALK TKIs. This highlights its potential to address ALK inhibitor resistance and warrants further clinical investigation.

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Poster Presentation – Abstract XV

A Multicentre Cross-Sectional Study Evaluation of Direct Coombs Test in Characterization of Haemolysis in Paroxysmal Nocturnal Haemoglobinuria Patients Receiving C5 Inhibitors Therapy

LAU Ka Ngai[1]; HWANG Yu Yan[2]; LAU Sze Man June[3]; LAU Wai Pan Chris[3]; MAK Wai Man Vivien[4]; LIN Shek Ying[5]; TAM King Wai Frankie[5]; KHO Bonnie[6]; LEUNG Yuk Yan Rock[7]; WONG Wai Shan[8]; LEUNG Fung Shan Kate[9]; KWONG Hoi Yi Joyce[10]; IP Ka Ling Rosalina[11]; YIP Sze Fai[1]

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Abstract

Introduction and objective:
C5 inhibitors are highly effective treatment for paroxysmal nocturnal haemoglobinuria (PNH) patients to reduce intravascular haemolysis and transfusion requirements, preventing thromboembolism and improving quality of life and survival. In addition to breakthrough haemolysis (BTH), PNH patients on C5 inhibitors experience C3-mediated extravascular haemolysis (EVH) due to uncontrolled C3 bounding on erythrocytes, opsonization and the subsequent removal by the reticuloendothelial system. We conducted a multicentre cross-sectional study to characterize the clinical response of PNH patients receiving C5 inhibitors. The usefulness of the Direct Coombs Test (DCT) in EVH identification was also evaluated.

Method:
The clinical and laboratory findings of patients with confirmed diagnosis of PNH from six hospitals at their latest follow up time point were collected and analyzed from the electronic patient record. Red blood cell transfusions, haemoglobin levels and residual haemolysis (LDH and absolute reticulocyte count) according to the AASWP of the EBMT assessed haematological response. Clinically significant EVH (csEVH) was defined by anaemia (haemoglobin <9.5g/dL) and absolute reticulocyte count >=120 x 10^9/L from the ALPHA study. DCT using monoclonal IgG and C3d were performed on all PNH patients' follow up peripheral blood samples. Intravascular haemolysis was defined by LDH level greater than 1.5 times the upper limit of normal. To evaluate whether DCT could be a potentially useful test to identify csEVH in patients receiving C5 inhibitors, the DCT results (in laboratory score 0-12) were analyzed together and compared with the other clinical and laboratory findings. We stratified the strength of anti-C3d reactivity in DCT into weak positive (<5) and positive (>=5) and compared with their haematological response.

Results:
Twenty-six patients were diagnosed with PNH. Sixteen patients received C5 inhibitor therapy. Two patients (2/16) were excluded from further analysis in view of likely concomitant aplastic anaemia as evident from leukopenia (<3x10^9/L) and marked thrombocytopenia (<20x10^9/L).

Among these 14 patients, the median duration of C5 inhibitors therapy was 50.5 months (range 6-154 months). The median haemoglobin, absolute reticulocyte count and LDH were 10g/dL, 156.8x10^9/L, and 221U/L respectively. Three patients received red cells transfusion within the last six months. Five patients achieved good response, 7 had partial response, and 2 had minor response. One patient had intravascular haemolysis (7.1%) while 3 patients had csEVH (21.4%). Two of the patients with csEVH achieved partial response and one achieved minor response only.

Eleven of the 14 patients (78.6%) had positive DCT and they were all positive for anti-C3d. Four patients had weak anti-C3d positivity (<5) while 7 patients had positive anti-C3d (>=5). The haematological response of the 4 patients with weakly positive anti-C3d (<5) were compared with the 7 patients with positive anti-C3d (>=5) (Table 1). All 3 patients with csEVH had positive DCT and anti-C3d+ with a score >=5. None of the patients with negative DCT (3/14) had csEVH.

