PROGRAMME

8th Greco-Israeli Hematology Meeting

8 - 9 May 2026
Hilton Nicosia

Organizer:

CYPRUS SOCIETY OF HAEMATOLOGY

Under the auspices of:

ISRAEL SOCIETY OF HEMATOLOGY AND TRANSFUSION MEDICINE

HELLENIC SOCIETY OF HAEMATOLOGY

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WELCOME ADDRESS

Dear colleagues,

It is our great pleasure to announce the **8th Greco-Israeli Hematology Meeting**, which will take place in Nicosia, Cyprus, on 8–9 May 2026.

We are delighted to invite you in our meeting with physical presentation and will once again bring together highly esteemed colleagues from Israel, Greece, and Cyprus for another successful and inspiring meeting.

The scientific program will present the latest data across key areas of hematology, alongside the continuation of our highly successful working-group sessions, offering participants valuable opportunities to present their work, exchange ideas, and engage in meaningful scientific discussion.

We warmly invite you to join us in Nicosia to share knowledge, foster collaboration, and advance our field together.

Looking forward to welcoming you to Nicosia this May!

Chairmen of the Organizing Committee

Benjamin Brenner
Professor of Hematology
Rambam Health Care Campus
Haifa

Damianos Sotiropoulos
Director, Dept. of Hematology
G.Papanikolaou Hospital
Thessaloniki

Marios Antoniades
Director, Dept. of Hematology
Nicosia General Hospital
Nicosia

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Benjamin Brenner, Damianos Sotiropoulos, Marios Antoniades

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** Για ασθενείς με μυελοδυσπλαστικά σύνδρομα (MDS) πολύ χαμηλού, χαμηλού ή ενδιάμεσου κινδύνου σύμφωνα με το IPSS-R, χωρίς del(5q), με ή χωρίς άλλες κυτταρογενετικές ανωμαλίες και με ποσοστό δακτυλιοειδών σιδηροβλαστών (RS) <15% (ή RS <5% σε περίπτωση μετάλλαξης SF3B1) και επίπεδα ερυθροποιητίνης ορού (sEPO) ≤500 mU/mL, ή με MDS χωρίς del(5q), με ή χωρίς άλλες κυτταρογενετικές ανωμαλίες, και με RS ≥15% (ή RS ≥5% σε περίπτωση μετάλλαξης SF3B1).

Βιβλιογραφία: 1. REBLOZYL®, Περίληψη Χαρακτηριστικών του Προϊόντος, 02/2025 **2.** PlatzbeckerU, Della Porta MG, Santini V, et al. Efficacy and safety of luspaterceptversus epoetin alfa in erythropoiesis-stimulating agent-naive, transfusion-dependent, lower-risk myelodysplastic syndromes (COMMANDS): interim analysis of a phase 3, open-label, randomisedcontrolled trial. Lancet. 2023;402(10399):373-385. **3.** Κατευθυντήριες Οδηγίες Κλινικής Πρακτικής στην Ογκολογία του NCCN (NCCN Guidelines®) για τα Μυελοδυσπλαστικά Σύνδρομα, Έκδοση 3.2026.. Πρόσβαση στις 23 Μαρτίου 2026. Για την πιο πρόσφατη και πλήρη έκδοση των κατευθυντήριων οδηγιών, επισκεφθείτε την ιστοσελίδα NCCN.org.

Συντομογραφίες: LR-MDS: μυελοδυσπλαστικό σύνδρομο χαμηλότερου κινδύνου (πολύ χαμηλού, χαμηλού ή ενδιάμεσου κινδύνου σύμφωνα με το IPSS-R), ESA: ερυθροποιητίνη, sEPO: ερυθροποιητίνη ορού, RS+: παρουσία σιδηροβλαστών, RS-: απουσία σιδηροβλαστών

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Ενδείξεις:
- Το Reblozyl ενδείκνυται για χρήση σε ενήλικες για τη θεραπεία της εξαρτώμενης από μεταγγίσεις αναιμίας λόγω μυελοδυσπλαστικών συνδρόμων (ΜΔΣ) με πολύ χαμηλό, χαμηλό και ενδιάμεσο κίνδυνο
- Το Reblozyl ενδείκνυται για χρήση σε ενήλικες για τη θεραπεία της αναιμίας που σχετίζεται με την εξαρτώμενη από μεταγγίσεις και τη μη εξαρτώμενη από μεταγγίσεις βήτα-θαλασσαιμία

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Μπορείτε να ανατρέξετε στην Περίληψη Χαρακτηριστικών του Προϊόντος, σκανάροντας το QR code.

Ημερομηνία Αναθεώρησης Κειμένου: 02/2025

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SCIENTIFIC PROGRAM

Friday, May 8th 2026

08:30 - 09:00	Registration
09:00 - 09:30	THROMBOSIS WORKING GROUP
09:30 - 10:00	MDS WORKING GROUP
10:00 - 11:30	ROUND TABLE **LYMPHOMAS** Chairs: **T. Vassilakopoulos – M. Antoniades**
	• The paradox of POD24 in follicular lymphoma, **S. Ringelstein-Harlev** • The new combinations of ABVD in Hodgkin's Lymphoma, **T. Vassilakopoulos** • Accelerated phase CLL/richter syndrome, **T. Tsitskari** • When Lymphomas change identity. From NonHodgkin to Hodgkin, **A. Frangoulides**
	Discussion
11:30 - 12:00	Coffee Break
12:00 - 13:00	ROUND TABLE **ACUTE LEUKEMIAS** Chair: **T. Tsitskari – N. Vyridou**
	• Hot topics in AML, **E. Hatzimichael** • Diagnosis of leukemia from the peripheral blood, **L. Shlush**
13:00 - 13:30	LECTURE Chair: **Maria Michael**
	Innovations in CLL therapy, **T. Tadmor**
13:30 - 14:30	Lunch Break
14:30 - 16:00	ROUND TABLE **MULTIPLE MYELOMA** Chairs: **E. Katodritou – M. Michael**
	Definition and management of High-risk myeloma, **E.Katodritou** Precision Management of the Frail Multiple Myeloma Patient, **I. Avivi** Multiple Myeloma: The road to cure, **M. Michael**

16:00 - 16:30	SATELLITE SYMPOSIUM Chair: **P. Costeas**
	RDW: 40 Years of Presence in Diagnosis, **G.Paterakis**
16:30 - 17:00	Coffee Break
17:00 - 17:45	SATELLITE SYMPOSIUM Chair: **P. Lambropoulou**
	Expanding the Role of Blinatumomab in Acute Lymphoblastic Leukemia: Integration into Frontline Treatment Strategies, **P. Tsirigotis**
17:45 - 18:15	**Opening Ceremony**
	Greetings • **Marios Antoniades**, President of the Cyprus Hematology Society • **George Vasilopoulos**, President of the Hellenic Society of Hematology • **Natanel Horowitz**, President of the Israeli Association for Hematology and Transfusion Medicine • **H.E Oren Anolik**, Ambassador of Israel to Cyprus • **Petros Agathangelou**, President of the Cyprus Medical Association • **Neophytos Charalambides**, Minister of Health, Cyprus
18:15 -18:45	Opening Lecture
	Networks of solidarity: Encounters among Greeks, Jews and Cypriots in the British detention camps in Cyprus, 1946-1949, **E. Matthopoulou**
19:00	Welcome Reception

