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Cyprus Society of Haematology

Under the auspices of

Hellenic Society of Haematology

Israeli Society of Hematology and Blood Transfusion

7th GRECO - ISRAELI Hematology Meeting

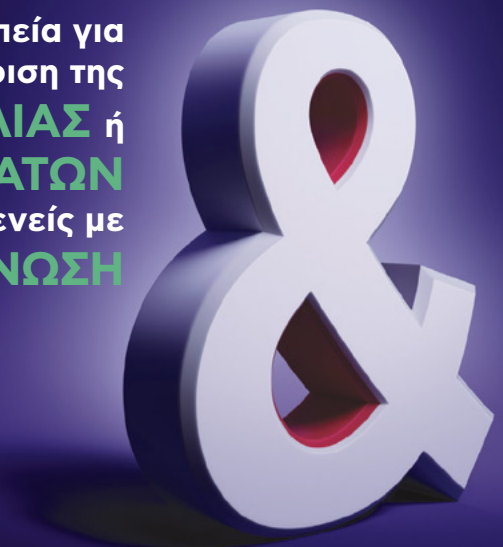
8th-10th MAY 2025

Porto Palace Hotel
THESSALONIKI
GREECE

FINAL PROGRAM
& ABSTRACT BOOK

Ο 1ος εγκεκριμένος JAK1/2 & ACVR1 αναστολέας.[1]

Μια θεραπεία για τη διαχείριση της ΣΠΛΗΝΟΜΕΓΑΛΙΑΣ ή των ΣΥΜΠΤΩΜΑΤΩΝ σε ασθενείς με ΜΥΕΛΟΪΝΩΣΗ



μέτρια έως σοβαρή ΑΝΑΙΜΙΑ[1]

Για την Περίληψη των Χαρακτηριστικών του Προϊόντος σκανάρετε το **QR code.** Σε έντυπη μορφή είναι διαθέσιμη κατόπιν αιτήσεως στην εταιρεία.

Omjjara
momelotinib

Ποιοτική και Ποσοτική σύνθεση:
Κάθε επικαλυμμένο με λεπτό υμένιο δισκίο Omjjara 100, 150, 200mg περιέχει μονοϋδρική διυδροχλωρική μομελοτινίμπη που ισοδυναμεί με 100, 150, 200mg μομελοτινίμπης αντίστοιχα. Έκδοχο με γνωστή δράση: 50.8, 76.1, 101.5mg μονοϋδρικής λακτόζης ανά δισκίο 100, 150, 200mg αντίστοιχα.

Σύνοψη του προφίλ ασφάλειας[1]:
Η ασφάλεια του Omjjara αξιολογήθηκε σε τρεις τυχαιοποιημένες, ελεγχόμενες με δραστικό παράγοντα, πολυκεντρικές μελέτες σε ενήλικες με μυελοΐνωση (MOMENTUM, SIMPLIFY-1, και SIMPLIFY-2). Σε αυτές οι πιο συχνές ανεπιθύμητες ενέργειες ήταν διάρροια (23%), θρομβοπενία (21%), ναυτία (17%), κεφαλαλγία (13%), ζάλη (13%), κόπωση (12%), εξασθένιση (11%), κοιλιακό άλγος (11%) και βήχας (10%). Η πιο συχνή σοβαρή ανεπιθύμητη ενέργεια (Βαθμού ≥ 3) ήταν η θρομβοπενία (12%).

Βιβλιογραφία
1. Omjjara (momelotinib) Περίληψη Χαρακτηριστικών του Προϊόντος, Ιανουάριος 2024

Λιανική Τιμή: € 5141,54

% επιχορήγησης από τους οργανισμούς κοινωνικών ασφαλίσεων: αναμένεται.

Φαρμακευτικό προϊόν για το οποίο απαιτείται ιατρική συνταγή. Η έναρξη της θεραπείας γίνεται σε νοσοκομείο και μπορεί να συνεχίζεται εκτός νοσοκομείου υπό την παρακολούθηση ειδικού ιατρού. Τα ανωτέρω ισχύουν κατά την ημερομηνία σύνταξης του υλικού/καταχώρησης. Παρακαλούμε επικοινωνήστε με την εταιρεία για επιβεβαίωση πλήρως ενημερωμένων δεδομένων, για οποιαδήποτε πληροφορία ή/και αναφορά Ανεπιθύμητων Ενεργειών στο τηλέφωνο 210 6882100.

Πριν τη συνταγογράφηση συμβουλευτείτε την Περίληψη Χαρακτηριστικών του Προϊόντος η οποία διατίθεται κατόπιν αιτήσεως στην εταιρεία.

▼Το φάρμακο αυτό τελεί υπό συμπληρωματική παρακολούθηση. Αυτό θα επιτρέψει τον γρήγορο προσδιορισμό νέων πληροφοριών ασφάλειας. Ζητείται από τους επαγγελματίες υγείας να αναφέρουν οποιεσδήποτε πιθανολογούμενες ανεπιθύμητες ενέργειες.

Βοηθήστε να γίνουν τα φάρμακα πιο ασφαλή και Αναφέρετε ΟΛΕΣ τις ανεπιθύμητες ενέργειες για ΟΛΑ τα φάρμακα Συμπληρώνοντας την "ΚΙΤΡΙΝΗ ΚΑΡΤΑ"
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WELCOME ADDRESS

Dear colleagues,

It's a privilege welcome you to the **7th Greco-Israeli Hematology Meeting**, which will be held in **Thessaloniki**, **8-10 May 2025**.

This 7th one aims to present the new data in the most important topics of hematology to be presented in the scientific program. Colleagues from Israel, Cyprus and all over around Greece will join us in another successful and of high scientific level meeting. We also continue the successful sessions of working groups, which are active, offering the participants the opportunity to discuss their work and research.

We invite you all to join the meeting to present data, discuss and work together.

The Co-Chairmen

Damianos Sotiropoulos
G. Papanikolaou Hospital
Thessaloniki, Greece

Benjamin Brenner
Rambam Health Care Campus
Haifa, Israel

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COMMITTEES

Co-chairs:

Benjamin Brenner, Damianos Sotiropoulos

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SCIENTIFIC PROGRAM

Thursday 8th May 2025

09.30-10.30	**LYMPHOMA WORKING GROUP**
10.30-11.00	**MEETING OPENING**
11.00-13.00	**JOINT SESSION WITH TEL-AVIV MYELOPROLIFERATIVE NEOPLASMS** Chair: **D. Sotiropoulos, G. Vasilopoulos**
11.00-11.30	Is it MDS, CCUS, MDS/MPN, or CMML? **A. Tefferi**
11.30-12.00	Therapy of MPN with interferon: historical perspective, current recommendations and future directions **P. Heike**
12.00-12.30	Diagnosis of myelofibrosis from peripheral blood **G. Dadi**
12:30-13.00	Use of NGS myeloid gene panel in the management and diagnosis of MDS and MPN: a case-based discussion **V. Vainstein**
13.00-13.30	**LECTURE**
	INFECTIOUS DISEASES Candida Auris: a rising threat **A. Nikopoulou**
13.30-14.30	***Lunch break***

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Thursday 8th May 2025

14.30-15.30	**ROUND TABLE**
	RARE DISEASES Chair: **E. Mandala** New medicines for old diseases **E. Gavrilaki** HLH associated hyper inflammation: from bedside to the bench and back. **A. Zoref-Lorenz**
15.30-16.00	***Coffee break***
16.00-17.00	**ROUND TABLE**
	CELLULAR THERAPIES Chair: **I. Sakellari, O. Beyar-Katz** Enhancing CAR-T cell therapy: the power of combinational approaches **O. Beyar-Katz** An update on haploidentical HSCT **I. Baltadakis**
17.00-17.30	**LECTURE**
	CAR-T CELLS Chair: **D. Sotiropoulos** In-house CAR-T cells: G. Papanikolaou hospital project **E. Yannaki**
17.30-18.00	***Coffee break***

Thursday 8th May 2025

18.00-19.00	**Opening Ceremony**
18.00-18.30	**Greetings**
18.30-19.00	**LECTURE** The antiquities in Thessaloniki metro stations: 23 centuries of history **E. Tsigarida**
19.00	**Welcome reception**

Friday 9th May 2025

08.00-09.00	**MULTIPLE MYELOMA WORKING GROUP**
09.00-10.00	**MPN WORKING GROUP**
10.00-11.00	**ROUND TABLE**
	HEMATOLOGICAL EMERGENCIES Chair: **M. Michail** Hematological emergencies in AML **P. Tsirigotis** Bleeding emergencies in hematological patients **G. Kaiafa**
11.00-11.30	**LECTURE CLL**
	Chair: **N. Stavroyianni** Ten years of BTK inhibitors: what next in CLL management **G. Itchaki**
11.30-12.00	***Coffee break***
12.00-13.00	**ROUND TABLE**
	MULTIPLE MYELOMA Chair: **I. Avivi, M.A. Dimopoulos** Early detection of plasma cell disorders: Can we? Should we? And if so, how? **I. Avivi** Drug combinations in newly diagnosed symptomatic multiple myeloma: How many drugs finally? **M.A. Dimopoulos**

Friday 9th May 2025

13.00-13.30	**LECTURE**
	QUALITY MANAGEMENT IN CELLULAR THERAPIES Chair: **I. Batsis** JACIE accreditation in cellular therapies **S. Gounopoulos**
13.30-14.30	***Lunch break***
14.30-15.30	**ROUND TABLE**
	LYMPHOMAS Chair: **N. Horowitz, A. Kioumi** Treating advanced-stage mycosis fungoides and sezary syndrome: the hematologist's perspective **S. Ringelstein-Harlev** First line therapy in Hodgkin's lymphoma **T. Vassilakopoulos**
15.30-16.30	**SATELLITE SYMPOSIUM**
	NOVEL APPROACHES IN THE TREATMENT OF HEMATOLOGICAL MALIGNANCIES Chair: **M.A. Dimopoulos, M. Pagoni** Recent advances in myelofibrosis: a guide to new therapeutic strategies for anemic patients **D. Sotiropoulos** Advancements in therapies for relapsed/refractory multiple myeloma: clinical impact and value **M. Gavriatopoulou** *Sponsored by* GSK
16.30-17.00	***Coffee break***

Friday 9th May 2025

17.00-18.00	ROUND TABLE

ACUTE LEUKEMIAS
Chair: **M. Spanoudakis**

Bispecific molecules and CAR-T cells in ALL
M. Angelopoulou

AML in 2025: can we improve the standard of care?
O. Wolach

Saturday 10th May 2025

09.30-10.30	**ROUND TABLE**
	HEMOGLOBINOPATHIES Chair: **A. Kourakli-Symeonidou** Recent advances in the treatment of transfusion dependent thalassemia **A. Pyrovolaki** Thrombosis in hemoglobinopathies **E. Vlachaki**
10.30-11.30	**ROUND TABLE**
	THROMBOSIS AND HEMOSTASIS Chair: **B. Brenner, V. Garypidou** Hormonal therapy and thrombosis: new is not always better **E. Papadakis** Neuropsychiatric aspects of hypercoagulability **B. Brenner**
11.30-12.00	***Coffee break***
12.00-13.00	**SATELLITE SYMPOSIUM**
	EVOLVING TREATMENT MANAGEMENT FOR MF AND PNH PATIENTS Chairs: **M. Dimou, I. Asimakopoulos** Optimizing treatment strategies in myelofibrosis for improved patient outcomes **M. Dimou** Paroxysmal nocturnal hemoglobinuria: challenges in the management and patient care **I. Asimakopoulos** *Sponsored by* NOVARTIS

Saturday 10th May 2025

13.00-14.00	**ROUND TABLE**
	MDS Chair: **A. Symeonidis, M. Antoniadis** Microenvironment in MDS **Ch. Pontikoglou** What new in MDS treatment? **E. Hatzimichael**
14.00-15.00	***Lunch break***
15.00-15.30	**LECTURE**
	CML Chair: **P. Panagiotidis** How I treat CML patients after 1st line TKI failure **M. Dimou**
15.30-16.00	***Coffee break***
16.00-17.00	**Plenary Session** Chair: **B. Brenner, D. Sotiropoulos**
O01	**HLA-G REACTIVATION VIA EPIGENOME EDITING AS A NEW APPROACH TO IMMUNE TOLERANCE** **Simonopoulos P.**[1,2], Christofi P.[1,3], Papageorgiou V.[1,4], Papadopoulos P.[1,4], Sakellari I.[1], Galanis A.[2], Psatha N.[4], Yannaki E.[1,5], Papadopoulou A.[4] *[1]Gene and Cell Therapy Center, Hematology Department - Hematopoietic Cell Transplantation Unit, George Papanikolaou General Hospital, Thessaloniki, [2]Department of Molecular Biology and Genetics, Faculty of Health Sciences, Democritus University of Thrace, [3]Institute of Cell Therapy, University of Patras, [4]Department of Genetics, Development and Molecular Biology, School of Biology, Aristotle University of Thessaloniki, [5]Department of Medicine, Division of Hematology, University of Washington*

Saturday 10th May 2025

O02

PROTECTIVE EFFECTS OF UCB-DERIVED CD34+ MICROPARTICLES AGAINST LPS-INDUCED INFLAMMATION IN MONONUCLEAR AND HL-60 CELLS

Nasiadis A.[1,2], Xagorari A.[1], Alexandridou A.[1,3], Sianidou K.[1,4], Kritis A.[4], Papadopoulou A.[3], Sakellari I.[1], Chlichlia K.[2], Sotiropoulos D.[1]