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Poster Presentation – Abstract XV

A Multicentre Cross-Sectional Study Evaluation of Direct Coombs Test in Characterization of Haemolysis in Paroxysmal Nocturnal Haemoglobinuria Patients Receiving C5 Inhibitors Therapy

LAU Ka Ngai[1]; HWANG Yu Yan[2]; LAU Sze Man June[3]; LAU Wai Pan Chris[3]; MAK Wai Man Vivien[4]; LIN Shek Ying[5]; TAM King Wai Frankie[5]; KHO Bonnie[6]; LEUNG Yuk Yan Rock[7]; WONG Wai Shan[8]; LEUNG Fung Shan Kate[9]; KWONG Hoi Yi Joyce[10]; IP Ka Ling Rosalina[11]; YIP Sze Fai[1]

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Abstract

Discussion:
Overt intravascular haemolysis was uncommon (7.1%). The incidence of csEVH (21.4%) was similar to the findings observed in clinical trials (20-25%). Anti-C3d+ by DCT was common (78.6%). Although not all patients with anti-C3d score >=5 (7/14) fulfilled the csEVH criteria from the clinical studies, we observed a trend of stronger anti-C3d positivity correlated adversely with the haematological response despite the limited sample size. The strength of anti-C3d positivity from DCT could potentially identify more patients with suboptimal response due to EVH that might benefit from alternative pathway inhibitors.

Table 1: Haematological responses stratified by strength of anti-C3d positivity

	Weakly positive anti-C3d (<5) (N=4)	Positive anti-C3d (>=5) (N=7)
Good response	3	1
Partial response	1	4
Minor response	0	2

References:
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Poster Presentation – Abstract XVI

IGHV Mutation Testing in Chronic Lymphocytic Leukaemia Patients: Experience in Hong Kong East Cluster

Dr MAK Hiu Chun Rory; Mr TSE Hing Fung; Dr KHO Chi Shan Bonnie; Dr IP Ka Ling Rosalina

Haematology Division, Department of Clinical Pathology, Haematology Division, Department of Clinical Pathology

Abstract

Objective
The somatic hypermutation status of the rearranged immunoglobulin heavy variable (IGHV) gene represents one of the most pivotal prognostic and predictive markers in chronic lymphocytic leukaemia (CLL), differentiating between mutated IGHV (M-CLL) and unmutated IGHV (U-CLL) patients. This study evaluates the analytical performance of the LymphoTrack Dx IGHV Leader Somatic Hypermutation Assay for the Illumina MiSeq at Haematology Division, Department of Clinical Pathology, Pamela Youde Nethersole Eastern Hospital to enable IGHV mutation testing to enhance prognostic stratification and to guide treatment implication.

Method
A total of nine CLL patients with known IGHV mutation status having follow-up at the Haematology clinic at Pamela Youde Nethersole Eastern Hospital in 2022 are included in this cohort study. The IGHV mutational status testing is performed on peripheral blood DNA utilizing the LymphoTrack Dx IGHV Leader Somatic Hypermutation Assay for the Illumina MiSeq. Analytical accuracy is evaluated using twenty external quality assurance programme (EQAP) reference samples, while sample agreement is assessed by inter-laboratory cross-validation. Patient characteristics are compared between the U-CLL and M-CLL groups using paired t-tests and Fisher's Exact Test.

Results
All the results obtained by LymphoTrack IGHV assay are concordant with and within the acceptable ranges of the reference answers provided by the EQAP. The accuracy of the test kit is considered acceptable. Also, the positive percentage agreement, negative percentage agreement, and overall percentage agreement are all 100% in the inter-laboratory cross-validation, indicating strong correlation.

Our analysis reveals that U-CLL (44%) is less prevalent than M-CLL (55%), aligning with the findings in Asian populations. A significant gender distribution is identified, with the M-CLL group demonstrating a higher proportion of male patients (p=0.014). The M-CLL group exhibits significantly more patients in Binet stage A (80% versus 0%, p=0.014) and CLL-IPI low-risk category (80% versus 0%, p=0.014), indicating more favourable prognostic profiles. In contrast, our study indicates a trend that U-CLL has association with more adverse prognostic markers. Unfavourable cytogenetic abnormalities, including del11q and del17p, are exclusively identified in U-CLL patients, with 50% harbouring del11q and 25% del17p, while absent in all M-CLL patients. TP53 mutations are similarly restricted to the U-CLL group, identified in 50% of U-CLL patients. No stereotyped subset is assigned to any of the patients in this study. The U-CLL group also demonstrates higher CD38 positivity (75% versus 20%).