Saturday, May 9th 2026

08:30 - 09:00	MYELOMA WORKING GROUP
09:00 - 09:30	LYMPHOMA WORKING GROUP
09:30 - 10:30	ROUND TABLE **CELLULAR THERAPIES** Chairs: **I. Sakellari – D. Mallouri**
	• Cord blood transplantation -the role of nicotinamide-modified stem cell therapy, **R. Ram** • Hematology in the era of CAR-T cells, **I. Batsis**
10:30 - 11:00	Coffee Break
11:00 - 12:30	ROUND TABLE **THROMBOSIS AND HEMOSTASIS** Chairs: **B. Brenner – M. Antoniades**
	• Thrombophilia revisited, **E. Papadakis** • AI in Thrombosis Risk Prediction: Are we ready to move beyond traditional scores?, **V. Danilatou** • Challenges in balancing hemostasis and thrombosis in therapy tailoring for hemophilia, **S. Levy Mendelovich**
12:30 - 14:00	ROUND TABLE **MYELOPROLIFERATIVE NEOPLASMS** Chairs: **D. Sotiropoulos – A. Banti**
	• New treatments for Essential Thrombocythemia, **G. Vasilopoulos** • Myelofibrosis, **N. Lavi** • Polycythaemia Rubra Vera, From Phlebotomy to Precision Medicine,, **N. Vyridou**
14:00 - 15:00	Lunch Break

15:00 - 16:00	ROUND TABLE **HEMOGLOBINOPATHIES** Chair: **A. Kourakli – A. Pyrovolaki**
	• Beta-Thalassemia and Sickle Cell Disease: Clinical Experience and Research from the Emek Medical Center, **C. Levin** • Advances in Hemoglobinopathies: Diagnostics, Therapy and Data Resources, **P. Papasavva**
16:00 - 17:00	ROUND TABLE **MYELODYSPLASTIC SYNDROMES** Chairs: **M. Mittelman – A. Symeonidis**
	• MDS 2026: Diagnosis and treatment of lower-risk disease - today and tomorrow, **M. Mittelman** • Clinical trials in HR-MDS: A riddle, wrapped in a mystery, inside an enigma? **I. Kotsianidis**
17:00 - 18:00	PLENARY SESSION Chairs: **B. Brenner – D. Sotiropoulos**
001	OPERATIONALIZING TRUSTWORTHY AI: AN ETHICAL FRAMEWORK FOR CLINICAL PREDICTION MODELS IN THROMBOSIS. **V. Danilatou,** C. Markou, DJ Arachchillage *European University Cyprus*
002	MODIFYING QUALITY AND EQUITY OF CARE: EMERGING ROLES OF GENETIC DISEASE MODIFIERS IN B-HEMOGLOBINOPATHIES Atinuke M. Dosunmu-Ogunbi[1,*], Panayiota L. Papasavva[2,*,†], Oni O. Morohuntodun[1], Petros Kountouris[2], Julie Makani[3], Bin Alwi Zilfalil[4], Baba P.D. Inusa[5], Natasha M. Archer[1], and **Carsten W. Lederer[2,†]**, on behalf of the International Hemoglobinopathy Research Network (INHERENT)[‡] *The Cyprus Institute of Neurology and Genetics. Department of Blood Disorder Genetics and Thalassaemia*

003 GENERATION OF α^0-THALASSEMIA HUDEP CELL LINE MODELS FOR GENE THERAPY DEVELOPMENT USING CRISPR/CAS9

C. Constantinou, M. Phylactides, P. Patsali, P. L. Papasavva, C. W. Lederer

The Cyprus Institute of Neurology and Genetics. Department of Blood Disorder Genetics and Thalassaemia

004 TOWARDS SAFER IRON-RESTRICTIVE THERAPIES: VALIDATING CYTOSINE BASE EDITING OF TMPRSS6 AS A DSB-FREE ALTERNATIVE TO CAS9

M. Azzam[*], N. Y. Papaioannou, A. Hendel, C. W. Lederer[†‡], P. Patsali[†‡], **P. L. Papasavva**[†‡]

The Cyprus Institute of Neurology & Genetics

005 IMPACT OF CD34 + MICROPARTICLES ON THE LPS INDUCED EXPRESSION PROFILE OF IMMUNOMODULATORY MEDIATORS

A. Alexandridou[1,2], V. Stavrianidou[2], V. Hatsiou [3], D. Papaioannou[1,2], I. Sakellari[1], A. Xagorari1, **D. Sotiropoulos**[1]

[1] *Public Cord Blood Bank of Thessaloniki, Dept. of Hematology-BMT Unit, "Papanicolaou" Hospital, Thessaloniki, Greece*

[2] *Department of Genetics, Development and Molecular Biology, School of Biology, Aristotle University of Thessaloniki, Thessaloniki, Greece*

[3] *Biochemistry Laboratory, "Papanicolaou" Hospital, Thessaloniki, Greece*

18:00 - 18:30 Awards and Closing Remarks

E - POSTERS

P01 INTERSTITIAL LUNG DISEASE PRESENTING AS ORGANIZING PNEUMONIA INDUCED BY NILOTINIB A RARE BUT SEVERE ADVERSE EVENT

Kanava V, Makri E, Lalayanni C, Karypidis S, Papathanasiou M, Touloumenidou T, Paphianou E, Kyriakou I, Papalexandri A, Sakellari I

G Papanikolaou Hospital, Thessaloniki

P02 PRECLINICAL VALIDATION OF CRISPR/CAS GENOME EDITING APPROACHES AS ADVANCED THERAPY FOR HBBIVSI-110(G>A) THALASSEMIA

P. Patsali, C.G. Constantinou, K. Paschoudi, N. Papaioannou, B. Naiisseh, P.L. Papasavva, M. Tomazou, P. Christofi, S. Christou, M. Sitarou, A. Pirovolaki, M. Hadjigabriel, T. Athanasopoulos, C. Mussolino, T. Cathomen, M. Kleanthous, N. Psatha, E. Yannaki, C.W. Lederer

The Cyprus Instittute of Neurology and Genetics. Department of Blood Disorder Genetics and Thalassaemia

P03 PROGNOSTIC SIGNIFICANCE OF NEUTROPHIL-TO-LYMPHOCYTE RATIO AT DIAGNOSIS IN ACUTE MYELOID LEUKEMIA

Bountoura S, **Lalayanni C**, Tzanninis R, Kyriakou I, Salvaras G, Papathanasiou M, Iskas M, Marvaki N, Varelas C, Athanasiadou A, Vadikoliou C, Kosmidou A, Papadopoulou E, Papadopoulos I, Syrigou A, Panteliadou K, Papalexandri A, Sakellari I

G Papanikolaou Hospital, Thessaloniki

P04 SUCCESSFUL PREGNANCY OUTCOME IN A PATIENT WITH PAROXYSMAL NOCTURNAL HEMOGLOBINURIA TREATED WITH RAVULIZUMAB

Dadaki E, **Lalayanni C**, Gavrilaki E, Marvaki A, Papadopoulou- Skordou E, Kaliou M, Koravou E, Marvaki A, Papaioannou N, Sourri S, Papalexandri A, Sakellari I