[1]Public Cord Blood Bank of Thessaloniki, Department of Hematology-BMT Unit, George Papanicolaou General Hospital, Thessaloniki, [2]Department of Molecular Biology and Genetics, Democritus University of Thrace MSc program Infectious Diseases - International Medicine: from the laboratory to clinical practice, [3]Department of Genetics, Development and Molecular Biology, School of Biology, Aristotle University of Thessaloniki, [4]Regenerative Medicine Center, Basic and Translational Research Unit of Special Unit for Biomedical Research and Education, Faculty of Health Sciences, School of Medicine, Aristotle University of Thessaloniki

O03

COMPARABLE OUTCOMES OF POINT-OF-CARE AND COMMERCIAL CD19 CAR-T THERAPIES: A PATIENT-MATCHED ANALYSIS IN LARGE B-CELL LYMPHOMA

Marcus R.[1], Avigdor A.[1], Greenbaum U.[2], Brown S.[3], Fried S.[1], Golan-Accav N.[1], Shem-Tov N.[1], Yerushalmi R.[1], Danylesko I.[1], Jacoby E.[1], Nagler A.[1], Shimoni A.[1], Itzhaki O.[1], Esensten J.[1], Ben Valid O.[1], Ballweg A.[4], Deschenes-Simard X.[4], Luttwak E.[4], Shah G.[4], Scordo M.[4], Dahi P.[4], Perales M.[4], Zuckerman T.[5], M Devlin S.[3], Henig I.[5], Yehudai-Ofir D.[5], Khatib H.[5], Shouval R.[5], **Beyar Katz O.**[5]

[1]Division of Hematology and Bone Marrow Transplantation, Chaim Sheba Medical Center, Tel HaShomer, Israel 2- School of Medicine, Faculty of Medical and Health Sciences, Tel Aviv University, Tel Aviv, Israel, [2]Department of Hematology, Soroka University Medical center and Faculty of Health and Science, Ben Gurion University of the Negev, Beer Sheva, Israel, [3]Department of Biostatistics and Epidemiology, Memorial Sloan Kettering Cancer Center, New York, NY, [4]Department of Medicine, Adult Bone Marrow Transplant Service, Cellular Therapy Service, Memorial Sloan Kettering Cancer Center, New York, NY, USA. 6- Department of Medicine, Weill Cornell Medical College, New York, NY, USA, [5]Department of Hematology and Bone Marrow Transplantation, Rambam Health Care Campus, Haifa. Ruth and Bruce Rappaport Faculty of Medicine, Technion, Haifa, Israel

Saturday 10th May 2025

O04 A NOVEL BLOOD-BASED BIOMARKER FOR EARLY DETECTION OF TREATMENT RESPONSE AND RELAPSE PREDICTION IN DIFFUSE LARGE B-CELL LYMPHOMA (DLBCL)

Rabizadeh E.[1,2], Gafter-Gvili A.[2,3,5], Itchaki G.[2,6], Ofran Y.[2,4], Avivi I.[2,8], Cherny I.[1,7], Gurion R.[2,3]
[1]Hemato-Oncology Research Laboratory, Felsenstein Medical Research Center, [2]Faculty of Medicine, Tel-Aviv University, [3]Institute of Hematology, Davidoff Cancer Center, [4]Shaarei-Tzedek Medical Center, [5]Internal Medicine A, Beilinson Hospital, Rabin Medical Center, [6]Hematology, Meir Medical Center, [7]Biochemistry Laboratory, Beilinson Hospital, Rabin Medical Center, [8]Sorasky Medical Center

O05 DUAL MRD MONITORING BY FLOW CYTOMETRY AND STR-PCR: ENHANCING MRD RISK ASSESSMENT IN PH (-) ALL POST-ALLOHCT

Douka V., **Kyriakou I.**, Kaliou M., Kika F., Avramidou R., Paleta A., Koravou E., Zerva P., Papathanasiou M., Marvaki A., Boussiou Z., Karavalakis G., Vadikolia C., Papalexandri A., Sakellari I.
George Papanikolaou General Hospital, Thessaloniki

O06 RITUXIMAB-DOSE-ADJUSTED EPOCH (R-da-EPOCH) IN PRIMARY MEDIASTINAL LARGE B-CELL LYMPHOMA (PMLBCL): A MULTINATIONAL REAL-LIFE STUDY

Vassilakopoulos T.[1], Ferhanoglu B.[2], Horowitz N.A.[3], Mellios Z.[4], Kaynar L.[5], Zektser M.[6], Symeonidis A.[7], Piperidou A.[1], Kalpadakis C.[8], Arapaki M.[1], Akay O.M.[9], Atalar S.C.[9], Katodritou E.[10], Leonidopoulou T.[11], Papageorgiou S.G.[12], Tadmor T.[13], Gutwein O.[14], Karakatsanis S.[15], Ganzel C.[16], Karianakis G.[17], Isenberg G.Y.[3], Gainaru G.[17], Vrakidou E.[17], Palassopoulou M.[18], Ozgur M.[19], Siakantaris M.[1], Paydas S.[20], Tsirigotis P.[12], Tsirogianni M.[21], Hatzimichael E.[22], Tuglular T.F. [23], Chatzidimitriou C.[1], Liaskas A.[1], Lefaki M.[1], Labropoulou P.[24], Kanellias M.[25], Zikos P.[26], Koumarianou A.[27], Gafter-Gvili A.[28], Angelopoulou M.K.[1], Karmiris T.[4], Gurion R.[28]
[1]Department of Hematology and Bone Marrow Transplantation, National and Kapodistrian University of Athens, Laikon General Hospital, Athens, Greece, [2]American Hospital, Istanbul, Turkey, [3]Rambam Medical Center, Haifa, Israel, [4]Department of Hematology and Lymphoma, Evangelismos General Hospital, Athens, Greece, [5]Erciyes University, Turkey, [6]Soroka

Saturday 10th May 2025

Medical Center, Beer Sheba, Israel, [7]Hematology Division, Department of Internal Medicine, University of Patras, Greece, [8]Department of Hematology, University General Hospital of Heraklion, Crete, Greece, [9]Koc University, Turkey, [10]Department of Hematology, Theagenion Anticancer General Hospital, Thessaloniki, Greece, [11]Department of Hematology, Sismanoglion General Hospital, Athens, Greece, [12]Second Propaedeutic Department of Internal Medicine, National and Kapodistrian University of Athens, Attikon General Hospital, Greece, [13]Bnai Zion Medical Center, Israel [14]Shamir Medical Center, Be'erYa'akov, Israel, [15]Third Department of Internal Medicine, National and Kapodistrian University of Athens, Sotiria Hospital, Athens, Greece, [16]Shaare Zedek Medical Center, Jerusalem, Israel, [17]Hygeia Hospital, Athens, Greece, [18]Department of Hematology, University Hospital, University of Thessaly, Larisa, Greece, [19]Kocaeli University, Turkey, [20]Cukurova University, Turkey, [21]Department of Hematology and Bone Marrow Transplantation, Saint Savvas Regional Cancer Hospital, Athens, Greece, [22]Department of Hematology, University of Ioannina, Greece, [23]Marmara University, Turkey, [24]Department of Hematology, Metaxa Cancer Hospital, Piraeus, Greece, [25]Department of Therapeutics, National and Kapodistrian University of Athens, Alexandra Hospital, Athens, Greece, [26]Hematology Department, General Hospital of Patras Agios Andreas, Greece, [27]Department of Hematology, Metropolitan Hospital, Athens, Greece, [28]Rabin Medical Center, Petach Tikva, Israel

17.00-17.30 **Closing remarks**

E-POSTERS

P01

LYMPHOID BLAST CRISIS OF PEDIATRIC CHRONIC MYELOID LEUKEMIA PRESENTING AS B-ACUTE LYMPHOBLASTIC LEUKEMIA

Podberezin M.
Maimonides Medical Center, State University of New York

P02

P53 MUTATIONS: ARE ALL THE SAME? ARIADNE'S THREAD IN APPROACHING CLASSIFICATION MAZE OF P53-MUTATED MYELODYSPLASTIC SYNDROME/ ACUTE MYELOID LEUKEMIA

Podberezin M.
Maimonides Medical Center, State University of New York

P03

ADENOVIRUS DETECTION IN UMBILICAL CORD BLOOD UNITS, IMPROVING SAFETY OF TRANSPLANTATION

Keramari S.[1,2,3], Xagorari A.[1], Sakellari I.[2], Sotiropoulos D.[2], Savopoulos C.[3], Kaiafa G.[3]
[1]Department of Pediatrics, AHEPA University Hospital of Thessaloniki, [2]Public Cord Blood Unit, Hematology Department and Bone Marrow Transplantation Unit, George Papanicolaou General Hospital, Thessaloniki, [3]First Propaedeutic Department of Internal Medicine, AHEPA University Hospital of Thessaloniki

P04

ENDOVASCULAR TREATMENT OF A PATIENT WITH ACQUIRED HEMOPHILIA A: DIAGNOSTIC AND THERAPEUTIC CHALLENGES

Makris E.[1], Bountola S.[1], Gougoula V.[1], Patriarcheas V.[1], Tsiatsiou P.[2], Ztriva E.[1], Perifanis V.[1], Savopoulos C., Kaiafa G.[1]
[1]First Propaedeutic Department of Internal Medicine AHEPA University Hospital of Thessaloniki, Aristotle University of Thessaloniki, [2]Department of Microbiology, AHEPA University Hospital of Thessaloniki, School of Medicine, Aristotle University of Thessaloniki

P05

PILOT TESTING OF A NATIONAL REGISTRY FOR PATIENTS WITH SEVERE THROMBOEMBOLIC DISEASE OR RARE HEREDITARY AND ACQUIRED THROMBOPHILIC DISORDERS

Danilatou V., Kyriakou E., Nomikou E., Pergantou H., Papadakis E., Girtovitis F.
European University of Cyprus, Cyprus

P06

UPREGULATION OF CCR5/CCR2 DURING IMMUNE CELL-ASSOCIATED NEUROTOXICITY SYNDROME FOLLOWING CAR-T CELL THERAPY

Rozenberg A.[1], Shelly S.[1], Winer R.[1], Fineman R.[1], Ringelstein-Harlev S.[1], Henig I.[1], Yehudai-Ofir D.[1], Zuckerman T.[1], Tadmor T.[3], Harel R.[2], Ganelin-Cohen E.[4], **Beyar Katz O**.[1]
[1]Rambam Health Care Campus, Israel, [2]Emek Medical Center, Israel, [3]Bnai Zion, Israel, [4]Schneider Children's Medical Center of Israel

P07

CYTOF ANALYSIS IN PHASE 2 PROSPECTIVE INTERVENTIONAL STUDY: NIVOLUMAB ADDITION TAILORED BY CAR-T CELL EXPANSION IN PATIENTS WITH STABLE OR PROGRESSIVE LARGE B-CELL LYMPHOMA DURING LYMPHODEPLETION

Khatib A.[3], Grau A.[1], Amit O.[2], Zuckerman T.[1], Fridberg G.[2], Setter-Marco N.[3], **Beyar Katz O**.[1], Ram R.[2]
[1]Rambam Health Care Campus, Israel, [2]Tel Aviv Sourasky Medical Center, Tel Aviv, Israel, [3]The Ruth & Bruce Rappaport Faculty of Medicine, Technion-Israel Institute of Technology, Haifa, Israel

P08

CD34+ DERIVED MICROPARTICLES INCREASE THE ANTIOXIDANT MECHANISM OF UMBIICAL CORD BLOOD

Kaiopoulos G.[1,2], Ioannidou A.[1,2], Papaioannou D.[1], Sakellari I.[1], Sotiropoulos D.[1], Touraki M.[2], **Xagorari A**.[1]
[1]Public Cord Blood Bank, Department of Hematology-BMT Unit, George Papanicolaou General Hospital, Thessaloniki, [2]Department of Genetics, Development and Molecular Biology, School of Biology, Aristotle University of Thessaloniki

P09

AGE-SPECIFIC DIFFERENCES IN CEREBRAL SINUS VENOUS THROMBOSIS: A RETROSPECTIVE COHORT STUDY FROM A TERTIARY CARE CENTER

Peley Neuman Y.[1], Sawaid K.[1,2], Cohen O.[3,4], Lalezari S.[5,6], Barg A.[5,6], Waldman Radinsky L.[5,6], Kenet G.[5,6], Lubetsky A.[5,6], **Levy-mendelovich S.**[5,6,7]
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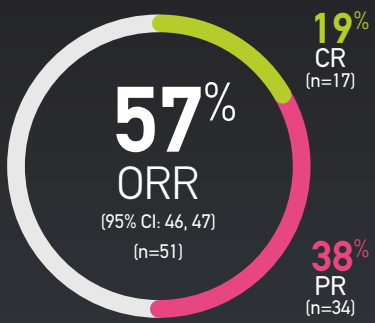

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BTK=τυροσινική κινάση του Bruton, CI=διάστημα εμπιστοσύνης, CR=πλήρης ανταπόκριση, IRC=ανεξάρτητη επιτροπή επιθεώρησης, LDi=μέγιστη εγκάρσια διάμετρος, ΛΚΜ=λέμφωμα από κύτταρα του μανδύα, ORR=ποσοστό συνολικής ανταπόκρισης, PR=μερική ανταπόκριση.