Conclusion
Our evaluation study concludes an overall acceptable performance of the LymphoTrack Dx IGHV Leader Somatic Hypermutation Assay for the Illumina MiSeq in CLL, making the assay suitable for clinical application.

Our study also demonstrates that U-CLL is associated with more adverse prognostic features, including unfavourable cytogenetic abnormalities and TP53 mutations, supporting the utility of IGHV mutation status in CLL patient risk stratification and guidance of therapeutic decisions. Future planning will focus on larger-scale studies for comparative analysis to investigate the frequency distribution of the BcR stereotyped subsets in the local populations and evaluation of minimal residual disease monitoring.

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Poster Presentation – Abstract XVII

Molecular and Clinical Prognostic Factors in Systemic Mastocytosis: Evaluation through Prognostic Models Review and Local Case Analysis

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Princess Margaret Hospital

Abstract

Background and Objective
Systemic mastocytosis (SM) is a rare disorder due to clonal mast cell proliferation with a spectrum of clinical presentations. Challenges for accurate prognostic predication emerges due to variety of clinical, laboratory and molecular variables. This retrospective study aims to review multiple prognostic models to identify the key prognostic factors of SM, with a focus on genetic mutations, and to highlight prognostic insights with local cases.

Method
Literature review was conducted on prognostic models including Mutation-Adjusted Risk Score for Advanced Systemic Mastocytosis (MARS), Mayo Alliance Prognostic System (MAPS), International Prognostic Scoring System for Mastocytosis (IPSM), and Global Prognostic Score Model (GPSM). Three cases of SM from Princess Margaret Hospital in 2021-2025 were analysed with myeloid next-generation sequencing (NGS) using a 40-gene targeted panel to assess associated genetic mutations.

Results
All reviewed models identified advanced age, cytopenias, and elevated biochemical markers as adverse prognostic factors. High molecular risk mutations (SRSF2/ASXL1/RUNX1) were shown to predict inferior survival in advanced SM. KIT D816V allele burden was not demonstrated to correlate with mast cell burden and prognosis. Studied case demonstrated adverse mutations (S/A/R) and other myeloid-associated gene mutations (EZH2, IDH2). Overall findings suggest the importance of integrating molecular findings into prognostication in advanced SM.

Conclusion
Prognostic models for SM have incorporated clinical and molecular parameters, with S/A/R mutations becoming one of the most important determinants in advanced disease. Individualized risk assessment is necessary, and ongoing research is needed to improve prognostic tools to guide management, especially in the targeted therapy era.

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Poster Presentation – Abstract XVIII

Co-mutations of CSF3R and SETBP1 in Chronic Neutrophilic Leukaemia and Myelodysplastic / Myeloproliferative Neoplasm with Neutrophilia

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Abstract

Background: Chronic neutrophilic leukaemia (CNL) and myelodysplastic/myeloproliferative neoplasm with neutrophilia (previously named as atypical chronic myeloid leukaemia, aCML) are rare BCR::ABL1 negative myeloid neoplasms with granulocytic proliferation and are considered differential diagnoses of one another. CNL is defined by isolated peripheral blood neutrophilia. Activating mutations in the CSF3R gene are present in most cases and are recognized as one of the essential diagnostic criteria in the WHO fifth edition. aCML is a heterogeneous myeloid neoplasm exhibiting both myelodysplastic and myeloproliferative features. Mutations in the SETBP1 gene have been identified as significant pathogenic events and are included among the desired diagnostic criteria in the WHO fifth edition. Recent studies have reported concurrent mutations of CSF3R and SETBP1 in both CNL and aCML cases, though the frequency varies across different research teams.

Method: We conducted a retrospective review of all patients who underwent bone marrow exam at Prince of Wales Hospital (PWH) from January 2016, to December 2025, identifying two CNL patients and nine aCML patients. All bone marrow specimens were evaluated by two hematopathologists. Sanger sequencing was employed to detect CSF3R and SETBP1 mutations, targeting exon 14 and exon 17 of CSF3R, and hotspot codons 858–871 of SETBP1.