G Papanikolaou Hospital, Thessaloniki

P05 LINKING MICROPARTICLES ISOLATION TECHNIQUES TO IN VITRO MODELS OF INFLAMMATION

A. Alexandridou [1,2], V. Stavrianidou[2], V. Hatsiou [3], D. Papaioannou[1,2], I. Sakellari[1], **A. Xagorari**[1], D. Sotiropoulos[1]

G Papanikolaou Hospital, Thessaloniki

CLL ΧΡΟΝΙΑ ΛΕΜΦΟΚΥΤΤΑΡΙΚΗ ΛΕΥΧΑΙΜΙΑ

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TN CLL

Μελέτη SEQUOIA[2]

PFS 6 ετών: 74%[4]

Ποσοστό των ασθενών που επιβίωσαν χωρίς εξέλιξη της νόσου έναντι BR: 32% (INV-assessed, χωρίς del(17p))

R/R CLL

Μελέτη ALPINE[3]

PFS 5 ετών: 47%[5]

Ποσοστό των ασθενών που επιβίωσαν χωρίς εξέλιξη της νόσου

Στην τελική ανάλυση της μελέτης στα 3 έτη για την PFS, BRUKINSA 65,4% έναντι ibrutinib 54,4%
- η παρακολούθηση 5 ετών αφορά μόνο το σκέλος του BRUKINSA

ΣΥΝΤΟΜΟΓΡΑΦΙΕΣ: BR: bendamustine+rituximab, CLL: Chronic Lymphocytic Leukemia-Χρόνια Λεμφοκυτταρική Λευχαιμία, INV-assessed: αξιολογημένη από τον ερευνητή, PFS: επιβίωση χωρίς εξέλιξη της νόσου.

▼ Το φάρμακο αυτό τελεί υπό συμπληρωματική παρακολούθηση. Αυτό θα επιτρέψει τον γρήγορο προσδιορισμό νέων πληροφοριών ασφαλείας. Ζητείται από τους επαγγελματίες υγείας να αναφέρουν οποιεσδήποτε πιθανολογούμενες ανεπιθύμητες ενέργειες Βλ. παράγραφο 4.8 για τον τρόπο αναφοράς ανεπιθύμητων ενεργειών.

Για περισσότερες πληροφορίες μπορείτε να ανατρέξετε στην Περίληψη Χαρακτηριστικών του Προϊόντος, σκανάροντας το QR code.
Ημερομηνία Αναθεώρησης Κειμένου: Οκτώβριος 2025

ΣΧΕΔΙΑΣΜΟΣ ΜΕΛΕΤΗΣ SEQUOIA: Η SEQUOIA ήταν μια τυχαιοποιημένη, ανοιχτού σχεδιασμού, φάσης 3 μελέτη, σε πρωτοθεραπευμένους ασθενείς με ΧΛΛ που σύγκρινε το BRUKINSA με τον συνδυασμό Bendamustine+Rituximab. Επειδή οι ασθενείς με ΧΛΛ/ΜΛΛ των οποίων οι όγκοι παρουσιάζουν del(17p) έχουν δυσμενή πρόγνωση και μικρή ανταπόκριση στην καθιερωμένη ανοσοχημειοθεραπεία, αυτοί έλαβαν BRUKINSA σε ξεχωριστή διερευνητική ανάλυση μονού σκέλους. Στην κοορτή 1 συμμετείχαν ασθενείς χωρίς del(17p): BRUKINSA έναντι BR (n=479). Στην κοόρτη 2 συμμετείχαν ασθενείς με del(17p), BRUKINSA μονού σκέλους (n=111).[2]

ΣΧΕΔΙΑΣΜΟΣ ΜΕΛΕΤΗΣ ALPINE: Η ALPINE ήταν μια τυχαιοποιημένη, ανοιχτού σχεδιασμού, πολυκεντρική, φάσης 3 μελέτη σε ασθενείς με υποτροπιάζουσα/ανθεκτική ΧΛΛ που είχαν λάβει ≥1 προηγούμενη συστηματική θεραπεία που σύγκρινε το BRUKINSA με το ibrutinib (N=652).[3]

Ημερομηνία Σύνταξης: 03.2026

ΒΙΒΛΙΟΓΡΑΦΙΑ: 1. BRUKINSA® Περίληψη των Χαρακτηριστικών του Προϊόντος, Οκτώβριος 2025. **2.** Tam CS, et al. Lancet Oncol. 2022;23(8):1031-1043. **3.** Brown JR, et al. N Eng J Med. 2023;388(4):319-332. **4.** Tam CS, et al. BGB-3111-304, ASH Annual Meeting, 6-9 December 2025, Abstract 2029. **5.** Tam CS, et al. «Long-term extension from the Phase 3 ALPINE study: Sustained Efficacy of Zanubrutinib vs Bendamustine + Rituximab.» ASH Annual Meeting, 6-9 December 2025; Abstract 2123.

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GENERAL INFORMATION

Meeting dates and venue

May 8th - 9th, 2026
Hilton, Nicosia, Cyprus
(1 Achaion Street, Egkomi, Nicosia, 2413, tel.22 695111)

Official language

English will be the official language of the Meeting.

No simultaneous translation will be provided.

Registration Desk

A Registration Desk will be operating throughout the duration of the Meeting.

The Registration Desk will also be operating as an Information Desk for any information or assistance participants may require during the Meeting.

Registration Desk Opening Hours:

Friday, 8th of May: 08:30 – 18:30
Saturday, 9th of May: 08:00 – 18:30

Meeting Room

Main Meeting Room: LEDRA B

Name Badges

The badge of each participant gives access to all sessions, exhibition, coffee breaks, lunch breaks and the Welcome Cocktail Reception. It is mandatory for the delegates to show their Meeting badge at the entrance of the meeting room in order to calculate their attendance time in all sessions.

Poster viewing

All posters will be projected digitally on a monitor screen, at the coffee break area, throughout the duration of the Meeting.

Accreditation Points CME

The Meeting has been accredited by the Cyprus Medical Association for 11 CME credit hours. Each medical specialist should claim only those credit hours corresponding to their actual participation in the educational activity.

Certificates of Attendance

Certificates of attendance will be sent electronically after the Meeting is completed.
CME points will be also mentioned on the certificate.

Welcome Cocktail Reception

Welcome Cocktail Reception will take place on Friday, May 08th, between 19:00 - 21:00 at the Hotel.

Exhibition

There will be an exhibition of medical equipment and pharmaceutical products at the Meeting venue.

Presentations - Technical support

Available visual equipment for all presentations will be through power point presentation.

Presenters are kindly requested to submit their presentations in advance to the Meeting Secretariat. Alternatively, all presentations must be uploaded to the technical support desk at least one hour prior to the scheduled presentation. The use of personal computers during sessions will not be permitted.

Registration fees

REGISTRATION TYPE	COST
Specialist Hematologists	€100
Other Specialists	€60
Other Health professionals	€60
Residents	€50
Nurses	€20
Undergraduate students*	€10

* Kindly please note that there is no possibility of covering by the pharmaceutical companies.
Students should cover the registration fees on their own.