Βιβλιογραφία:
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ABSTRACT BOOK
ORAL PRESENTATIONS
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ORAL PRESENTATIONS

O01

HLA-G REACTIVATION VIA EPIGENOME EDITING AS A NEW APPROACH TO IMMUNE TOLERANCE

Simonopoulos P.[1,2], Christofi P.[1,3], Papageorgiou V.[1,4], Papadopoulos P.[1,4], Sakellari I.[1], Galanis A.[2], Psatha N.[4], Yannaki E.[1,5], Papadopoulou A.[4]

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Introduction and Purpose: Human leukocyte antigen G (HLA-G) is an immunomodulatory molecule, crucial for orchestrating maternal-fetal tolerance during pregnancy, thus preventing immune rejection of the semi-allogeneic fetus. After birth, HLA-G becomes epigenetically silenced, shifting immune regulation. Epigenome editing has emerged as a promising strategy to reactivate silenced genes, without altering the DNA sequence. We aimed to reactivate HLA-G gene using epigenome editing, by identifying the most effective combination of CRISPR activators (CRISPRa) and HLA-G cis-regulatory elements (cCREs) and assess its immunosuppressive potential.

Material and Methods: We identified four HLA-G cCREs: two promoter-like regions (PR1, PR2) and two enhancer-like regions (ER1, ER2), based on chromatin accessibility and post-translational histone modifications. Four CRISPRa/dCas9 systems (dCas9/VPR, dCas9/MSK1, dCas9/p300, dCas9/SunTag) and 20 non-overlapping guide-RNAs (gRNAs) targeting HLA-G cCREs (5 gRNAs/cCRE) were evaluated. gRNAs were delivered into K562 cells expressing the CRISPRa and HLA-G expression was measured, by qRT-PCR and Flow Cytometry. Cells expressing the CRISPRa alone served as mock controls.

Results: The combination of dCas9/VPR with a gRNA pool targeting PR1, significantly tuned HLA-G expression, compared to mock controls, at both RNA (138,348±19,340-fold increase, p=0.036) and protein levels (21.8±3.1% vs 0.7±0.1%, p=0.0004). Other combinations of CRISPR activators and target sequences failed to upregulate HLA-G, suggesting PR1's regulatory dominance. Subsequent test, of five individual PR1-targeting gRNAs identified two highly effective gRNAs, with one demonstrating a superior HLA-G induction over mock control (38±6.1% vs 0.77±0.08%, p=0.00004). Im-

portantly, immunomagnetically enriched HLA-G+ PR1-edited K562 cells, significantly inhibited T-cell cytolytic activity compared to mock K562 cells (% lysis at 40:1 ratio: 32±3.7 vs 51.5±2.4, p=0.01), confirming HLA-G's immunosuppressive role.

Conclusions: Overall, we identified key regulatory sequences of HLA-G and demonstrated the feasibility of reactivating its expression, through CRISPR-epigenome editing. This approach presents a novel therapeutic strategy for modulating immune responses in transplantation and autoimmunity, potentially enhancing tolerance and reducing adverse immune reactions.

O02

PROTECTIVE EFFECTS OF UCB-DERIVED CD34+ MICROPARTICLES AGAINST LPS-INDUCED INFLAMMATION IN MONONUCLEAR AND HL-60 CELLS

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Introduction and Purpose: Umbilical cord blood is an alternative source of hematopoietic stem cells. Hematopoietic stem cells, during their activation or apoptosis, release enriched microparticles with the surface marker CD34⁺. MPs take place in intracellular and intercellular communication, as well as in a variety of activities such as cytokines' regulation or proteins' expression. UCB provides high sensitivity and specificity of neonatal early-onset sepsis caused by lipopolysaccharide. LPS is an endotoxin, major component of Gram-negative bacteria outer membrane and via TLR4/MD2/CD14 complex induces proinflammatory cytokines. The aim of the present study was to determine the effect of UCB derived CD34⁺ MPs on the viability of stimulated cord blood monocytes and HL60 cells by LPS (E. coli O128:B12).

Material and Methods: MNCs were also fixed with trichloroacetic acid and stained with SRB assay for cytotoxicity screening. The viability was measured with Trypan blue assay whereas MNCs were isolated with Ficoll-Paque density gradient centrifugation. The effect of LPS was studied on the gene expression of cellular receptors: CD14 and TLR4. Proteome profiler human cytokine array was conducted for the detection of cytokines or proteins related to inflammation.

Results: The results illustrated reduction of HL60 with LPS treatment (200ng/ml) in the presence or absence of CD34⁺ MPs and increased expression levels of CD14 and TLR4 while CD34⁺ MPs decreased their expression. The cytokines and proteins CCL1/I-309, CCL5/RANTES, ICAM-1/CD54, IFN-γ, IL-8 and MIF were increasingly detected in HL60. A notable decrease in the expression of CCL1/I-309, CCL5/RANTES and MIF was observed. MNCs were significantly ($p \leq 0,05$) reduced with LPS treatment at various concentrations and incubation times. CD34⁺ MPs significantly induced cytotoxicity in MNCs in the presence or absence of low concentration LPS compared to control.

Conclusions: UCB derived CD34⁺ MPs appear to play a potential anti-inflammatory and protective role against lipopolysaccharide, in both mononuclear cells (MNCs) and HL60 cells.

O03

COMPARABLE OUTCOMES OF POINT-OF-CARE AND COMMERCIAL CD19 CAR-T THERAPIES: A PATIENT-MATCHED ANALYSIS IN LARGE B-CELL LYMPHOMA

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Background: Point-of-care (POC) chimeric antigen receptor T-cell (CAR-T) therapy offers a shorter vein-to-vein time, but its efficacy compared to commercial CAR-T products remains uncertain. This study compares an autologous CD19 POC CAR-T product with commercial alternatives (axi-cel and tisa-cel) in patients with large B-cell lymphoma (LBCL).

Methods: We included LBCL patients who received CD19-directed CAR-T therapy after at least two prior treatments across three academic centers. Two commercial CAR-T products (axi-cel and tisa-cel) were compared with a POC CAR-T product using propensity score matching, adjusting for age, Karnofsky performance status (KPS), LDH levels, primary refractory disease, and transformed lymphoma. The primary outcomes assessed were overall survival (OS) and progression-free survival (PFS).

Findings: A total of 330 patients were included: 132 received axi-cel, 104 received tisa-cel, and 94 received POC CAR-T. Patients treated with POC CAR-T were younger, had better KPS, and higher rates of elevated LDH and primary refractory disease. POC CAR-T had a significantly shorter median vein-to-vein time (11 days) compared to axi-cel (38 days) and tisa-cel (44 days) ($p<0.001$). In propensity score matching analysis, POC showed a trend towards lower PFS compared to axi-cel (HR 1.54 [95% CI 1.00-2.37], $p=0.051$) and was similar to tisa-cel (HR 0.71 [95% CI 0.45-1.11], $p=0.13$). Aditionally, comparable OS was shown between POC, axi-cel (HR 1.35, $p=0.18$), and

tisa-cel (HR 0.85, p=0.48). POC CAR-T also showed lower rates of grade >2 cytokine release syndrome but higher rates of grade >2 immune effector cell-associated neurotoxicity syndrome (ICANS).

Interpretation: This is the first direct comparison of CD19 POC CAR-T with commercial products. POC CAR-T is a safe and effective treatment for LBCL, showing outcomes comparable to commercial CAR-T products. Its rapid vein-to-vein time may offer particular advantages for patients with rapidly progressing disease.

O04

A NOVEL BLOOD-BASED BIOMARKER FOR EARLY DETECTION OF TREATMENT RESPONSE AND RELAPSE PREDICTION IN DIFFUSE LARGE B-CELL LYMPHOMA (DLBCL)

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Background: Diffuse large B-cell lymphoma (DLBCL) is an aggressive malignancy. Approximately 30% of patients exhibiting resistance to first-line (1L) therapy. Currently, resistance is only detected after treatment failure, delaying the initiation of second-line therapy and reducing its effectiveness. While PET/CT remains the gold standard for disease monitoring, its limitations in predicting relapse highlight the need for supplementary biomarkers. Fibrinogen-Like Protein 2 (FGL2) has been implicated in tumor progression and metastasis, based on published evidence of its procoagulant activity in cancer biology. This study evaluates FGL2 coagulant activity in platelets as a minimally invasive biomarker for early relapse detection and treatment response monitoring in DLBCL.

Objective: This study monitors variations in FGL2 activity in platelets (Lymfollow a blood-based assay), throughout the disease course, including diagnosis, treatment, remission, and relapse during remission or relapse, as determined by PET/CT. Lymfollow, aims to enable early detection of primary refractory disease and relapse, facilitating timely therapeutic adjustments. A multicenter clinical trial is underway to validate its role in identifying non-responders to 1L therapy and patients at risk of early relapse.

Methods: A prospective trial in Israel assessed Lymfollow activity in 74 DLBCL patients and 54 healthy controls. FGL2 activity was measured based on its ability to mimic Factor X in converting prothrombin to thrombin.

Results: Lymfollow's baseline was significantly higher in DLBCL patients vs. controls (128% vs. 100%, p≤0.025). It predicted remission with 85% specificity/65% sensitivity and relapse with 82% specificity/60% sensitivity. Increased FGL2 activity preceded relapse, even when PET/CT showed no significant changes. Among 60 patients, 7 showed stable FGL2 activity despite complete response by PET/CT; 4 later relapsed, confirmed by clinical symptoms and imaging.

Conclusions

Integrating FGL2 activity measurements with PET/CT may enhance disease monitoring, improve treatment outcomes, and guide early therapeutic interventions such as CAR-T and other 2L therapies, potentially improving survival.

O05

DUAL MRD MONITORING BY FLOW CYTOMETRY AND STR-PCR: ENHANCING MRD RISK ASSESSMENT IN PH (-) ALL POST-ALLOHCT

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Monitoring MRD in Ph(-) ALL patients post-allo-HCT identifies relapse risk, guiding immunotherapy. We evaluated concurrent use of eight-color flow cytometry (FC) and STR-PCR chimerism in 90 patients (median age 33) undergoing allo-HCT for B-(60), T-(26) ALL or MPAL(4) mainly after TBI-containing conditioning (55/90). Forty-three out of 90 transplants were matched unrelated, 36 sibling and 11 alternative ones. Monitoring of MRD with both methods was available for all patients, performed on days +30, +60, +90 and quarterly for 2 years. Median follow-up was 48 (7-193) months and 411 samples were analysed with both methods. Of 90 patients, only 16 relapsed. FC and STR-PCR showed concordant results in 78% of 411 analyzed samples. Discordant cases included: (i) FC+/STR- (16 patients, 26 samples): early MRD detection via FC led to immunosuppression reduction, preventing relapse. Three later developed concurrent FC+/STR+, and two had subsequent low level mixed chimerism (LLMC)/FC-, confirming both methods' sensitivity. (ii) FC-/STR+ (26 patients, ≥3 months post-allo-HCT): 3 relapsed, indicating STR's higher sensitivity in these cases. (iii) Early post-transplant discordance (FC-/STR+): observed during the first trimester, before full engraftment. Time to Relapse (TTR) did not differ in the two groups (FC+/ STR-,FC-/STR+) compared to the entire cohort. Similarly, no differences were found in TTR when each method was assessed separately. However, concurrent FC+/STR+ MRD indicated significantly shorter TTR (mean 10 vs 30 months, p=0.001) and OS. FC+/STR- patients had better TTR (mean 12 vs 24 months, p=0.05) but no OS benefit, likely due to successful immunotherapy.

Conclusion: The combined use of FC and STR-PCR enhances MRD monitoring, identifying high-risk patients (FC+/STR+), who show significantly worse TTR and OS. FC detects MRD earlier (FC+/STR-), allowing timely immunosuppression reduction without relapse. LLMC (FC-/STR+) warrants closer monitoring. Dual-method monitoring improves risk stratification, guiding targeted interventions for better outcomes.

O06

RITUXIMAB-DOSE-ADJUSTED EPOCH (R-da-EPOCH) IN PRIMARY MEDIASTINAL LARGE B-CELL LYMPHOMA (PMLBCL): A MULTINATIONAL REAL-LIFE STUDY

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Introduction and Purpose: Real-life studies of moderate size have shown satisfactory but less impressive results compared with the original study of R-da-EPOCH in PMLBCL. The efficacies of R-da-EPOCH, the dose- escalation-schedule compliance and its effect on the outcomes, and the use of consolidative radiotherapy (RT) have not been evaluated.

Materials and Methods: The study included 329 patients (≤60 years), enrolled from

Greek (n=176), Israeli (n=75), Turkish (n=34), Saudi Arabian (n=28), Maltese (n=11) and Cypriot (n=5) centers. Consolidative RT was given at the treating physician's.