Results: Among the eleven patients, the female-to-male ratio was 4:7, with an average age at diagnosis of 72 years. Six patients had a normal karyotype, while the others exhibited various cytogenetic abnormalities. Survival times ranged from 1 to 23 months after diagnosis, with two patients lost to follow-up. Of the two CNL patients, one presented with co-mutations in CSF3R and SETBP1, while the other was negative for both mutations. Among the aCML patients, mutations in SETBP1 and CSF3R were each detected in three cases, with two of the nine patients exhibiting co-mutations in CSF3R and SETBP1. Notably, one aCML patient had a CSF3R mutation without detectable SETBP1 mutation.

Conclusion: Concurrent mutations of CSF3R and SETBP1 were not uncomment in CNL and aCML patients within the Hong Kong population. Currently, the diagnosis of CNL and aCML relies mainly on morphological features. The presence of SETBP1 and/or CSF3R mutations may support the diagnosis but offers limited value for differential diagnosis.

Limitation: The small number of CNL and aCML patients diagnosed at PWH over the past 10 years may introduce bias in the assessment of concurrent mutations. Additionally, the detection method employed, including the sensitivity of Sanger sequencing and the selected gene regions for analysis, may result in certain mutations being undetectable.

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Poster Presentation – Abstract XIX

A Cross-sectional Study of Cardiac Complications in Transfusion and Non-transfusion Dependent Thalassaemia

Wong SW; Mak WMV; Lee KKH; Kwok CHK; Ho SKS; Ho WBL; Chan KLL; Lau WNG; Chan HY; Wu LY; Ng YNB

Department of Medicine and Geriatrics, Princess Margaret Hospital, Hong Kong

Abstract

Objectives:
Cardiac complications used to be one of the leading causes of mortality in thalassemia patients. This study aims to determine the incidence of cardiac complications in thalassemia patients.

Method:
Total 225 patients (male 44%; female 56%) were identified from the adult thalassemia registry of Kowloon West Cluster, including 59 patients with transfusion dependent thalassaemia (TDT) (requiring regular transfusions before 10 years of age) and 166 with non-transfusion dependent thalassaemia (NTDT). Data collection included latest MRI cardiac T2* with paired serum ferritin, latest echocardiogram (interested parameters included left ventricular ejection fraction (LVEF), right ventricular systolic pressure [RVSP] and features of diastolic dysfunction), serial ECGs and history of cardiac complications. The association between clinical features and disease phenotype were studied with Mann-Whitney U tests for continuous variables, and Fisher's exact tests for categorical variables. Pearson correlations were used to study the relationship between serum ferritin and MRI myocardial T2* values.

Results:
Median age at last follow up was 38 years (range 22-55) for TDT and 56 years (range 20-87) for NTDT ($p<0.001$). All TDT patients, except two with Hb-CS disease and Hb-Bart disease, had β-thalassaemia. For NTDT, 142 patients had HbH disease while 24 patients had β-thalassaemia. Cardiac MRI and echocardiogram were performed in 183 and 110 patients respectively.

Serum ferritin was significantly higher in patients with TDT than NTDT (4586 vs. 1402mmol/L, $P<0.001$). There were 4 patients (7%) with TDT having significant cardiac iron overload but none for NTDT ($P=0.01$). Considering echocardiographic findings, the proportion of patients having hyperdynamic left ventricle (LVEF >70%) or pulmonary hypertension (RVSP >40mmHg) was insignificant between TDT and NTDT patients, but more NTDT patients had diastolic dysfunction (29% vs 0%, $p=0.002$) (Table 1). Of note, one NTDT patient had severe pulmonary hypertension confirmed by right heart catheterization and was recruited to receive regular transfusion. Respectively 5% of TDT and 7% of NTDT patients had atrial fibrillation.

In our exploratory analysis, we showed that serum ferritin had a moderate negative linear correlation with MRI myocardial T2* values (Pearson coefficient -0.42, $p=0.001$) in TDT, but not in NTDT (Pearson coefficient -0.17, $P=0.05$).