Registration fees include

- Admission to the scientific sessions
- Meeting material
- Certificate of attendance
- Admission to the Exhibition Area, Coffee Breaks, Light Lunches and Cocktail Reception according to the programme

Registration can be completed online using the link bellow
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Registration

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Βιβλιογραφία:
1. Sharman JP et al. Blood. 2025 Sep 11;146(11):1276-1285 **2.** Brown JR, et al. N Engl J Med. 2025. **3.** Wang M et al, J Clin Oncol. 2025 Jul 10;43(20): 2276-2284 **4.** CALQUENCE® Summary of Product Characteristics. Jul 2025

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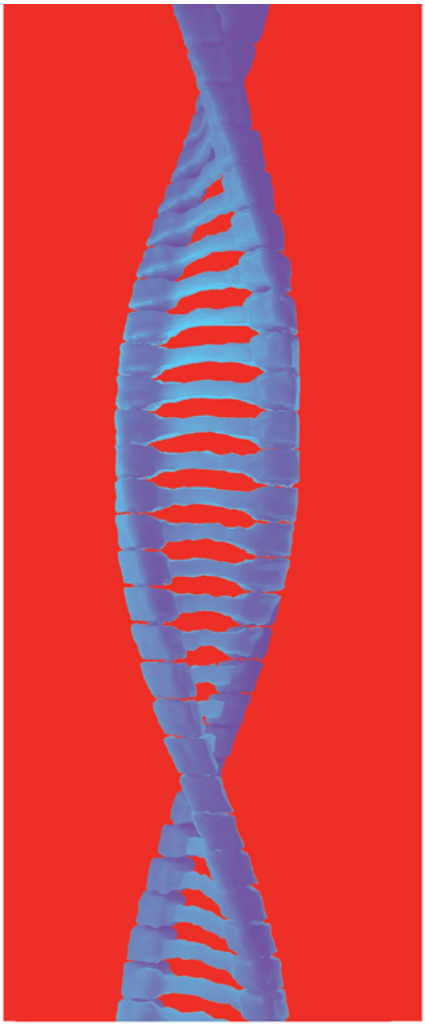

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VHP HADJIPANAYIS

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ORAL PRESENTATIONS

001

OPERATIONALIZING TRUSTWORTHY AI: AN ETHICAL FRAMEWORK FOR CLINICAL PREDICTION MODELS IN THROMBOSIS.

V. Danilatou [1,2] C. Markou [2,3] DJ Arachchillage [4]

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[4] *Centre for Haematology, Department of Immunology and Inflammation, Imperial College London*

Purpose of the study
Traditional predictive models based on statistical methods require internal and external validation before clinical implementation. The increasing use of artificial intelligence (AI) in clinical prediction introduces additional challenges, including bias, lack of transparency, and post-deployment monitoring. In thrombosis care, inaccurate risk prediction may lead to inappropriate management: overestimation can result in unnecessary anticoagulation and bleeding risk, while underestimation may expose patients to preventable thrombotic events. Accordingly, clinicians and healthcare institutions require structured governance mechanisms to ensure safe and accountable use.

With the entry into force of the EU AI Act, the deployment of high-risk AI systems in healthcare is no longer solely an ethical issue but also a matter of legal compliance. The Assessment List for Trustworthy Artificial Intelligence (ALTAI), developed by the European Commission, provides a structured framework aligned with these principles. However, its high-level nature limits direct clinical applicability. This creates a gap between regulatory expectations and real-world implementation. The aim of this study is to translate ALTAI principles into a practical, measurable framework supporting safe and accountable use of AI prediction models in thrombosis care.

Material and Methods
We applied the framework to a published ML model for thrombosis outcome prediction using electronic health record data (doi: 10.3390/ijms23137132). Using a structured operationalisation approach informed by the literature, we mapped ALTAI's seven principles into clinically relevant governance components. For each principle, we defined: (a) potential patient safety or clinical risks; (b) mitigation strategies and clinical actions; and (c) candidate indicators for monitoring safety and performance. The framework was iteratively refined through multidisciplinary expert review and applied to the ML model to ensure practical relevance.

Results
The framework identified 15 clinically relevant risk scenarios across all ALTAI principles, each linked to specific clinical actions and measurable indicators (Table 1). Key findings included the importance of enabling clinicians to interpret (Explainability) and override AI outputs (Human Oversight), performing regular local performance audits and standardised reporting (Technical Robustness), ensuring adequate data protection measures (Privacy and Data Governance), monitoring performance across patient subgroups such as age, sex, and ethnicity (Fairness), and establishing clear accountability structures for AI-assisted decisions (Accountability).

Conclusions

This framework provides a practical roadmap for clinicians and institutions deploying AI in thrombosis care by translating ethical principles into actionable steps and measurable safety checks. Several methodological gaps remain. While performance metrics such as discrimination and calibration are well established, many indicators for ethical AI governance, particularly those related to clinician-AI interaction, fairness assessment, and transparency, lack standardised definitions and validated benchmarks. In addition, there is no consensus on monitoring intervals for AI performance deterioriation in routine practice or on how AI recommendations should be weighted relative to clinical judgement. The proposed approach is adaptable to other areas of haematology and clinical medicine where AI-driven prediction models are increasingly used.

Table 1. Operational framework for trustworthy AI in thrombosis care: mapping ALTAI ethical pillars to clinical risks, recommended governance actions, and measurable safety indicators.

ALTAI Pillar risk	Clinical Risk	Recommended actions and mitigation	Suggested metrics and thresholds
1. Human Agency & Oversight			
Over-reliance on AI (Automation bias)	Clinician accepts AI prediction without critical review, leading to inappropriate treatment	Explainable AI; Display AI explanations showing top contributing factors; Clear display of uncertainty; Require active clinician confirmation before high-risk decisions; mandatory AI literacy training; All oversight mechanisms (human-in-the-loop, human-on-the-loop, human-in-command) should be active	Rate of clinician override; Blind acceptance rate (target <5%); Regular concordance audits
Misinterpretation of AI output	Clinician misunderstands uncertainty levels or prediction meaning, leading to inappropriate treatment	Plain-language output summaries; Clear display of uncertainty; Regular comprehension checks for clinical users	Clinician comprehension score (target >80%);
2. Technical Robustness & Safety			
Poor data quality	Model trained or run on incomplete or unrepresentative records, producing unreliable predictions	Standardised data preprocessing; Document missing data per variable; Pre-specified imputation strategy	Data completeness score; Missing data rate per variable; Outlier detection rate
Unsafe model performance	Model underperforms in clinical use, e.g., misses high-risk patients or over-predicts events	Regular performance audits (sensitivity, specificity, calibration); Define performance threshold below which model is suspended pending review	AUC, sensitivity, specificity, F1 score for imbalanced datasets; Calibration slope AUC change from baseline (review threshold: >0.05 change)