Results: The median age of the patients was 33 years (16-60), 38% had B symptoms, 36% were stage E or IV, 68% had bulky disease, 83% elevated LDH. The median follow-up was 35 months (0.3-100.3). Only 32 patients received RT. The 5-year Freedom From Progression (FFP) was 85%. Six patients developed therapy-related (t-) AML at 10.5-28.9 months, and one Hodgkin lymphoma. The 5-year overall survival (OS) was 92%. Protocol violations were common (165/291 patients with available data), mainly consisting of insufficient dose escalation. Among 302 patients with available data, 62% reached level ≥3 and 31% ≥4. The 5-year FFP was 88% vs 84% for patients with strict protocol adherence or not (p=0.33); OS results were similar. The prognostic systems including extranodal involvement (E/IV) and highly elevated LDH (≥2x) or bulk significantly affected prognosis. E/IV-LDH model: the 5-year FFP was 90%, 84% and 72% for patients with 0, 1 or 2 adverse factors (p=0.009), while 5-year OS was 95%, 93% and 83% (p=0.08). E/IV-bulk model: the 5-year FFP was 91%, 89% and 73% (p=0.001), while 5-year OS 98%, 94% and 85% (p=0.005). Based on the results of the largest series reported for R-da-EPOCH in PMLBCL, FFP appeared better than the expected with R-CHOP. Consolidative RT was safely omitted in the majority of responders. However, the appearance of 6 cases of t-AML is worrisome. Significant dose-escalation violations were recorded in the real-life; their impact on outcomes appears to be modest.

E-POSTERS

P01

LYMPHOID BLAST CRISIS OF PEDIATRIC CHRONIC MYELOID LEUKEMIA PRESENTING AS B-ACUTE LYMPHOBLASTIC LEUKEMIA

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Even though it is not uncommon for CML to initially present in blast crisis, lymphoid blast crisis presentation as B-ALL, particularly in children, is extremely rare. In the practical clinical setting, such situations are extremely challenging. However, differential diagnosis is crucial given it affects treatment choice.

We describe a case of 11 y/o boy who manifested with severe leukocytosis (WBC-325K) and 90% blasts. Blasts were positive for CD34, TdT, and B-cell markers. Cytogenetic study demonstrated t(9;22) in 19 metaphases, and FISH was positive for BCR/ABL in 94% of blasts. B-acute lymphoblastic leukemia was diagnosed, and the patient was treated with B-ALL protocol. Morphologic remission was achieved, and Day 29 bone marrow was negative for blasts without minimal residual disease by flow cytometry. However, in the absence of blasts, FISH was positive for BCR-ABL1 in 11% of cells with major breakpoint (p210) detected. Based on the above findings, it was concluded that the actual diagnosis was previously undiagnosed chronic myeloid leukemia (CML) with initial presentation as lymphoid blast crisis. Patient was referred to allo-HSCT from matched sibling.

In the largest described case series, 10-15% of CML patients manifest with blast crisis mimicking acute leukemia. Myeloid variant is much more common, with pediatric cases being exquisitely rare. We suggest that helpful criteria in differentiating de novo B-ALL from lymphoid blast crisis, sometime retrospectively, during remission, are following:

1) Type of breakpoint (M-BCR vs. m-BCR)
2) Discrepancy between percentage of BCR-ABL positive cells and blast numbers
3) Presence of BCR-ABL in myeloid cells
4) Presence of CML-related morphologic changes in “remission” (blast-free) bone marrow.

Though not included in either ICC or 5th WHO edition, there have been recent studies of children with so-called CML-type B-ALL which are biologically unique, should be differentiated from B-ALL with BCR::ABL1-like features, and carry the same prognosis as conventional B-ALL. This topic is also included in the discussion. In addition, there have been genomic microarray studies which aimed on deciphering difference in genomic profiles between de novo B-ALL and lymphoid blast crisis of CML. This will be briefly discussed in this presentation.

P02

P53 MUTATIONS: ARE ALL THE SAME? ARIADNE'S THREAD IN APPROACHING CLASSIFICATION MAZE OF P53-MUTATED MYELODYSPLASTIC SYNDROME/ ACUTE MYELOID LEUKEMIA

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TP53 is a tumor suppressor gene which codes multi-functional protein which regulates cell cycle, promotes apoptosis, and plays crucial role in preventing tumorigenesis. TP53 mutations, besides directly affecting tumor cells, modulate tumor environment shifting the balance towards immune suppression thus altering tumor surveillance by immune system which culminates in tumor escape. Bi-allelic TP53 mutations are associated with complex karyotype and high-risk disease in myelodysplastic syndrome (MDS) and acute myeloid leukemia (AML). In 2022, TP53 mutated myeloid disorders were designated as new entity by 5th edition WHO as well as International Consensus Classification (ICC). At the same time, implementation of two separate classification led to significant diagnostic challenges and discrepancies often necessitating hematopathologists to issue two separate diagnoses for the same patient which often makes it more difficult for hematologists to make management decisions. These discrepancies are exemplified in the presented clinical case. In contrast to ICC classification, WHO 5th edition does not assign TP53-mutated AML as a separate entity and makes more emphasis on morphology and myelodysplasia-related cytogenetic and molecular criteria. Both classifications emphasize the fact that, with blast increase in the range of 10-20%, presence of bi-allelic TP53 mutation, puts them in the biologically unique entity when number of blasts does not affect prognosis. Besides presence of TP53 mutation, per se, bi-allelic mutation status is required. Criteria of bi-allelic status, which are often surrogate assumptions, will be discussed. However, in certain cases, there are multifactorial challenges precluding definitive proof of bi-allelic mutation which will be cited in this review. The fact that these two classifications confer different importance to VAF percentage, creates even more diagnostic challenge when it comes to differentiation between TP53-related MDS, other types of MDS, and even clonal-related hematopoiesis. Besides bi-allelic status, there are other very important factors which are essential in biologic characteristics of this entity which include, but not limited to, variant allele frequency (VAF), presence of co-mutations, associations with other cytogenetic abnormalities and driver mutations will be mentioned. In addition, it has been recently suggested that certain hot-spot mutation, rather than any TP53 abnormality, are particularly associated with loss of p53 protein activity which, eventually, could further refine prognostic stratification and patient management. Finally, a pitfall of TP53-clonal hematopoiesis, which can mimic presence of minimal

residual disease in treated patients, is discussed. All the above-mentioned factors are crucial for therapeutic decisions. Novel methods of treatment, some of which directly target p53 and some indirectly modulating tumor microenvironment, are briefly mentioned. Considering recent findings, decision to choose management option for any specific patient involves multi-specialty team and often requires patient informed decision.

P03

ADENOVIRUS DETECTION IN UMBILICAL CORD BLOOD UNITS, IMPROVING SAFETY OF TRANSPLANTATION

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Introduction and Purpose: Viral infections are major causes of morbidity and mortality in patients undergoing Bone Marrow Transplantation (BMT). Umbilical cord blood (UCB) is an alternative source of BMT. Adenovirus (HAdV) may be transmitted through UCB to immunosuppressed causing mainly disseminated infection, liver infection, hemorrhagic pneumonia, hemorrhagic cystitis, or even graft failure. HAdV genome was detected in 20.5% of the full term placenta according to international literature and can lead to high mortality rates in immunosuppressed patients, with a variety of known complications. The purpose of this study is to examine the presence of HAdV in cord blood units (UCBs) by Real-Time PCR.

Materials and Methods: Plasma samples (n=98) from cryopreserved cord blood units were tested. CBUs were collected during 2012-2023 and were processed using the automated system Sepax (Biosafe). Informed consent was signed by the parents for research use. DNA was isolated from plasma (extraction kit QIAamp DNA Blood Mini Kit, QIAGEN). Molecular detection of HAdV was performed by Real-Time PCR using the Altona RealStar® Adenovirus PCR Kit (Altona, Hamburg, Germany) on Rotor-Gene_Q_cycler.

Results: There were 3 positive sample (2.94%) in UCBs samples analysed for HAdV. The three positive HAdV samples contained $0.41x10^6$, 8,75x $x10^6$ and $9,84x10^6$ viral DNA copies, retrospectively. One HAdV(+) sample was also CMV IgG(+)/IgM(+) while the two others CMV IgG(+)/IgM(-) and CMV IgG(-)/IgM(-) where without reaching statistical significant correlation. All samples were negative for HTLV_I-II, HCV, HBsAg and HIV_I-II. Our data shows that the percentage of Human Adenovirus was 2,94%. The results of our study indirectly indicate the need for pre-transplatation laboratory control of this virus in the UCBs. There is necessity for further investigation to validate the results of our present study.

P04

ENDOVASCULAR TREATMENT OF A PATIENT WITH ACQUIRED HEMOPHILIA A: DIAGNOSTIC AND THERAPEUTIC CHALLENGES

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Introduction and Purpose: Acquired hemophilia A (AHA) is a rare and potentially life-threatening bleeding disorder caused by the development of autoantibodies against coagulation factor VIII (FVIII), leading to bleeding diathesis. The standard management approach typically involves the use of immunosuppressive therapies to eradicate the FVIII inhibitor, along with bypassing agents to control bleeding. However, in some cases, this conventional approach may be insufficient, requiring the consideration of interventional strategies. There are few reports in the literature regarding the use of endovascular treatments in AHA. This case highlights the successful application of endovascular therapy in an elderly patient with AHA and a life-threatening muscle hematoma that was unresponsive to conventional therapy.

Material and Methods: An 82-year-old female with a medical history of rheumatic polymyalgia (under corticosteroid treatment), dyslipidemia, and hypertension, presented to the emergency department with left thigh stiffness, extensive ecchymosis, and isolated prolonged activated partial thromboplastin time (aPTT). Diagnostic evaluation confirmed the presence of a FVIII inhibitor, with FVIII activity <1% and an inhibitor titer of 7.2 Bethesda units. Despite treatment with immunosuppressive therapy (prednisone and cyclophosphamide) and recombinant factor VIIa, the patient's condition deteriorated. Hematocrit levels dropped, and hematoma continued to expand. The patient developed signs of compartment syndrome in the left thigh and became hemodynamically unstable. Given the severity of the situation, an interventional approach was pursued. Endovascular embolization was performed via the left radial artery access, with continuous recombinant activated human FVIII administration to control bleeding.

Results: Following successful embolization, bleeding ceased immediately, and the patient's condition improved significantly. Hemoglobin levels stabilized, aPTT progressively normalized, and FVIII activity gradually increased. The patient was discharged with a tapering corticosteroid regimen and scheduled for monthly follow-ups. At a 4-month follow-up, there were no signs of the FVIII inhibitor, and the aPTT had returned to normal.

P05

PILOT TESTING OF A NATIONAL REGISTRY FOR PATIENTS WITH SEVERE THROMBOEMBOLIC DISEASE OR RARE HEREDITARY AND ACQUIRED THROMBOPHILIC DISORDERS

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Introduction and Purpose: Disease registries are vital for comprehending the epidemiology, prognosis, and current therapeutic practices of various medical conditions. In Greece, there has been a lack of organized registries for patients with severe venous thromboembolic or rare hereditary and acquired thrombophilic disorders. To address this gap, the Steering Committee for Hemostasis of the Hellenic Society of Hematology initiated the development of four specific patient registries: Pulmonary Embolism, Unusual Site Thrombosis, Rare Thrombophilias, and Antiphospholipid Syndrome.

Materials and Methods: This initiative was funded by the Hellenic Society of Hematology, and the registries were developed and hosted by the National Center for Research and Technological Development (CERTH). To ensure the protection of patient data, access to these registries is restricted to attending physicians who use personal credentials. Data are pseudonymized, and patients provide informed consent in compliance with the General Data Protection Regulation (GDPR) (EU 2016/679). Inclusion criteria are based on specific ICD10 codes: (i) Pulmonary Embolism (I26), (ii) Rare Thrombophilias (D68.59), (iii) Thrombosis in Unusual Sites (I63.6, I67.6, O87.3, O22.5, I81, I82.0, K75., I82, I82.3, I82.2, I82.210, I82.21) (iv) Confirmed Antiphospholipid Syndrome (D68.61).

Results: The registries were launched in January 2025. Each patient file is designed with user-friendly sections that document recent and past thrombosis history, risk factors, diagnostic details, laboratory data, treatment status, and any complications. These registries are accessible to hematologists and other physicians who manage these specific conditions.

Conclusion: The establishment of these national registries is expected to facilitate epidemiological studies, the identification of new biomarkers, and the development of prognostic systems. These advancements aim to enhance public health prevention strategies and inform up-to-date therapeutic management. The initial study phase is planned for five years, with the potential for extended follow-up.