Conclusion:
We demonstrated that the prevalence of significant cardiac overload in TDT is low nowadays with modern iron chelators. Significant cardiac deposition is not a concern in NTDT patients. However, we have to regularly screen for pulmonary hypertension and diastolic dysfunction in NTDT patients at risk. ECG screening for atrial fibrillation is also useful in thalassaemia patients. A study with serial measurements of cardiac parameters is currently undergoing, which would be useful to determine the risk factors of cardiac complications in thalassaemia patients.

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Poster Presentation – Abstract XIX

A Cross-sectional Study of Cardiac Complications in Transfusion and Non-transfusion Dependent Thalassaemia

Wong SW;Mak WMV; Lee KKH; Kwok CHK; Ho SKS; Ho WBL; Chan KLL; Lau WNG; Chan HY; Wu LY; Ng YNB

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Abstract

Table 1. Comparison of cardiac parameters and complications between patients with transfusion dependent thalassaemia and non-transfusion dependent thalassaemia (N=225)

	Transfusion Dependent Thalassaemia (TDT) N = 59	Non-transfusion Dependent Thalassaemia (NTDT) N = 166	P
Median Ferritin (mmol/L)	4,586 (728-49,414)	1,402 (42-14,994)	<0.001*
MRI myocardial T2* (ms) – median (range)	36.6 (9.3-50.1)	37.7 (20.4-54.5)	0.21
Cardiac iron overload (T2* <20ms)	4 (7%)	0 (0%)	0.01*
Left ventricular ejection fraction (%) – median (range)	62 (50-75)	64 (44-78)	0.29
Hyperdynamic left ventricle (LVEF >70%)	4 (16%)	13 (16%)	1.00
Echocardiographic features of pulmonary hypertension	1 (4%)	13 (16%)	0.18
Echocardiographic features of diastolic dysfunction	0 (0%)	24 (29%)	0.002*
Atrial fibrillation	3 (5%)	11 (7%)	1.00
Acute coronary syndrome	0 (0%)	3 (2%)	-

Acknowledgement:
The authors would like to thank the late Dr Stephen Choy for his dedication in designing the echocardiography protocol for our thalassaemia patients. We would also like to thank our thalassaemia team of nurses and clerks for data management.

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Poster Presentation – Abstract XX

Acute Promyelocytic Leukaemia Hypogranular Variant - A Case with Diagnostic Challenge

Yuen, Carmen Michelle

Prince of Wales Hospital

Abstract

Acute promyelocytic leukaemia (APL) with PML::RARA fusion is a subtype of acute myeloid leukaemia (AML) characterized by proliferation of abnormal promyelocytes. The genetic hallmark is the fusion of the promyelocytic leukaemia (PML) gene with the retinoic acid receptor alpha (RARA) gene. APL is a highly aggressive type of acute leukaemia that is frequently associated with coagulopathy and fatal bleeding complications. However, if given timely treatment, it is readily curable with excellent prognosis. The initial diagnosis of APL is made based on the typical morphological findings of abnormal promyelocytes or Faggot cells on examination of the peripheral blood film. The definitive diagnosis is confirmed by the detection of PML::RARA fusion by molecular studies. In addition to morphologic evaluation, immunophenotypic study by flow cytometry showing the triad of weak or absent CD34, negative HLA-DR expression, as well as positivity to CD117 supports the diagnosis of APL before molecular results are available.

Here I report a case of APL hypogranular variant which presented as a diagnostic challenge.