Model drift	Performance deteriorates over time as patient population or clinical practice changes	Monitor model predictions vs. actual outcomes at regular intervals; Schedule periodic retraining on updated data	Calibration slope (target 0.85–1.15); AUC trend monitoring; Population stability index
Poor generalisability	Model validated in one clinical centre or dataset performs poorly in a different hospital or patient population	External validation in independent cohorts; Document training conditions; Document model versioning	AUC difference vs external cohort (target <0.05)
3. Privacy & Data Governance			
Data privacy breach	Patient data used for AI training or inference is exposed or misused	Privacy-by-design architecture; Encryption; Pseudonymisation of training data; Anonymization of shared data in open access repositories; Controlled access repository; GDPR compliance	Data breach incidence; Time to detection and containment; Time to regulatory notification (target <72 hrs per GDPR Art.33)
Inadequate consent & anonymisation	Patients unaware their data informs AI; Re-identification risk from supposedly anonymous datasets;	GDPR compliance; Right to withdraw and right to erasure defined; Designated Data Protection Officer (DPO); Documented consent procedures; Re-identification risk assessment before any data release	Consent completion rate; Comprehension score post-consent; Re-identification risk score (k-anonymity)
4. Transparency & Explainability			
Black-box predictions	Clinician cannot understand why the AI made a specific recommendation, undermining trust and accountability	Explainable AI (XAI); Provide explanation of top contributing clinical features for each prediction (e.g., using SHAP); Partial dependence plots	Clinician comprehension score (target >80%); Feature importance stability across patient subgroups
5. Fairness & Non-Discrimination			
Algorithmic bias	Model performs differently across different patient subgroups (e.g., sex, age, ethnicity, socioeconomic status), leading to unequal care	Identify possible subgroups that may be subject to unfair treatment or bias; Evaluate performance separately by key subgroups; Use bias-aware algorithms; Recalibrate if performance gaps detected	AUC difference across subgroups (target <0.05); Brier score per subgroup; Bias detection rate; PROBAST-AI tool

6. Societal & Environmental Well-being			
Impact on workforce	Clinicians' over-reliance on AI leads to deskilling; Possible additional workload burden on already stretched clinical teams	AI literacy training; Model integration into electronic health record reduces manual entries; Workflow redesign or co-designed with clinical staff; Monitor workload impact post-deployment;	User trust score; Blind acceptance rate (target <5%); Clinicians' workload survey; Time required to review AI output
Environmental footprint	Computationally intensive AI models carry a significant carbon cost	Model optimisation and dimensionality reduction; Preference for energy-efficient architectures; Align with Green AI principles	GPU hours per training run; Energy consumption per model execution (kWh)
7. Accountability & Governance			
Lack of auditability	No clear record of which AI version made which recommendation, making adverse event review impossible	Full audit trail of model versions and predictions; Daily log backups; All model updates documented	Audit log completeness; Version traceability score
Unclear accountability	When AI-assisted decisions cause harm, responsibility is unclear — between clinician, institution, and developer	AI registry with named clinical lead; Define clear responsibility roles; Ethics Review Board and AI Governance Committee oversight; Human-in-the-loop mandate for high-risk decisions; Red Teaming programme	All safety metrics above; Adverse event rate attributable to AI; Governance review frequency

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002

MODIFYING QUALITY AND EQUITY OF CARE: EMERGING ROLES OF GENETIC DISEASE MODIFIERS IN B-HEMOGLOBINOPATHIES

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Abstract

Genetic disease modifiers (GDMs) strongly affect disease and therapy outcomes, rendering their characterization essential for prognosis, treatment decisions, and therapy development. A paradigm for the importance of GDMs are β-hemoglobinopathies, for which a growing pipeline of emerging curative and disease-modifying therapies are based on the manipulation of GDMs. However, beyond their role as therapeutic targets, GDMs are also gaining clinical importance in the choice and planning of disease-modifying therapies, the selection and customization of curative therapies, and the equitable provision and global reach of therapies across ethnic groups and geo-economic divides. Here we define roles of GDMs in all four of these fields, highlight their potential to promote choice, effectiveness, affordability, and equitable provision of treatment, and emphasize the importance of capacity building through multiethnic GDM-related research. We conclude that consideration of GDMs at all stages of development and care will be critical to addressing the needs of affected communities.

003

GENERATION OF α^0-THALASSEMIA HUDEP CELL LINE MODELS FOR GENE THERAPY DEVELOPMENT USING CRISPR/CAS9

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Purpose of the Study:
Alpha thalassemia is a common inherited hemoglobin disorder caused by deletions or mutations in the HBA1 and HBA2 genes, which encode the α-globin chains essential for embryonic, fetal and adult hemoglobins. Mutations in these genes result in reduced or absent α-globin production and consequently, imbalance between the α-globin and β-like globin chains of hemoglobin, leading to ineffective erythropoiesis of varying severity, depending on the number of affected α-globin genes. The most severe form, α^0-thalassemia (hydrops fetalis), results from mutations in all four α-globin genes and leads to early postnatal or intrauterine fetal death. Accordingly, primary cells from affected patients are not readily available for research, making it essential to develop alternative model systems. To address this, we generated CRISPR/Cas9-edited in vitro α^0-thalassemia model cell lines using HUDEP-2 erythroid progenitor cells, which predominantly express adult hemoglobin (HbA).

Methodology:
CRISPR/Cas9 gene editing was employed to delete both the HBA1 and HBA2 genes in HUDEP-2 cells. Two gRNAs flanking the deletion region and the Cas9 protein were introduced via electroporation, and successful deletion was confirmed in the cell population using gap-PCR and Sanger sequencing for precise breakpoint validation. Single cells were then isolated using fluorescence-activated cell sorting (FACS) and the single-cell derived clones were screened using a duplex PCR approach to distinguish between biallelic, heterozygous, and non-deleted clones. Functional characterization of selected heterozygous and biallelic lines was performed, including erythroid differentiation assays and globin expression profiling by reverse-phase high-performance liquid chromatography (RP-HPLC).

Results:
Six biallelic HUDEP-2 α^0-thalassemia clones were identified by duplex PCR. Flow cytometry analysis using CD36-APC and CD235a-PE markers revealed delayed erythroid maturation in biallelic clones compared to wild-type, heterozygous, and inverted clones (i.e. clones where the editing event led to inversion of the gRNA-flanked region). Additionally, globin analysis by RP-HPLC confirmed the absence of α-globin expression in one biallelic clone that was analysed (α^0-thalassemia), highlighting its applicability for investigating α-thalassemia and potential therapies.

Conclusions:
We successfully generated HUDEP-2 α-thalassemia model cell lines, providing a potential platform for studying disease mechanisms and testing gene therapy strategies. Further characterization is necessary to validate these models through additional functional assays. These models could offer a valuable tool for the development of novel therapeutic interventions for α-thalassemia.

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004

TOWARDS SAFER IRON-RESTRICTIVE THERAPIES: VALIDATING CYTOSINE BASE EDITING OF *TMPRSS6* AS A DSB-FREE ALTERNATIVE TO CAS9

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Background:
Dysregulated iron homeostasis underlies multiple hereditary and acquired hematological disorders. TMPRSS6 (matriptase-2), a key suppressor of hepcidin, represents an attractive therapeutic target for durable iron-restrictive strategies. While CRISPR/Cas9 enables highly efficient gene disruption, its reliance on double-strand breaks (DSBs) motivates the development of DSB-free precision editing approaches. Here, we aimed to develop a non-viral hepatic editing platform to directly compare Cas9-mediated disruption and DSB-free cytosine base editing for therapeutic TMPRSS6 inactivation.