P06

UPREGULATION OF CCR5/CCR2 DURING IMMUNE CELL-ASSOCIATED NEUROTOXICITY SYNDROME FOLLOWING CAR-T CELL THERAPY

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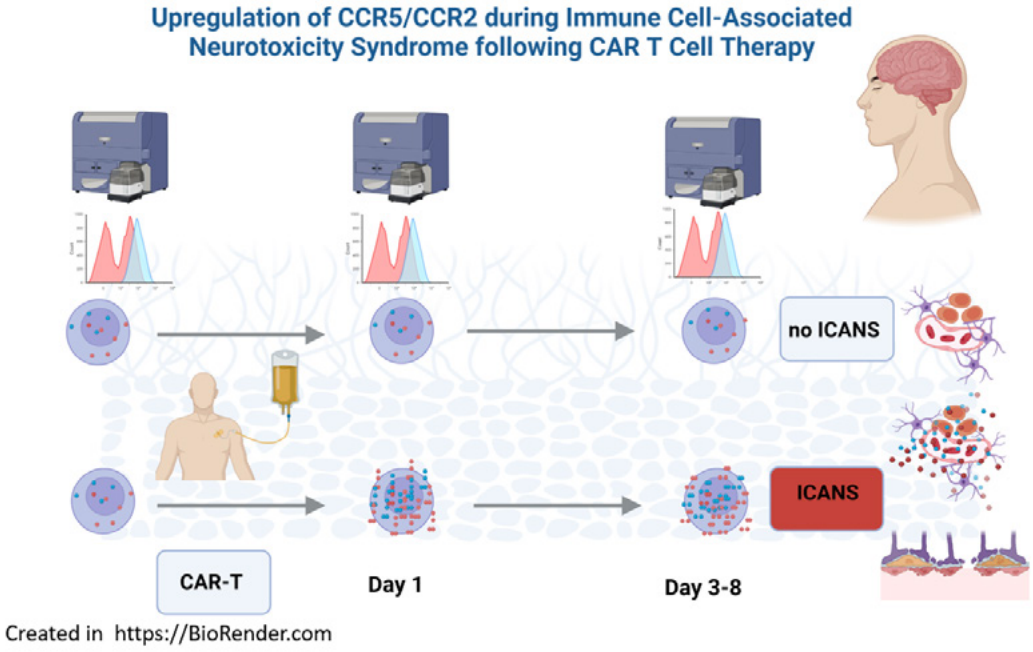
Introduction: Immune effector cell-associated neurotoxicity syndrome (ICANS) is a major complication of CAR T cell therapy. This therapy involves engineering T cells to precisely target specific proteins, yet ICANS presents a significant challenge due to its neurological toxic effects. .We aimed to investigate whether changes in chemokine profiles or macrophage markers, contribute to ICANS following CAR-T cell infusion.

Methods: Sixteen patients diagnosed with diffuse large B cell lymphoma (DLBCL) participated in this study, with 15 receiving Axicabtagene-Ciloleucel CAR-T cell therapy and 1 received tisagenlecleucel product. Blood samples were collected at several time points: before lymphodepletion, day +1, day +3 and on one of the following days: +4 to +8 after CAR-T cell administration. Samples were analyzed for $CD3^{pos}CD4^{pos}$ chemokine markers and $CD14^{pos}$ macrophage anti/pro-inflammatory markers.

Results: Of the 16 patients, 9 developed ICANS, while 7 did not. In those who developed ICANS, CCR5, expression significantly increased from an average of 55.4% to 89.5% (p = 0.001 whereas patients without ICANS experienced a slight decrease from from 61.9% to 44.8%. Similarly, CCR2 expression increased significantly in ICANS patients, from 35.1% to 65.8% (p = 0.0038), while it modestly decreased in those without ICANS, from 49.2% to 35.3%. Notably, when CCR5 levels were used as a predictor, they significantly increased in patients who developed ICANS, rising from 46.5% to 78.3% on day 1(p=0.031) and reaching 93.1% by day 3(p=0.0006) prior to clinical symptoms.
Discussion: The observed increase in $CD3^{pos}CD4^{pos}CCR5^{pos}$ and $CD3^{pos}CD4p^{pos}CCR2^{pos}$ cells in ICANS patients introduces a potential novel marker for ICANS detection and prediction, as this association has not been previously documented Additionally, the predictive role of CCR5 upregulation is newly identified.
Conclusion: This study provides preliminary insights into the mechanisms underlying ICANS following CAR T cell therapy and identifies a potential new predictive markers.


Upregulation of CCR5/CCR2 during Immune Cell-Associated Neurotoxicity Syndrome following CAR T Cell Therapy
no ICANS
ICANS
CAR-T
Day 1
Day 3-8
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P07

CYTOF ANALYSIS IN PHASE 2 PROSPECTIVE INTERVENTIONAL STUDY: NIVOLUMAB ADDITION TAILORED BY CAR-T CELL EXPANSION IN PATIENTS WITH STABLE OR PROGRESSIVE LARGE B-CELL LYMPHOMA DURING LYMPHODEPLETION

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Introduction: Patients with large B-cell lymphoma (LBCL) in stable or progressive disease (SD/PD) at lymphodepletion (LD) prior to chimeric antigen receptor T cell (CAR-T) therapy have an inferior outcome. PD-1 blockade in this setting has been assessed in our clinical trial to enhance response rate. The effect of PD-1 blockade on the immune cells in this setteing is unkown.

Methods: We previously conducted a Phase 2 prospective trial (NCT05385263) demonstrating the benefit of adding nivolumab in patients with SD or PD in LBCL treated with CAR-T cells.

Mononuclear cells were washed and stained with a panel of 39 metal-isotope tagged antibodies and analyzed by CyTOF.

Results: 26 samples were eligible for analysis based on adequate cell count from 9 patients. No significant differences in cell populations were observed between patients not receiving nivolumab and those treated with nivolumab. However, when comparing patients achieving complete response (CR) to those with partial response (PR), stable disease (SD), or progressive disease (PD)—regardless of nivolumab treatment—we found a significant increase in γδ T cells on day 7 and beyond post-CAR-T therapy in CR patients. Furthermore, CR patients exhibited fewer terminally differentiated CD8+ T cells. Notably, PD-1 expression analysis revealed a reduction in PD-1 levels in the following non-T cell populations: Natural Killer (NK) Cells, Myeloid Dendritic Cells (mDCs), Plasmacytoid Dendritic Cells (pDCs), Plasmacytoid Dendritic Cells (pDCs) following nivolumab treatment.

Conclusion: In-depth analysis of immune cells revealed that an increase in γδ T cells and a reduction in terminally differentiated CD8+ T cells may be associated with achieving CR. PD-1 blockade, through nivolumab, was linked to decreased PD-1 expression on cells other than T cells suggesting that the improved response rate in the nivolumab-eligible group may involve a broader activation of the immune system, extending beyond T cells alone.

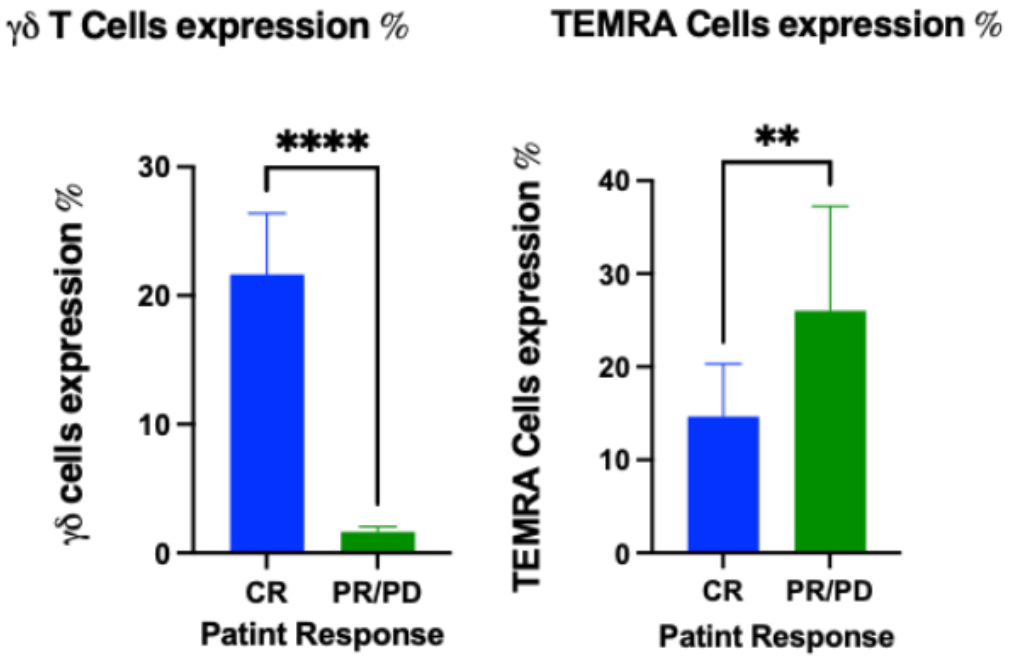

Figure 1. Expression of γδ T cells and TEMRA cells in patients with different treatment responses. Left panel: γδ T cell expression was significantly higher in complete response (CR) patients compared to partial response/progressive disease (PR/PD) patients (21.2% vs. 2.3%, ****p<0.0001). Right panel: TEMRA cell expression showed the opposite pattern, with significantly higher levels in PR/PD patients compared to CR patients (26.2% vs. 14.5%, **p<0.01). Error bars represent standard error of the mean. Statistical significance was determined using Student's t-test; **p<0.01, ****p<0.0001.

P08

CD34+ DERIVED MICROPARTICLES INCREASE THE ANTIOXIDANT MECHANISM OF UMBIICAL CORD BLOOD

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Introduction: Microparticles (MPs) are small membrane vesicles released from different cell types upon activation or stimulation. Oxidative stress is a result of prooxidant-antioxidant imbalance, in favor of the first. The antioxidant enzymes glutathione Reductase (GR) and glutathione-S-Transferase (GST) protect the cells from oxidative stress. Malondialdehyde (MDA) is a toxic product of lipid peroxidation and represent a potential biomarker of oxidative stress.

Purpose: The present study aims to investigate a possible correlation between oxidative stress and hematopoietic stem cell derived microparticles in acute promyelocytic leukemia cell line HL-60 and mononuclear cells (MNCs) from umbilical cord blood (UCB) in presence and absence of MPs derived from UCB stem cells (CD34+MPs).

Material and methods: MNCs were isolated from UCB using density gradient centrifugation and $CD34^+$MPs were isolated from the plasma of UCB using immunomagnetic separation technique. Cells were co-incubated with CD34+MPs for 24 and 48 hours. The glutathione reductase, glutathione-S-transferase activity activity and the concentration of malondialdehyde (MDA) produced by lipid peroxidation are measured using spectrophotometry.

Results: The measurements that were conducted, displayed a statistically significant increase in the antioxidant activity of the samples in the presence of CD34+microparticles in both HL60 and MNC after 24 and 48 hours of incubation. The GR activity was increased by $CD34^+$MPs in HL-60 in both time points 24h and 48h (38,3% and 32,2% respectively) and in MNCs (38,5% and 22,1% respectively) compared to control. Similarly the GST activity was increased in both in HL-60 (49,8% and 32,9% respectively) and in MNCs (37,7% and 31,1% respectively). Lastly, MDA concentration and hence lipid peroxidation was higher in cells co-incubated with $CD34^+$MPs in HL-60 (49,2% and 66% respectively) and in MNCs (52,1% and 59,8% respectively).
In conclusion $CD34^+$MPs activate the antioxidant mechanism of UCB derived mononuclear cells and HL60.

P09

AGE-SPECIFIC DIFFERENCES IN CEREBRAL SINUS VENOUS THROMBOSIS: A RETROSPECTIVE COHORT STUDY FROM A TERTIARY CARE CENTER

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Background: Cerebral sinus venous thrombosis (CSVT) varies between pediatric and adult patients in presentation and outcomes. This study compared clinical characteristics, risk factors, treatment patterns, and outcomes in these age groups.

Methods: A retrospective cohort study of patients diagnosed with CSVT (2012–2023) was conducted. Demographic, clinical, and outcome data were analyzed, including recanalization, recurrent thrombosis, and bleeding events.

Results: From 2012 to 2023, 242 patients were identified (157 adults, 85 children). Children (median age: 2.83 years) predominantly presented with provoked CSVT (85.9% vs. 63.1% in adults, $p<0.001$), often due to head and neck infections (34.1% vs. 3.8%, $p<0.0001$). Adults (median age: 41.5 years) had higher rates of hormonal (20.4%) and idiopathic cases (36.9% vs. 14.1%, $p<0.001$). Headache was the most common symptom in adults (63.1% vs. 30.6% in children, $p<0.0001$), while children frequently presented with seizures (31.8% vs. 8.3%, $p<0.0001$). Treatment duration was longer in adults, with two-thirds requiring extended anticoagulation (66.2% vs. 11.76% in children). Pediatric treatment favored low molecular weight heparin (75.3%), while adults more commonly received warfarin (45.2%) or direct oral anticoagulants (DOACs) (10.2%). Recanalization rates were higher in children (87% vs. 67.5%, $p=0.0009$), and recurrent thrombosis was more frequent in adults (5.7% vs. 1.17%). Rates of major bleeding were comparable in both groups (4.7% in children vs. 2.55% in adults, $p=0.457$).

Conclusion: CSVT exhibits age-specific differences in risk factors, clinical presentation, treatment patterns, and outcomes. Pediatric cases, typically provoked, require shorter treatment durations and show higher recanalization rates.

P10

INTAKE OF BENZODIAZEPINES IN TREATMENT NAÏVE CHRONIC LYMPHOCYTIC LEUKEMIA PATIENTS IS ASSOCIATE WITH A SHORTER TIME TO FIRST TREATMENT

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In this study, we explored the association between benzodiazepine (BZP) usage and time to first treatment (TTFT) in patients with chronic lymphocytic leukemia (CLL) under a watch-and-wait (W&W) approach. Utilizing data from Maccabi Healthcare Services in Israel, we analyzed 3,474 CLL patients, focusing on BZP and non-BZP users. Through inverse probability of treatment weighting and time-dependent Cox proportional hazards models, we observed that BZP usage was significantly associated with a shorter TTFT (HR 1.78, p=0.029). Particularly notable were findings related to Brotizolam and Clobazam. Our study suggested that BZP usage, particularly in patients with mental disorders, may accelerate the clinical progression of CLL. Despite retrospective design limitations, these findings emphasize the need for careful BZP administration in CLL patients and encourage further research..