A 13 year old boy presented with petechiae on limbs and fever. The peripheral blood counts showed anemia, thrombocytopenia, marked neutropenia with 77% circulating blasts. The blasts were medium in size with high nuclear to cytoplasmic ratio and irregular nuclear contour. Occasional blasts showed fine cytoplasmic granules or are bi-lobed. Rare Auer rods were found but there was no Faggot cells. Bone marrow examination was performed and sent for morphological examination as well as for cytogenetics and molecular studies. The marrow aspirate was predominated by blasts of similar morphology as the peripheral blood. Flow cytometry was performed on the marrow blood and the blasts were located in the typical blast gate, with moderate CD45 expression and low side scatter. They expressed CD34, HLADR, MPO, CD117, CD13, CD33, CD64, CD56, CD2 and CD4. Based on the morphological and immunophenotypic findings, a diagnosis of AML was made. The patient remained asymptomatic and two days later, molecular study showed the detection of PML::RARA fusion. It was confirmed to be the short isoform (bcr3) and FLT3-ITD was also detected. The final diagnosis of APL was made and the patient was started on induction therapy with the triple agents oral tretinoin, arsenic trioxide and ascorbic acid.

APL with bcr3 isoform is frequently associated with hypogranular, immature, blast-like morphology and FLT3-ITD mutation. The immunophenotype is more heterogeneous than classical APL, with frequent co-expression of CD34, CD2 and CD56. In rare cases there may also be expression of HLA-DR. This case underscores the critical need to include APL in the differential diagnosis when encountering immature, blast-like morphology together with the co-expression of CD34, CD2, and CD56. While molecular studies provide the definitive diagnosis of APL, they are more time consuming. A rapid fluorescent in-situ hybridization (FISH) test to demonstrate the presence of PML::RARA fusion can be completed in approximately 5 hours.

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Poster Presentation – Abstract XXI

Novel ETV6-FOXO1 Fusion Transcript in Mixed Phenotype Acute Leukemia (T/Myeloid): Diagnosis and Treatment

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Abstract

Background: Mixed phenotype acute leukemia (MPAL) is a rare and aggressive form of leukemia characterized by the co-expression of markers from multiple hematopoietic lineages. Despite its poor prognosis, treatment guidelines remain unclear due to its rarity and limited studies.

Objective: Here, we report a novel case of MPAL (T/myeloid subtype) in a pediatric patient, featuring a previously undescribed ETV6-FOXO1 fusion transcript.

Case Presentation: A 5-year-old female presented with pancytopenia, severe neutropenia, and blast cells on peripheral smear. Bone marrow analysis revealed hypercellularity with dense blast infiltration and a mixed phenotype profile expressing both cytoplasmic myeloperoxidase (cMPO) and cytoplasmic CD3 (cCD3). Cytogenetic analysis identified a translocation between chromosome 12p12 and 13q14.

Method: Transcriptome analysis by Nova seq and subsequent bioinformatic analysis using STAR fusion and Arriba software was carried out

Results: RNA seq confirmed the presence of a novel ETV6-FOXO1 fusion transcript, involving the PNT domain of ETV6 and the DNA-binding and transactivation domains of FOXO1.

Treatment and Outcome: The patient was treated with an intensive AML-oriented chemotherapy regimen, followed by ALL maintenance therapy. Remarkably, she achieved complete cytogenetic and morphological remission without requiring hematopoietic stem cell transplantation and remains in sustained remission with excellent performance status.

Conclusion: This is the first reported case of an ETV6-FOXO1 fusion in MPAL. The novel fusion highlights a mechanism of transcriptional dysregulation, contributing to leukemogenesis. The favorable outcome in this case demonstrates that intensive chemotherapy protocols can be effective in pediatric MPAL with complex karyotypes. Further studies are warranted to elucidate the functional consequences of this fusion and its potential as a therapeutic target.

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Poster Presentation – Abstract XXII

STAT5B N642H Mutation in Myeloid Neoplasms with Eosinophilia: Clinicopathologic Features in Three Cases from a local cohort

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Abstract

Background
STAT5B N642H activating mutation is recurrently described in T-cell lymphomas/leukemias and less commonly in myeloid neoplasms, where it has been reported to be associated with eosinophilia. However, local data from Asian populations, including Hong Kong, are scarce, and its clinicopathologic features and relationship with eosinophilia in myeloid disorders require further elucidation.

Objectives
To describe the clinical, morphologic, cytogenetic, and molecular characteristics of myeloid neoplasms harbouring STAT5B N642H mutation in patients from the Hong Kong New Territories East and West Clusters, and to evaluate its association with eosinophilia.