Material and Methods:
Guide RNAs were designed to introduce a premature stop codon in TMPRSS6. Cas9 ribonucleoproteins (RNPs) and CBEs (mRNA and RNP formats) were delivered to human Hep3B and murine AML12 hepatocytes using optimized nucleofection. Delivery efficiency (GFP-positive cells) and viability (SYTOX Red staining) were assessed by flow cytometry, and editing outcomes were quantified using Sanger sequencing (ICE and EditR analysis).

Results:
Optimized nucleofection achieved up to 70% transfection efficiency while maintaining 70-85% post-electroporation viability. While Cas9 RNPs yielded robust on-target editing (mean 84% in AML12; 88% in Hep3B), DSB-free CBE editing achieved clinically relevant efficiencies of up to 47% (RNPs) and 31% (mRNA) in Hep3B cells. These data demonstrate that therapeutically relevant levels of TMPRSS6 inactivation can be achieved without relying on DSB induction, using a DSB-free editing strategy.

Conclusions:
This establishes a comparative framework for TMPRSS6 inactivation in hepatic models, enabling direct evaluation of DSB-dependent and DSB-free editing strategies. The platform is now being advanced to functional interrogation of the hepcidin/BMP-SMAD axis and evaluation in disease-relevant animal models toward therapeutic modulation of iron homeostasis.

005

IMPACT OF CD34+ MICROPARTICLES ON THE LPS-INDUCED EXPRESSION PROFILE OF IMMUNOMODULATORY MEDIATORS

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Purpose of the study:
The umbilical cord blood (UCB) constitutes an alternative source for transplantation. Hematopoietic cells under stimulating conditions such as proliferation or apoptosis, release extracellular vesicles of 0.1–1 μm in size called microparticles (MPs). Previous studies have showed that MPs derived from stem cells that express the CD34+surface antigen (CD34+MPs), contribute to basic functions of hematopoietic cells including apoptosis and expression of antioxidant genes. Lipopolysaccharide (LPS) stimulates cells to induce proinflammatory cytokines and chemokines via the TLR4 receptor. The aim of the present study was to determine the effect of umbilical cord blood (UCB) derived CD34+MPs in hematopoietic cells in the presence of LPS, as well as to investigate the effect of LPS on IL-6 protein's expression in mononuclear cells (MNCs).

Material and Methods:
UCBs (n=12) inappropriate for transplantation due to low volume were collected after informed consent. UCB-derived MNCs were isolated with Ficoll-Paque density gradient centrifugation. CD34+MPs were isolated from UCB's plasma and their number was determined by flow cytometry and immunomagnetic separation using MACS technology (Miltenyi Biotec). MNCs (0.9 x 106) were treated with E. coli (O128:B12) LPS at concentration of 100ng/mL along with 800 CD34+MPs and incubated for 24 hours. The effect of CD34+MPs was studied on the mRNA expression of: IL-1β and IL-8 via quantitative-PCR (q-PCR). Proteome Profiler Human Cytokine Array (R&D Systems) was conducted for the detection of cytokines and chemokines related to inflammation. MNCs were also treated only with LPS for different time courses of incubation: 3 and 6 hours respectively. The protein levels of interleukin 6 (IL-6) were measured in culture supernatants using the Elecsys immunoassay COBAS analyzer (Roche).

Results:
Proteomic analysis revealed marked differences in protein expression. LPS stimulation in MNCs resulted in increased levels of pro-inflammatory cytokines: IL-1α, IL-1β, regulatory cytokine/inflammatory protein: MIF, chemokines: IL-8, CCL5 and adhesion/co-stimulatory molecules: ICAM1 related to control. The presence of CD34+MPs within LPS-treated MNCs resulted in a marked induction of IL-1α, IL-1β, IL 8, CCL5, ICAM-1 with a mild reduction of MIF. CD34+MPs reduced significantly the gene expression of IL-1β in LPS-treated MNCs in contrast to the proteomic analysis. Furthermore, LPS stimulation on MNCs significantly induced the expression of IL-6 with levels often detectable within 3 hours and showing a further increase within 6 hours, marking a rapid and robust inflammatory response.

Conclusions:
UCB derived-CD34+microparticles attenuate the expression of LPS-driven inflammation markers through complex regulatory dynamics; specifically, they modulate immune mediators via divergent effects on mRNA and protein levels ensuring a balanced inflammatory output.

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E-POSTERS

P01

INTERSTITIAL LUNG DISEASE PRESENTING AS ORGANIZING PNEUMONIA INDUCED BY NILOTINIB: A RARE BUT SEVERE ADVERSE EVENT

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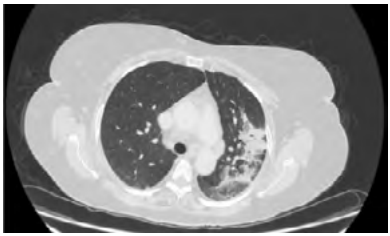
Chronic myeloid leukemia (CML) management is based almost exclusively on tyrosine kinase inhibitors (TKI), with allogeneic transplantation being indicated only for selected patients. Nilotinib is one of the second generation TKIs which have demonstrated improved responses, however, along with the risk of increased cardiovascular events. From a respiratory perspective, pleural effusions and pulmonary hypertension are listed as the commonest adverse events (AE). Interstitial lung disease (ILD) in the form of organizing pneumonia (OP, formerly known as BOOP) is a very rare AE of TKIs in CML. There are some case reports mentioning imatinib -induced OP which did not relapse after switching to nilotinib.

Here, we present a case of reversible OP in a patient receiving nilotinib. It is about a 74-year-old woman with an 8-year history of chronic phase CML. Initial treatment in 2017 was started with dasatinib, but was discontinued in 2021 due to bilateral pleural effusions and in July 2021 nilotinib was commenced with good tolerance. In December 2024, laboratory examinations revealed increased white blood cell count and inflammatory markers (ESR, CRP), with the patient being asymptomatic, apart from feeling fatigued. Investigations included chest x-ray and abdominal ultrasound, which were unremarkable. Computed tomography (CT) revealed scattered bilateral ground-glass opacities in the lungs. During the gradual tapering of nilotinib, a second CT chest in February 2025 showed a new consolidation on the left upper lobe with air bronchogram and scattered ground-glass opacities, with signs of fibrotic improvement. There was no clinical improvement with antibiotics and no etiological cause (infectious or autoimmune) was detected from the laboratory work-up. Thus, a diagnosis of nilotinib-induced ILD was concluded.

The patient received corticosteroid-based therapy followed by clinical and laboratory improvement. The molecular response was MR4 while being on nilotinib for a 4-year period. According to current guidelines, discontinuation of TKIs is feasible for patients in deep molecular response (MR4, MR 4.5) after at least 5 years of therapy. Hence, bearing in mind the above-mentioned AE and the good response of the underlying disease, nilotinib was stopped and the patient underwent monitoring. One year after the discontinuation, the patient's molecular response remains at MR4, with significant resolution of pulmonary infiltrates.