P11

BREAST CANCER IN PATIENTS WITH CHRONIC LYMPHOCYTIC LEUKEMIA: IMPACT ON DISEASE COURSE AND SURVIVAL OUTCOMES - A RETROSPECTIVE STUDY

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Introduction and Purpose: Chronic lymphocytic leukemia (CLL) is associated with an increased risk of secondary malignancies, with breast cancer (BC) being the most common among women. The interplay between CLL and BC, particularly its impact on disease progression and patient outcomes, remains poorly understood. This study seeks to assess the clinical outcomes of CLL patients diagnosed with BC, compare these outcomes to those of CLL patients without malignancies, and investigate the effect of the timing of BC diagnosis on survival and time to first treatment (TTFT) in CLL.

Material and Methods: A retrospective analysis was conducted on 272 female CLL patients treated at the Tel Aviv Sourasky Medical Center. Clinical and demographic data, including diagnoses, treatments, and survival outcomes related to both CLL and BC, were extracted from medical records. Overall survival (OS) and time to first treatment (TTFT) were analyzed using the Kaplan-Meier estimator, Cox regression, and the Log-Rank test, with statistical significance set at $P<0.05$.

Results: Among 272 female CLL patients, 44 (16.2%) were diagnosed with BC. Of these, 25 (56.8%) had BC diagnosed before CLL, while 19 (43.2%) were diagnosed with BC after CLL. The median ages at diagnosis for CLL and BC were 71.2 and 68.6 years (Y), respectively. Patients with BC before CLL were older at CLL diagnosis than those with BC after CLL (median 73.6 vs. 68.1 Y, P=0.016). The interval between BC and CLL diagnoses was longer for patients with BC preceding CLL (median 11.9 vs. 4.2 Y, P=0.076).

BC pathology was available for 24 patients (54.5%), with infiltrating ductal carcinoma being the predominant subtype (70.8%). Most patients (95.5%) received BC treatment, including surgery (88.1%), radiotherapy (64.3%), chemotherapy (26.2%), and maintenance therapy (76.2%). CLL treatment was initiated in 20 of the 44 BC patients (45.5%). Among the 19 patients diagnosed with BC after CLL, 15.8% had received prior CLL treatment, while 21.1% started CLL treatment after BC diagnosis.

Patients with BC diagnosed before CLL had significantly worse OS than those with BC diagnosed after CLL (Figure 1; median OS: 8.3 vs. 19.1 Y, P=0.003; HR=5.8, 95% CI 1.6–21.0). Additionally, they exhibited a trend toward shorter TTFT (median TTFT: 6.1

Y vs. not reached; P=0.058; HR=2.4, 95% CI 0.9–6.3).

Compared to female CLL patients without malignancies (n=228), those with BC showed a trend toward inferior OS (median 18.3 vs. 26.0 Y, P=0.071), with no significant differences in TTFT.

Overall, 18 patients (40.9%) with both CLL and BC died, with causes of death including infections (7/18, 38.9%), BC progression (11.1%), CLL progression (5.55%), other secondary malignancy (5.55%), and others (38.9%).

Conclusions: In CLL patients, BC especially when diagnosed before CLL is associated with worse OS and a trend toward shorter TTFT. The shorter interval between BC and CLL diagnoses in patients with BC after CLL suggests a role of immune dysregulation in cancer development. These findings highlight the need for tailored treatment and enhanced surveillance in this high-risk group.

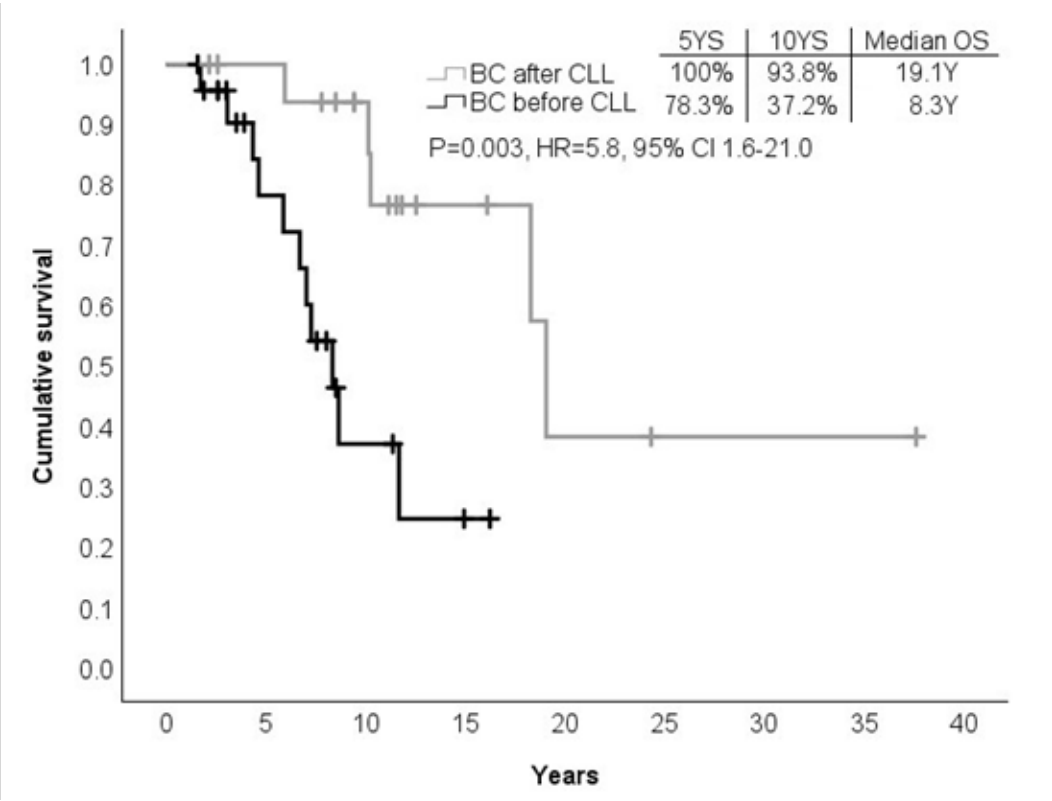

Figure 1. Overall survival, by timing of breast cancer diagnosis, before or after CLL

P12

SURVIVAL TRENDS IN HODGKIN LYMPHOMA (HL) FROM 1980 TO 2020: SINGLE-CENTER EXPERIENCE

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Background: The aim of the current study is to describe the outcome of patients with HL over consecutive time periods (1980-2020), describe the patients' characteristics and investigate the effect of treatment evolution on disease control and survival.

Methods: Retrospective analysis of 1795 patients with HL treated between 1980-2020, divided into three periods. During period A (1980-1990) early stage (ES) patients received combined modality and advanced stages (AS) chemotherapy ±radiotherapy (RT), with MOPP-like and anthracycline-based regimens. During period B (1990-2008) anthracyclines were universally applied, while autologous transplant was introduced as salvage. During period C (2009-2020) interim PET-based strategies were adopted for AS and after 2017 for ES, while novel agents were incorporated in salvage. Statistical endpoints were Freedom from Progression (FFP), Overall Survival (OS), Disease Specific Survival excluding deaths from other causes (DSS1) and DSS excluding unrelated deaths but including deaths from secondary neoplasms (DSS2).

Results: 229, 876 and 681 patients were treated during periods A,B and C respectively. Patients in period A were older with more frequent AS, B-symptoms and hypoalbuminemia. Period C patients were older than period B and had more frequently AS disease, anemia, hypoalbuminemia, elevated LDH, ESR ≥50mm/h and marginally lymphocytopenia. 10-year FFP improved over time and was estimated at 65.9%, 77.3% and 76.8% for periods A,B and C respectively. 10-year OS also improved significantly from 71.3% to 84.7% and 88.8%. There was an improvement in 10-year DSS2 from

87.3% to 91.1% between periods B and C respectively, and a marginal improvement in DSS1 from 90.1% to 93.1%. In multivariate analysis including only patients of periods B and C, AS, age ≥45 years and anemia were universal adverse prognostic factors for FFP, OS and DSS, while male gender adversely affected DSS2 and OS as well. Treatment period was not associated with FFP, but period B was associated with inferior OS, DSS1, and DSS2.

P13

THE PROGNOSTIC SIGNIFICANCE OF ADJUSTED SERUM B2-MICROGLOBULIN LEVELS (SB2M) ACCORDING TO RENAL FUNCTION IN DIFFUSE LARGE B-CELL LYMPHOMA (DLBCL)

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Background: The established Sβ2m cutoff of 3 mg/L represents the median value of the corresponding patient population, however, Sβ2m levels are influenced by the glomerular filtration rate (GFR). We aimed to investigate the prognostic significance of sβ2m levels adjusted to renal function (RFA-sβ2m) compared to measured sβ2m levels in a series of DLBCL patients in the rituximab era.

Methods: We included 916 patients with DLBCL treated with R-CHOP and available pretreatment sβ2m (mg/L), serum creatine levels, height and body weight data. The RFA-sβ2m was calculated as the ratio of observed sβ2m and expected sβ2m according to each patient's GFR. The analysis was performed using the cutoff of 3 mg/L for sβ2m and RFA-sβ2m quartiles (Q1-Q4) respectively. Freedom From Progression (FFP) was defined as time between treatment initiation and treatment failure (toxic death, primary refractoriness, PR with treatment switch or relapse); deaths of unrelated causes were censored.

Results: The median sβ2m and RFA-sβ2m levels were 2.9 mg/L and 1.68 respectively and were strongly correlated (Spearman's rho 0.821, p<0.001). FFP was worse in patients with sβ2m ≥3 mg/L with 2-year FFP of 64.0% vs 88.3%. Similar results were observed for RFA-sβ2m, with 2-year FFP rates of 90.7%, 87.6%, 72.4% and 56.6% for Q1-Q4 respectively. Among 465 patients with sβ2m <3 mg/L, 383 had RFA-sβ2m <1.68 (median value) and 82 had RFA-sβ2m >1.68 with 2-year FFP of 91.1% versus 75.4% respectively. Among 417 patients with sβ2m ≥3 mg/L, 63 had RFA-sβ2m <1.68 and 354 had RFA-sβ2m >1.68 with 2-year FFP of 77.1% versus 61.8% respectively. When sβ2m were analyzed together with IPI (0-1 vs 2 vs 3 vs 4-5) in multivariate analysis of FFP, both factors had independent prognostic significance. Adjusted for IPI, the hazard ratio (HR) for sβ2m ≥3 versus <3 mg/L was 1.67. When RFA-sβ2m was analyzed along with IPI in multivariate analysis of FFP, both factors had independent prognostic significance. Adjusted for IPI, the HR for Q4, Q3, and Q2 versus Q1 were 2.14, 1.46 and 0.99. RFA-sβ2m displaced sβ2m from the latter model.

P14

RITUXIMAB-DOSE-ADJUSTED EPOCH (R-da-EPOCH) VERSUS RITUXIMAB-CHOP (R-CHOP) IN PRIMARY MEDIASTINAL LARGE B-CELL LYMPHOMA (PMLBCL)

Vassilakopoulos T.[1], Mellios Z.[2], Papageorgiou S.G.[4], Piperidou A.[1], Verigou E.[3], Chatzidimitriou C.[1], Kalpadakis C.[5], Katodritou E.[6], Giatra H.[2], Xanthopoulos V.[7], Gainaru G.[7], Vrakidou E.[7], Leonidopoulou T.[8], Kotsopoulou M.[9], Palassopoulou M.[10], Karakatsanis S.[11], Tsirogianni M.[12], Hatzimichael E.[13], Terpos E.[14], Zikos P.[15], Poziopoulos C., Vervessou E., Arapaki M.[1], Kopsaftopoulou A.[1], Katsaouni P.[3], Asimakopoulos J.[1], Kourti G.[2,11], Kousiafes D.[8], Siakantaris M.P.[1], Karianakis G.[1], Symeonidis A.[1], Grentzelias D.[7], Pappa V.[4], Tsirigotis P.[4], Papadaki E.[5], Plata E.[1], Bakiri M.[1], Pangalis G.A.[1], Angelopoulou M.K.[1], Bouzani M.[2]
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Background: Retrospective comparisons have shown a modest, non-significant benefit in disease control with R-da-EPOCH vs R-CHOP in PMLBCL. The aim of our study was to evaluate the efficacy of R-da-EPOCH vs R-CHOP with an -as much as possible- unbiased selection of control group patients.

Methods: R-da-EPOCH was adopted in all consecutive patients with PMLBCL ≤65 years (n=156) in 18 participating Centers. The control group of R-CHOP-treated patients was devised from the same Centers' database. Due to lack of some appropriate controls in a few Centers they were substituted by consecutive patients treated in few of the other centers, which had comparable potential. R-CHOP-14 was given in 22/156 patients of the control group (14%).