Methods
We retrospectively reviewed the results of myeloid panel targeted next-generation sequencing (NGS) performed on bone marrow samples between January and December 2025 (n=129). Cases with pathogenic STAT5B variants were identified, and clinical data, peripheral blood counts, bone marrow morphology, eosinophil/basophil parameters, cytogenetics, co-mutations, and RNA-sequencing for gene fusion detection were analyzed.

Results
Three cases (2.3%) harboured STAT5B N642H mutation: one each diagnosed as chronic myelomonocytic leukemia (CMML), myelodysplastic neoplasm (MDS), and chronic eosinophilic leukemia (CEL) based on WHO classification (5th ed.). Patients were elderly (ages 70-79 years) with all demonstrating persistent eosinophilia (10-32% of leukocytes; absolute counts 1.02-5.82 x109/L) and potential eosinophil-mediated organ involvement (pruritic rash, splenomegaly, recurrent venous thromboembolism); the CEL case also had basophilia (4%). All of them had hypercellular marrows with low or mildly increased blast counts (1-9%), multilineage dysplasia, and mostly unremarkable eosinophil morphology. Reactive marrow mastocytosis was observed in the CMML case.

Conventional cytogenetics were normal or showed trisomy 8. STAT5B N642H variant allele fractions ranged from 0.29 to 0.45, possibly being subclonal in the MDS case whilst representing the dominant or co-dominant clone in the CMML and CEL cases. Co-mutations (1-3 per case) were detected in myeloid disorder-related genes, including SRSF2, TET2, ASXL1, IDH2, and PHF6. No concomitant SF3B1 mutation was found. All cases were negative for BCR::ABL1 and no defining tyrosine kinase gene fusion for myeloid/lymphoid neoplasms with eosinophilia, including FIP1L1::PDGFRA, was detected.

Conclusions
In this cohort, STAT5B N642H mutation was infrequently observed among chronic myeloid neoplasms in elderly patients, which was consistently associated with eosinophilia and possible organ manifestations, supporting a clonal eosinophilic driver role. These findings reinforce the need to incorporate STAT5B in molecular testing for myeloid neoplasms, especially in presence of eosinophilia. Preclinical evidence of JAK inhibitor sensitivity in STAT5B-mutant models suggests potential therapeutic implications, warranting further evaluation in larger cohorts and clinical trials.

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Poster Presentation – Abstract XXIII

Clinical, Immunophenotypic and Molecular Characterization of Six Cases of Adult Mixed Phenotype Acute Leukaemia (MPAL): A Single-Centre Retrospective Review

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Abstract

Background
Mixed phenotype acute leukaemia (MPAL) is rare, comprising ~3% of acute leukaemias, with blasts expressing antigens from two or more lineages. Diagnosis and management are challenging due to evolving classification criteria, molecular heterogeneity, and absence of standardized treatment guidelines. Prognosis is generally poor, with reported 2-year overall survival of 38-80%.

Objectives
To describe the clinical, morphological, immunophenotypic and molecular features of adult MPAL in a single-centre cohort and identify potential characteristics distinguishing an AML-like subset from MPAL.

Methods
All adult MPAL cases diagnosed at Tuen Mun Hospital from 2020 to 2025 were retrospectively reviewed. Diagnosis relied on flow cytometric immunophenotyping of blast population. Clinical, morphological, immunophenotypic and genomic data, including conventional cytogenetics, fluorescence in situ hybridization and targeted next-generation sequencing, were analysed.

Results
Six patients (aged 26-85 years) were identified: B/monocytic (n=1), T/myeloid (n=2), B/myeloid (n=2) and T/megakaryocytic (n=1). Four cases were bilineal and two were biphenotypic. Lymphoid lineage-predominant blasts occurred in three cases (B-lineage n=2; T-lineage n=1); myeloid-predominant in two. Bone marrow blast counts were high (59-98%) in most cases. Three out of six cases had complex karyotypes. Key genomic abnormalities were identified, including BCR::ABL1 fusion in the B/monocytic case, NUP98::NSD1 fusion in one B/myeloid MPAL; while KMT2A amplification and TP53 mutation were both detected in two other cases. No myelodysplasia-related gene mutations were found among the entire cohort.