TKIs have significantly improved the prognosis of CML. However, with the increasing use of targeted therapies, new, rare AEs have emerged, increasing morbidity. ILD has been a rare AE of nilotinib that could cause irreversible fibrotic changes. Literature references, mainly case reports, are limited. The diagnosis of OP is mainly clinical on the basis of history of drug exposure, pneumonia improving on corticosteroids and drug discontinuation, as well as relapse when restarting the drug. Clinicians should be aware of such rare AE in order to manage them on a timely manner.

P02

PRECLINICAL VALIDATION OF CRISPR/CAS GENOME EDITING APPROACHES AS ADVANCED THERAPY FOR HBBIVS$^{I\text{-}110(G>A)}$ THALASSEMIA

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β-Thalassemia, a global single-gene disorder, is caused by deficient β-globin production, with the prevalent mutation HBBIVS$^{I\text{-}110(G>A)}$ creating an aberrant intronic splice site. This mutation has a high carrier frequency in Cyprus (76%) and many EU countries (>20%). A mutation-specific gene therapy has been developed using two approaches: a DSB-dependent approach with CRISPR/Cas9 RNA-guided nuclease (IVSI-110 RGN) and a DSB-independent approach with a nearly PAM-less SpG Adenine Base Editor (SpG7 ABE). Both methods disrupt HBBIVSI-110 abnormal splicing elements, achieving clinically relevant efficiencies in patient-derived HSCs in vitro. IVSI-110 RGN introduces indels via the non-homologous end joining repair mechanism, while SpG7 ABE uses targeted base (T>C) substitutions.

The project aims to advance these methods by conducting preclinical evaluations of edited cells both in vitro and in vivo using chimeric NBSGW mice, specifically engineered to facilitate human HSC engraftment without irradiation. The primary objective is to confirm the therapy's readiness for clinical trials, focusing on efficacy, safety, and the long-term repopulation (LTR) potential of modified cells. Additionally, the study seeks to compare the mutation-specific approach with a universal therapy targeting the erythroid BCL11A enhancer element for HbF induction (sg1617 RGN), recently FDA-approved as the first CRISPR therapy for sickle cell disease.

RGNs and ABE were delivered via nucleofection to mobilized HBBIVS$^{I\text{-}110(G>A)}$ patient-derived HSCs as ribonucleoprotein complexes (RNPs) and in vitro transcribed mRNAs, respectively. The therapeutic potential was evaluated in vitro through induced erythroid differentiation (ED) cultures, assessing correction at DNA (on- and off-targeting, Sanger sequencing), protein (RP-HPLC), and late-stage ED levels (flow cytometry), as well as clonogenic assays for erythroid and myeloid lineage potential. In vivo assessment involved xenotransplantation in NBSGW mice to evaluate LTR potential 16 weeks post-transplantation (flow cytometry).

Overall, both mutation-specific genome editors let to high on-targeting (IVSI-110 RGN 85%; SpG7 ABE (T>C): IVSI-106 and -108: ~40%; -109: ~18%) with undetected off-targeting, and moderate disrutprion of erythroid BCL11A enhancer element (sg1617 RGN: ~40%). RP-HPLC analysis of the in-vitro ED cultures, showed significant increase of HBB/HBA ratios to normal levels (0.9-1) in IVSI-110 RGN- and SpG7 ABE-genome edited cells and a significant increase of HBG/HBA ratios in the sg1617 RGN edited population (~0.51) relative to UT control (HBB/HBA: ~0.39; HBG/HBA: ~0.27). There was a clear correction of late-stage erythroid differentiation in the mutation-specific edited populations, while genome editing didn't affect erythroid and myeloid lineage potential of HSCs. Analysis of BM chimerism in xenotransplanted NBSGW mice showed high engraftment for all samples (hCD45+: 65% and hCD34+ves: ~6.5%). When comparing genome editing levels between BM bulk inputs and primary recipient BM cells, a 50% reduction was observed in IVSI-110 RGN, while sg1617 RGN showed consistent levels, and SpG7 ABE demonstrated a 20% increase.

Even though, analysis of the biosafety of the genome editing tools is still in progress, the current data indicates ABE SpG7 as the most promising approach for clinical application, since therapeutic levels were achieved while the erythroid and myeloid-lineage and LTR capacity of the edited HSC population was maintained.

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P03

PROGNOSTIC SIGNIFICANCE OF NEUTROPHIL-TO-LYMPHOCYTE RATIO AT DIAGNOSIS IN ACUTE MYELOID LEUKEMIA

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The neutrophil-to-lymphocyte ratio (NLR) has been studied as a prognostic factor in inflammatory diseases, cardiovascular disorders, solid tumors, and hematologic malignancies (non-Hodgkin/Hodgkin lymphoma, multiple myeloma). In several cases, an elevated ratio has been associated with poor prognosis; however, no specific cut-off value has been established to date. Few studies exist in acute myeloid leukemia (AML), and it remains unclear whether NLR constitutes an independent prognostic factor.

We retrospectively evaluated the potential association of NLR with other prognostic factors, remission achievement, and survival in 379 patients with AML. The cohort included 216 men and 163 women, with a median age of 51 (range 14–75) years, who received intensive treatment at our center. De novo AML was present in 283/379 patients; favorable cytogenetics were observed in 45 patients, intermediate in 244 (182/244 with normal karyotype), and adverse in 78 patients.

Complete remission (CR) was achieved in 265/379 patients (70%), refractory disease was observed in 100/379, and early death during induction (TRM) occurred in 14/379 patients. Among those tested, 55/255 had FLT3-ITD mutation and 65/166 were NPM1 positive. White blood cell counts at diagnosis ranged from 500 to 250,000/μL (median 12,000/μL), and differential counts were assessed on peripheral blood smears. Absolute lymphocyte count (ALC) ranged from 2,200 to 60,000/μL (median 2,200/μL), while absolute neutrophil count (ANC) ranged from 10 to 45,000/μL (median 1,050/μL).

The median NLR value was low, at 0.44 (range 0–600). Cut-off values of 0.44, 1, and 2 were used (110 patients had NLR >1 and 50 had NLR >2). Overall survival (OS) did not differ significantly between patients with NLR below or above the median value of 0.44 (5-year OS 37.5% vs 32.8%, p=0.27), nor for a cut-off of 1 (5-year OS 38.7% vs 25.7%, p=0.066). However, a decrease in OS was observed with increasing NLR. When a cut-off of 2 was applied, the difference in OS became statistically significant (36.8% vs 24.4%, median OS 9 vs 20 months, p=0.029).

Disease-free survival did not differ significantly for any of the NLR cut-off values. In multivariate analysis (Cox regression), significant factors for OS included age, cytogenetic risk group, and secondary disease, whereas NLR and white blood cell count were not significant. The potential association of NLR with other prognostic indices and CR achievement was also evaluated (t-test, ANOVA). For all cut-off values (0.44, 1, 2), no correlation was found with white blood cell count, cytogenetic group, age, or FLT3 mutations; however, an association with secondary disease was observed. Additionally, NLR >1 (and NLR >2) was associated with remission achievement (p=0.016 and p<0.001, respectively), with CR rates of 62.4% for NLR >1 versus 74% for NLR ≤1.