Results: The groups of R-da-EPOCH and R-CHOP-treated patients (n=156) were absolutely comparable except for more frequent multiple extranodal involvement in R-CHOP (8.4% vs 16.0%, p=0.042). The 5-year freedom from progression (FFP), event-free survival (EFS) and overall survival (OS) rates for R-da-EPOCH vs R-CHOP were 87.5% vs 75.5% (p=0.011), 84.4% vs 75.5% (p=0.052, 4 t-AML cases after R-da-EP-

OCH) and 94.1% vs 86.9% (p=0.039). RT was administered to 10% vs 70% after R-da-EPOCH or R-CHOP. In multivariate analysis, after adjustment for age, gender, multiple extranodal sites and recently published prognostic models (extranodal and LDH >2x or bulk) the difference between R-da-EPOCH and R-CHOP remained significant regarding FFP, OS and EFS, when the extranodal-LDH model was assessed, but only for FFP with the extranodal-bulk model (0.10<p<0.20 for EFS and OS). 77 of 133 patients with currently available data had absolute adherence to R-da-EPOCH protocol. These patients had a 5-year FFP of 90.9%. Our non-randomized study is by far the largest one comparing R-da-EPOCH vs R-CHOP and carried the least possible systematic error in the retrospective setting showing that R-da-EPOCH minimized the need of RT with better disease control outcomes.

P15

PSYCHOLOGICAL BURDEN AND DEPRESSIVE SYMPTOMS IN CAREGIVERS OF HEMATO-ONCOLOGICAL PATIENTS: THE ROLE OF MEDICAL VISITS

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Background: Informal caregivers of cancer patients are known to experience extensive burdens, while this issue remains unresolved in the setting of hematological malignancies. Yet, these diseases are characterized by a prolonged course, numerous relapses and implementation of multi-line therapy, administered in outpatient facilities. The current study aimed to assess the factors contributing to burden and depressive symptoms in informal caregivers of patients with hematological malignancies, while concentrating on the role of medical visits.

Methods: The study population comprised patients and their caregivers, recruited at the Rambam Hematology Ambulatory Unit. Participants completed validated questionnaires, including the Center for Epidemiologic Studies Depression Scale and Zarit Caregiver Burden Interview.

Results: The cohort (n=185) included 115 patients [average age 62.8±14.5 years; 54 males] and 70 caregivers. Among caregivers, 80% reported high psychological burden and 50% reported significant depressive symptoms. The burden was higher if caregivers were females, and if patients were less educated, less healthy and more depressed. The caregiver burden and depressive symptoms were significantly linked and the medical visit frequency predicted the level of both. The caregiver burden fully mediated the link between the independent variables of self-rated health and medical visits, and the dependent variable of caregiver depressive symptoms.

Conclusion: Informal caregivers of ambulatory hemato-oncological patients experience high levels of psychological burden and depressive symptoms. This is partly attributed to the medical visit frequency. Hence, a decrease in the number and length of such visits via the implementation of advanced technology could essentially reduce burden and depressive symptoms of caregivers, without compromising patient outcomes.

P16

HIGH-THROUGHPUT LABORATORY AUTOMATION FOR TECHNOLOGY TRANSFER IN CLONAL HEMATOPOIESIS DETECTION: A USE CASE OF VITALE PROJECT (VERSATILE INTEGRATED TECHNOLOGY ADVANCING LIFE-SCIENCE EXPLORATION)

Kyriacou D.
Efevre Tech LTD

Background: Clonal hematopoiesis (CH) is a condition characterized by the accumulation of somatic mutations in hematopoietic stem cells, particularly in genes such as DNMT3A, TET2, and ASXL1. These mutations have been strongly linked to an increased risk of acute myeloid leukemia (AML) and cardiovascular diseases (CVDs), including atherosclerosis and stroke. Despite its growing clinical significance, current CH detection methods remain labor-intensive, time-consuming, and prone to human error. The need for a fully automated and scalable workflow is critical to improving early detection, risk stratification, and overall clinical outcomes. This study presents an advanced high-throughput laboratory automation platform for technology transfer, designed to enhance reproducibility and efficiency in CH detection through automated blood EDTA processing, DNA extraction, and next-generation sequencing (NGS) analysis.

Methods: The developed automation platform integrates robotic liquid handling, automated nucleic acid extraction, and NGS library preparation into a seamless workflow. High-throughput blood sample processing ensures standardized genomic DNA isolation, followed by fluorometric DNA quantification and normalization for accurate sequencing input control. The system automates NGS library preparation, encompassing enzymatic fragmentation, PCR amplification, and purification steps. Additionally, barcode scanning and real-time data tracking enable seamless integration with laboratory information management systems (LIMS), ensuring robust sample traceability and data integrity. The modular and scalable architecture of the platform allows for customization and adaptation to multiple research and clinical applications.

Results: The implementation of high-throughput laboratory automation has demonstrated a significant reduction in hands-on time compared to conventional manual workflows. By eliminating operator-induced variability, the system enhances reproducibility and accuracy in CH detection. Seamless integration with existing NGS pipelines has facilitated large-scale genomic analysis, increasing efficiency and throughput. The automation framework also provides long-term adaptability, supporting precision medicine applications in hematologic malignancies and cardiovascular disease

research. These findings underscore the potential of high-throughput laboratory automation as a transformative tool for improving genomic workflows and advancing technology transfer in biomedical research and diagnostics.

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P17

DIFFERENCES BETWEEN HYPERDIPLOID AND HYPODIPLOID COMPLEX KARYOTYPE IN MULTIPLE MYELOMA

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Introduction: Cytogenetic abnormalities in patients with multiple myeloma play a crucial role in disease staging and prognosis. While hyperploidy is considered a standard risk factor and hypodiploidy an adverse one, the significance of the number and type of chromosomal abnormalities is unclear. Although complex karyotype (both quantitative and structural abnormalities are required) is not considered as a high risk disease factor in myeloma, it has been reported to affect prognosis. Additionally, the role of coexistent FISH abnormalities in complex karyotype remains vague.

Methods: 37 patients, median age 64, with a complex karyotype underwent classic bone marrow chromosomal and FISH analysis.

Results: 24 patients had hyperdiploidy (48-65 chromosomes), whereas 13 hypodiploidy (<44 chromosomes). Concurrent structural abnormalities were more frequent in hypodiploidy: median of 6 (2-9) per patient, and in hyperdiploidy median of 3 (1-10). Interestingly, 9 patients with hyperdiploidy had only 1 structural abnormality. High-risk cytogenetic abnormalities detected by FISH were scarcer in patients with hyperdiploidy: 7/21 (33%, 1p/1q n= 5, del17p n=2) versus 9/12 (75%, 1p/1q: 9, IGH/FGFR3: 2, IGH/MAF: 2, del17p: 4) in hypodiploidy, p=0.02.
As expected, disease outcome was worse in patients with hypodiploidy vs hyperdiploidy. Median PFS was 15 vs 31 months (p=0.003) and median OS was 19 vs 92 months respectively (p=0.004). Patients with only 1 structural abnormality did not have a more favorable prognosis. Concerning patients <70 years old, ASCT improved outcome; no other statistically significant factors were found.

Conclusions: Among myeloma patients with complex karyotype, hypodiploidy compared to hyperdiploidy is a more complex disease genetically, with more concurrent structural abnormalities and coexistence of high-risk FISH abnormalities in the majority of patients, thus explaining its inferior prognosis. Structural abnormalities were fewer in hyperploidy, as were high-risk FISH abnormalities. Finally, ASCT enhances the disease outcome in patients with complex karyotype.

P18

IS TOTAL DOSE OF CYTARABINE IMPORTANT IN MOLECULARLY LOW-RISK ACUTE MYELOBLASTIC LEUKEMIA (AML) WITH NPM1 OR CEBPA MUTATIONS?

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Intermediate doses of cytarabine (AraC) are currently used in AML, since no survival benefit has been proven for higher doses, while greater toxicity has been observed. Cytogenetically low-risk AML (CBF AML, t8;21 and inv16) might stand as an exception, where HDAC seems to correlate with increased disease-free survival (DFS). There is limited data regarding patients with molecular low-risk characteristics, such as AML with NPM1/CEBPA mutations.

We retrospectively studied 57 patients with NPM1+ or CEBPA AML to assess the potential impact of the total dose of cytarabine on the outcome. Twenty-seven men and 30 women, with a median age of 48 (23-72)years, had CEBPA-bzip mutations (9/57) or NPM1 mutations (48/57, with concomitant FLT3-TKD: 5, CEBPA-non-bzip: 1). The karyotype was normal in 50 patients, and 7 had an abnormal intermediate-risk karyotype. All patients received 1-2 induction courses of “3+7” and 3-4 consolidation courses with intermediate or high doses of cytarabine, while 14 patients also received gemtuzumab. All patients achieved complete remission (100%, 51 after the first course, 6 after the second). Fourteen patients received a total cytarabine dose of <25g/m^2 (group 1), 20 patients received 25-36g/m^2 (group 2), and 23 received 37-49g/m^2 (group 3). Three patients underwent allo-HSCT due to positive MRD. The 5-year OS for group 1 was 78%, for group 2 was 54.5% and for group 3 was 82.6%, respectively (p=0.32). The 5-year DFS was 63.5%, 51.2%, and 64.1%, respectively (p=0.88). Overall, 20 patients (35%) experienced relapse, 18/48 of the NPM1+ patients (37.5%, with 8 of them without NPM1 mutation at relapse), and 2/9 of the CEBPA-bzip patients (22%). In the multivariate analysis, only age was found to be statistically significant for OS (p=0.002) and DFS (p=0.003).

In conclusion, AraC dose did not affect the outcome in patients with NPM1 or CEPBA mutations. Larger studies could provide further insight.

P19

PROGNOSTIC EFFECT OF TRANSLOCATION t(10;14) IN T-ACUTE LYMPHOBLASTIC LEUKEMIA

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Aim: The rearrangement t(10;14)(q24;q11) is found in 5-10% of adults with T-ALL and leads to the translocation of the delta gene (δ gene) of the T-cell receptor (TCR) on chromosomal region 14q11 with the oncogene TLX1 (10q24). The rearrangement of TLX1 has been reported in the literature to confer a rather favorable prognosis, although it is not yet considered an independent prognostic entity.

Methods: We retrospectively studied adult patients with T-ALL with the t(10;14)(q24;q11) rearrangement. Flow cytometry on bone marrow samples was used for all patients at diagnosis and for MRD monitoring, while primer-specific PCR was used in two patients. Conventional karyotyping and FISH were performed on all patients at diagnosis and confirmed remission.

Results: Six adult patients (5 males/1 female), median age 55 (17-66)years, were diagnosed with primary T-ALL with t(10;14)(q24;q11) rearrangement among 73 T-ALL cases (8%). Two patients also exhibited trisomy 7, trisomy 8, and del(9)(p21). The median white blood cell count was $24x10^9$/L. A mediastinal mass was present in 4/6, organomegaly 2/6, and none had CNS involvement. One young patient received the pediatric BFM protocol, while the other five were treated with less intensive protocols due to age/comorbidities. All patients received L-asparaginase and two years of maintenance therapy. Morphological and cytogenetic remission was achieved early by all patients, with MRD negativity after the first induction (5/6 patients), or after the second (1/6). Four patients remain in first complete remission with negative MRD, median DFS 5 (3.5–10)years. Two patients died in CR (pancreas and colon cancer at 4 and 5 years).

Conclusions: Molecular and cytogenetic classification of T-ALL is essential for better stratification of risk groups. The t(10;14)(q24;q11) rearrangement appears to confer a more favorable prognosis, with sustained remissions and without the need for allogeneic transplantation. Larger-scale prospective studies are necessary.

P20

UNEXPECTED CLINICAL TUMOUR LYSIS SYNDROME (TLS) WITH COMBINATION THERAPY OF AZACITIDINE (AZA) PLUS VENETOCLAX (VEN) IN OLDER PATIENT WITH ACUTE MYELOID LEUKAEMIA (AML)-CHALLENGES IN MANAGEMENT: A CASE REPORT

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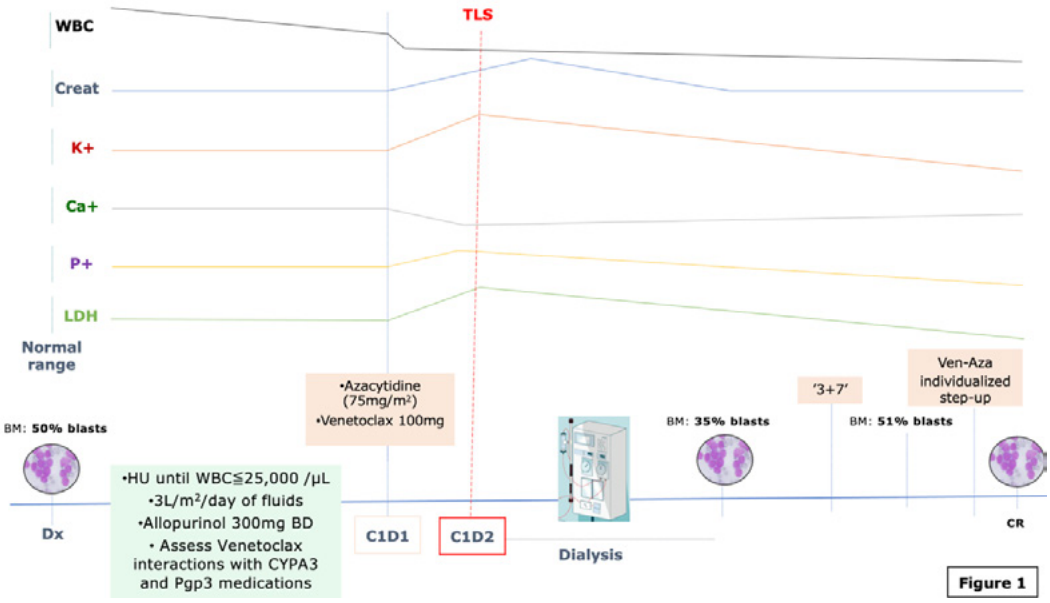
Introduction and Purpose: For elderly, unfit patients with AML who lack a molecular therapeutic target, Ven-Aza is regarded as an effective, safe, and frequently the sole treatment option. Thus, determining the appropriate treatment strategy when the former is ineffective or associated with unacceptable side-effects poses challenges to clinicians.