One T/myeloid MPAL case showed lower marrow blast percentage (20%), myeloid blast predominance, multilineage dysplasia with complex karyotype including -5, del(7q), -13, -17, and TP53 mutation, representing an AML-like subset or possibly reclassifying as AML, myelodysplasia-related per WHO classification (5th ed.).

Induction therapies varied (AML-directed e.g. cytarabine-daunorubicin; ALL-directed e.g. hyper-CVAD) with no clear correlation to the predominant blast lineage. The BCR::ABL1-positive MPAL case achieved complete remission with hyper-CVAD plus imatinib. Four cases were primary refractory or relapsed early; two of which proceeded to allogeneic haematopoietic stem cell transplantation (HSCT) after successful salvage chemotherapy and achieved durable remission with long-term survival (>3 years), whereas the other two cases, which showed complex karyotype with KMT2A amplification and TP53 mutation, remained chemo-refractory and succumbed within 3 months of diagnosis.

Conclusions
This small cohort highlights the diagnostic and therapeutic complexity of adult MPAL. Cases showing myeloid blast predominance, multilineage dysplasia, myelodysplasia-related cytogenetics (e.g. -5/del(5q), -7/del(7q), -17) and/or KMT2A amplification may be considered as AML-like disease and potentially benefit from AML-directed therapies. MPAL cases with lymphoid predominant phenotype and absence of myeloid-related genomic profiles may otherwise be managed in ALL-directed approaches. MPAL with KMT2A amplification and TP53 mutation are associated with treatment resistance and dismal prognosis. Early HSCT consolidation is critical for long-term survival in eligible patients. Integrated immunophenotypic and molecular profiling could possibly guide lineage-matched strategies and hopefully improve patient outcomes. Prospective studies are needed to correlate immunophenotypic lineage findings with molecular signatures and establish biologically informed treatment guidelines.

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Poster Presentation – Abstract XXIV

Seroprevalence of Neutralizing Antibodies Against Adeno-Associated Virus 5 Capsid in Moderate to Severe Hemophilia B Patients

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Abstract

Background: Adeno-associated virus serotype 5 (AAV5) vector-based gene therapy is a promising one-time treatment option for patients with haemophilia. Pre-existing neutralizing antibodies (NAbs) against AAV5 may, however, reduce transduction efficiency. Data on the local seroprevalence of neutralizing anti-AAV5 antibodies among adults with moderate to severe haemophilia B in Hong Kong are limited. This single-centre cross-sectional study therefore aimed to determine the seroprevalence and titre levels of neutralizing anti-AAV5 antibodies in haemophilia B patients.

Method: Between February and July 2025, seven male patients with moderate to severe haemophilia B (baseline factor IX <2%), aged 27–59 years (median 39 years), under the care of Queen Mary Hospital, Hong Kong, were approached for screening. Two patients declined participation; serum samples from five consenting patients were tested in a designated reference laboratory for neutralizing antibody titres against AAV5.

Results: None of the five participants had a factor IX inhibitor. Three patients (60%) had detectable neutralizing antibodies against AAV5, with titres ranging from 228 to 1337.5, while the remaining two patients (40%) had titres below 18.5, below the assay cut-off for positivity.

Conclusion: In this small cohort of adults with moderate to severe haemophilia B, the seroprevalence of neutralizing antibodies against AAV5 was 60%, which appears higher than reported in healthy northern Chinese populations. The small sample size limits generalizability, but the findings suggest that pre-existing AAV5 immunity may substantially affect eligibility for AAV5-based gene therapy locally. Territory-wide screening of the haemophilia population will be important to refine gene therapy planning and to inform choice of vector strategies.

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(Alphabetically ordered)

Gold

abbvie
AMGEN
AstraZeneca 阿斯利康
CSL Behring
Johnson&Johnson
MENARINI
NOVARTIS
PharmaEssentia

Silver

astellas
BeOne
Bristol Myers Squibb
HEALTHCARE CELLTRION
Daiichi-Sankyo
FOSUN KAIROS 复星凯瑞
GSK
IASO BIOTHERAPEUTICS
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HUTCHMED
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