In conclusion, an NLR >1 was negatively associated with remission achievement. An NLR >2 was also negatively associated with overall survival in univariate analysis; however, it was not an independent prognostic factor, possibly due to its association with other variables such as secondary disease, as also demonstrated in our study.

P04

SUCCESSFUL PREGNANCY OUTCOME IN A PATIENT WITH PAROXYSMAL NOCTURNAL HEMOGLOBINURIA TREATED WITH RAVULIZUMAB

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Paroxysmal nocturnal hemoglobinuria (PNH), a rare acquired clonal disorder of the hematopoietic stem cell, is characterized by chronic hemolysis, increased thrombotic risk, and bone marrow failure due to complement-mediated hemolysis. During pregnancy, increased complement activation is associated with higher maternal morbidity and mortality, mainly due to worsening anemia, intravascular hemolysis, and an increased risk of thrombosis. Significant fetal complications have also been reported, including preterm delivery and miscarriage.

Complement inhibitors have significantly improved patients' quality of life and overall survival. The C5 inhibitors eculizumab and ravulizumab demonstrate comparable efficacy in symptom control and thrombosis prevention. During pregnancy, there is extensive experience with eculizumab, showing improved outcomes and an acceptable safety profile for both mother and fetus. However, despite the increasing use of ravulizumab, data regarding its use in pregnancy remain limited. Furthermore, it is unclear whether dose adjustment is required during pregnancy, although some reports suggest that this may be necessary. Herein, we present a case of a patient with PNH and a successful pregnancy outcome under ravulizumab treatment.

A 33-year-old woman was diagnosed with classical PNH in 2023 following evaluation for abdominal pain and hemolytic anemia. Her medical history included one spontaneous miscarriage one year before. After appropriate vaccination, treatment with intravenous ravulizumab was initiated (loading dose followed by maintenance dosing of 3.000 mg intravenously every 8 weeks). The patient achieved a good response (Hb >10 g/dL), with persistent mild anemia without transfusion requirements, and opted to continue ravulizumab due to her excellent clinical status.

In October 2024, a 5-week pregnancy was confirmed. Switching to eculizumab was discussed, however, after shared decision-making, continuation of ravulizumab was chosen, with monitoring of terminal complement activity (soluble C5b-9). The patient was initially started on aspirin, and from the second trimester, low molecular weight heparin was added (enoxaparin 40 mg subcutaneously once daily, later increased to 40 mg twice daily).

She continued ravulizumab at 3.000 mg, with a decline in hemoglobin levels requiring transfusions (total of 7 units of packed red blood cells) and an increase in LDH levels (up to 412 IU/L, normal level <250 IU/L). Soluble C5b-9 levels were regularly measured using ELISA and showed a progressive increase after the 26th week of gestation (from 196 to 214, 234, and 361 ng/mL, normal level <245 ng/mL), prompting an increase in ravulizumab dosing frequency. Two doses were administered at shorter intervals, with the last dose given prior to delivery.

At 37 weeks of gestation, the patient delivered a healthy neonate weighing 3,62 kg via cesarean section. To date, both mother and infant remain in good clinical condition without complications, and the mother continues treatment with laboratory parameters comparable to pre-pregnancy levels.

In conclusion, PNH is a complex disease with potentially life-threatening complications during pregnancy. Although eculizumab remains the standard of care in this setting, ravulizumab appears to offer a comparable safety and efficacy profile. Monitoring terminal complement activity may assist in dose adjustment during the perinatal period.

P05

LINKING MICROPARTICLES ISOLATION TECHNIQUES TO IN VITRO MODELS OF INFLAMMATION

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Purpose of the study:
Umbilical cord blood (UCB) represents a rich source of hematopoietic stem cells (HSCs). These cells are capable of releasing extracellular vesicles, including microparticles (MPs) generated during cellular activation or apoptosis. Microparticles act as carriers of bioactive molecules, participate in intracellular and intercellular communication, and may influence multiple cellular processes, such as cytokine regulation or protein expression. The study aimed to characterize the vesicle population using different isolation methods and to investigate the effect of microparticles on hematopoietic cell cultures under inflammatory conditions. To determine their potential immunomodulatory properties, an in vitro inflammation model was applied using lipopolysaccharide (LPS) derived from E. coli.

Material and Methods:
MPs derived from UCB were co-cultivated with THP-1 cells and HL-60 cells, separately. Three different microparticle isolation protocols were evaluated: i) microparticles derived from whole plasma, ii) microparticles obtained after plasma concentration, and iii) CD34+ microparticles (CD34+MPs) derived from whole plasma after immunomagnetic separation. CD34+MPs were isolated from the plasma of UCBs after centrifugation and magnetic bead MACS purification (Miltenyi Biotec). The number of CD34+ MPs was estimated by flow cytometry using CD34-PE and Annexin V-FITC Abs. The particle size distribution was determined by dynamic light scattering (DLS) using a Zetasizer Nano ZS (Malvern Instruments Ltd, Malvern, UK) equipped with a 633 nm He-Ne laser. Samples were diluted 10-fold with water prior to analysis, and measurements were performed at 25 °C in triplicate. THP-1 cells, a human monocytic leukemia cell line, maintained at 2×105 cells/ml in RPMI 1640 medium supplemented with 10% FCS, 1% penicillin-streptomycin and 1% L-glutamine 200mM. THP1 cells (9×105/ml) were differentiated into macrophages using 200 nM phorbol 12-myristate 13-acetate (PMA, Sigma-Aldrich) for 24hrs, followed by 24hrs incubation with fresh medium. Differentiated and non-differentiated THP-1 cells were cultivated with microparticles derived from whole and concentrated plasma and 100 ng/mL Lipopolysaccharide (LPS). The HL60 promyelocytic leukemia cell line was maintained in RPMI (Life Technologies) supplemented with 10% fetal calf serum (FCS) and 2% penicillin-streptomycin. After 24 hours of incubation, RNA was extracted from the cells, cDNA was synthesized, and PCR was performed to evaluate IL-1β gene expression.

Results:
DLS illustrated that the particle population after plasma concentration was more homogeneous, consisting of particles with median size 194,4nm, with 87.82% corresponding to microparticle size. The percentage of 91.7nm vesicles derived from CD34+ selection was 89.6%. The results illustrated no reduction in cell viability, neither in THP-1 cells nor in HL-60 cells. Regarding gene expression in THP-1 cells, LPS stimulation increased the expression of the cytokine IL-1β, while the effect of microparticles varied depending on the isolation protocol employed. Specifically, microparticles isolated from whole blood plasma induced an increase in IL-1β expression, whereas microparticles obtained after plasma concentration did not alter gene expression levels. In addition, LPS increased the expression of IL-1β in HL-60 cells, while CD34+ microparticles did not alter its expression.

Conclusions:
Overall, the findings suggest that the isolation method of the microvesicles reflects size differences and has various effect on IL1 β gene expression in THP-1 derived monocytes and macrophages. However additional experiments are required to determine their effects in HL-60 cells.

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