Material and Methods: Herein, we present a case of an elderly patient with newly diagnosed AML, with complex karyotype and leukaemia cutis. According to protocol, she initially received Hydroxurea to lower the WBC <25,000/μL prior to initiating Ven-Aza. Despite taking all the recommended precautions (hydration, allopurinol, lab monitoring, assessment of potential drug interactions), the patient developed clinical TLS following the first Ven titration dose 100mg. Intensive support that included eleven hemodialysis sessions were required as shown in Figure 1.

Results: Following TLS resolution, the patient received '7+3' without response. Thus, it was decided to rechallenge her to Ven-Aza with an individualized step-up dosing (100mg d1-2, 200mg d3, 400mg qd thereafter) based on TLS markers. Hereby, she tolerated treatment well this time and achieved complete remission. After 4 cycles of Ven-Aza, the disease was consolidated with BU (6.4mg/kg) + Flu (150mg/m^2) + TT (10mg/kg) + ATG (3mg/kg) reduced toxicity conditioning and graft from HLA-identical sibling (Fig1). Our case reinforces the effect of Ven-Aza in AML with unfavourable prognosis, which is commonly chemorefractory, either as bridge therapy to transplant or as definite treatment option. According to current knowledge TLS is a rare complication. On the other hand in some case, despite all necessary precautions, TLS remains a realistic risk that may complicate significantly disease course and may affect the outcome. In addition, as implicated in our case, rechallenge with Ven-Aza with more gradual step-up dosing is feasible once major complications are adequately controlled.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Ven-Aza individualized step-up
BM: 50% blasts
BM: 35% blasts
BM: 51% blasts
•HU until WBC≦25,000 /μL
•3L/m²/day of fluids
•Allopurinol 300mg BD
• Assess Venetoclax interactions with CYPA3 and Pgp3 medications
Dx
C1D1
C1D2
Dialysis
CR

Figure 1

P21

ADVANCING MYELOID NEOPLASM RISK ASSESSMENT THROUGH NGS-BASED GERMLINE MUTATION DETECTION IN ALLOGENEIC HEMATOPOIETIC CELL TRANSPLANTATION

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Background: Understanding the genetic landscape of myeloid malignancies is crucial for risk assessment and personalized clinical management. This study aims to identify genetic factors influencing post-transplant complications and disease relapse in donor-recipient pairs undergoing allogeneic hematopoietic stem cell transplantation (allo-HSCT).

Methods: Nineteen sibling donor-recipient pairs were analyzed using targeted next-generation sequencing (NGS) to detect germline variants and clonal hematopoiesis (CH)-associated mutations. Variants were classified as pathogenic, likely pathogenic, or variants of uncertain significance (VUS) based on ACMG guidelines. CH was defined by somatic mutations with a variant allele frequency (VAF) ≥2%.

Results: Donor Variants: i) Copy Number Variations (CNVs): Three donors (16%) carried CNVs absent in recipients ii) VUS: Seven donors (37%) harbored germline VUS in key myeloid genes, including GATA2 (n=2), PRPF8 (n=2), KMT2A, RAD21, CALR, NF1, CSF3R, and BCOR. iii) CH: Three donors (16%) had CH-associated mutations in DNMT3A, BCOR, and PTPN11.

Recipient Variants: NGS analysis at diagnosis identified potential germline variants (VAF 40-60%) in 10 patients, requiring validation in remission samples. In one patient, germline ASXL1 and CSF3R variants were confirmed, which were absent in the donor. Clinical Observations**:** Three patients relapsed, no one with donor-derived leukemia. In one case, the donor harbored two germline variants, two CNVs, and BCOR-associated CH; in second, the donor carried CH in PTPN11. No strong correlation was observed between donor VUS or CH and recipient outcomes, possibly due to limited follow-up data.

Conclusion: These findings suggest that comprehensive genetic screening in allo-HSCT may contribute to refining risk stratification and guiding long-term outcome monitoring. Further validation, including confirmation of patients' germline variants and expansion to larger cohorts, is warranted.

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Ζητείται να αναφέρονται οποιεσδήποτε πιθανολογούμενες ανεπιθύμητες ενέργειες μέσω του εθνικού συστήματος αναφοράς.
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BRK.ADV(001/04.2024

Για επιλέξιμους ενήλικες ασθενείς με CML που αποτυγχάνουν σε ένα 2G TKI, το **ICLUSIG® προσφέρει δεδομένα, εμπειρία και τη δυνατότητα να βελτιώσουν το μέλλον** τους[1-3]

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(ponatinib) tablets

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Για την ΠΧΠ σκανάρετε τον κωδικό QR:

Περίληψη του προφίλ ασφάλειας[1].
Οι πιο συχνές σοβαρές ανεπιθύμητες ενέργειες > 2% ήταν πνευμονία, παγκρεατίτιδα, κοιλιακό άλγος, κολπική μαρμαρυγή, πυρεξία, έμφραγμα του μυοκαρδίου, περιφερική αποφρακτική αρτηριοπάθεια, αναιμία, στηθάγχη, μείωση αριθμού αιμοπεταλίων, εμπύρετη ουδετεροπενία, υπέρταση, νόσος των στεφανιαίων αρτηριών, συμφορητική καρδιακή ανεπάρκεια, αγγειοεγκεφαλικό επεισόδιο, σήψη, κυτταρίτιδα, οξεία νεφρική βλάβη, λοίμωξη των ουροφόρων οδών και αύξηση λιπάσης.[1]

Τρόπος Διάθεσης: Περιορισμένη ιατρική συνταγή. Η έναρξη της θεραπείας γίνεται σε νοσοκομείο και μπορεί να συνεχίζεται εκτός νοσοκομείου υπό την παρακολούθηση ιατρού.

Περαιτέρω πληροφορίες διατίθενται από τη Γένεσις Φάρμα Α.Ε.

Ενδεικτική Τιμή (Ν.Τ.): ICLUSIG F.C.TAB 15MG/TAB BTx30 BOTTLE: 2.391,19€, ICLUSIG F.C.TAB 30MG/TAB BTx30 BOTTLE: 4.097,88€, ICLUSIG F.C.TAB 45MG/TAB BTx30 BOTTLE: 4.014,02€

2G: δεύτερη γενιάς. **CML:** χρόνια μυελογενής λευχαιμία.**TKI:** αναστολέας τυροσίνης κινάσης.

Βιβλιογραφία:
1. ICLUSIG® Περίληψη Χαρακτηριστικών Προϊόντος.
2. Cortes JE, et al. Blood. 2018;132(4):393–404.
3. Cortes J, et al. Blood. 2021;138(21):2042–2050.

Βοηθήστε να γίνουν τα φάρμακα πιο ασφαλή
και Αναφέρετε ΟΛΕΣ τις ανεπιθύμητες ενέργειες
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Πριν τη συνταγογράφηση του Fabhalta συμβουλευθείτε την Περίληψη των Χαρακτηριστικών του Προϊόντος Fabhalta (iptacopan) (Ημερομηνία Αναθεώρησης Κειμένου: 31 Μαρτίου 2025), η οποία είναι διαθέσιμη μεσω του **κώδικα QR που μπορείτε να σκανάρετε εδώ**:

Περαιτέρω πληροφορίες διατίθενται από τον Τοπικό Αντιπρόσωπο,
Novartis (Hellas) A.E.B.E., 12ο χλμ. Εθνικής Οδού Αθηνών - Λαμίας, 144 51, Μεταμόρφωση, Τηλ.: +30 210 281 1712, κατόπιν αιτήσεως.

▼Το φάρμακο αυτό τελεί υπό συμπληρωματική παρακολούθηση. Αυτό θα επιτρέψει το γρήγορο προσδιορισμό νέων πληροφοριών ασφάλειας. Ζητείται από τους επαγγελματίες υγείας να αναφέρουν οποιεσδήποτε πιθανολογούμενες ανεπιθύμητες ενέργειες.

ΟΝΟΜΑΣΙΑ ΤΟΥ ΦΑΡΜΑΚΕΥΤΙΚΟΥ ΠΡΟΪΟΝΤΟΣ: FABHALTA 200 mg σκληρά καψάκια. **ΠΟΙΟΤΙΚΗ ΚΑΙ ΠΟΣΟΤΙΚΗ ΣΥΝΘΕΣΗ:** Κάθε καψάκιο περιέχει υδροχλωρική μονοϋδρική ιπτακοπάνη, η οποία ισοδυναμεί με 200 mg ιπτακοπάνης. **ΚΑΤΟΧΟΣ ΤΗΣ ΑΔΕΙΑΣ ΚΥΚΛΟΦΟΡΙΑΣ:** Novartis Europharm Limited, Vista Building, Elm Park, Merrion Road, Dublin 4, Ιρλανδία.

Συσκευασία/Τιμή: Ενδεικτική Λιανική Τιμή: FABHALTA CAPS 200MG/CAP BT X 56 ΚΑΨΑΚΙΑ ΣΕ BLISTER (PVC/PE/PVDC/ALU): €29.424,52
Ενδεικτική Νοσοκομειακή Τιμή: FABHALTA CAPS 200MG/CAP BT X 56 ΚΑΨΑΚΙΑ ΣΕ BLISTER (PVC/PE/PVDC/ALU): €24.469,09
Ημερομηνία δελτίου τιμών: 11/03/2025 – σε περίπτωση τροποποίησης του δελτίου τιμών ισχύει η νεότερη τιμή (συμπεριλαμβανομένου ΦΠΑ όπως ισχύει).
Τρόπος διάθεσης: Φαρμακευτικό προϊόν για το οποίο απαιτείται ιατρική συνταγή.

Βοηθήστε να γίνουν τα φάρμακα πιο ασφαλή και Αναφέρετε
ΟΛΕΣ τις ανεπιθύμητες ενέργειες για **ΟΛΑ τα φάρμακα**
Συμπληρώνοντας την **«ΚΙΤΡΙΝΗ ΚΑΡΤΑ»**

FAB_ADV_001_Apr/2025/FA-11394880

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ORGANIZER

Cyprus Society of Haematology

Under the auspices of

Hellenic Society of Haematology

Israeli Society of Hematology and Blood Transfusion

7th GRECO - ISRAELI Hematology Meeting

8th-10th MAY 2025

Porto Palace Hotel

THESSALONIKI GREECE

GENERAL INFORMATION

Meeting dates and venue

May 8th-10th, 2025

Porto Palace Hotel, Thessaloniki, Greece

(65, 26th Oktovriou Avenue, Port 54628, tel.:+30 2310 504504, www.portopalace.gr)

Certificate of attendance

The certificates will be sent via email by the end of the meeting and as soon as the evaluation form will be submitted to the meeting secretariat.

Congress badge

It is mandatory for the delegates to show their congress badge at the entrance of the Meeting halls in order to calculate their attendance time in all sessions.

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. Presentations must be uploaded at the technical secretariat one hour prior to the presentation. The use of personal computers will not be feasible.

Registration fees

Registration type	Cost
Specialists	**120,00 €**
Residents	**50,00 €**
Other health professionals	**60,00 €**
Nurses	**30,00€**
Undergraduate students*	**10,00 €**

** Students should cover the registration fees on their own, because pharmaceutical companies is not allowed to cover undergraduate students.*

24% VAT is not included in the above registration fees.

7th GRECO - ISRAELI Hematology Meeting

8th-10th MAY 2025
Porto Palace Hotel
THESSALONIKI
GREECE

Registration fees include:

- Admission to the scientific sessions
- Congress material
- Certificate of attendance
- Admission to the exhibition area

Registration should be completed via online platform at the website www.globalevents.gr

Event secretariat

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Conference & Event Management

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ACKNOWLEDGEMENTS

The Organizing Committee of the Meeting would like to acknowledge the following companies

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ΠΡΟΪΟΝ	ΔΡΑΣΤΙΚΗ ΟΥΣΙΑ	ΤΙΜΗ ΠΑΡΑΓΩΓΟΥ	ΝΟΣΟΚΟΜΕΙΑΚΗ ΤΙΜΗ	ΝΟΣΟΚΟΜΕΙΑΚΗ ΤΙΜΗ -5%
XOSPATA F.C.TAB 40MG/TAB BTx84 δισκία σε blisters (OPA/αλουμινίου/PVC/αλουμινίου)	GILITERITINIB	15.485,14 €	14.131,74 €	13.425,15 €

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Περαιτέρω πληροφορίες διατίθενται από τον κάτοχο της άδειας κυκλοφορίας κατόπιν αιτήσεως και περιλαμβάνονται στη συνοπτική περιγραφή χαρακτηριστικών του προϊόντος και το φύλλο οδηγιών χρήσης του φαρμάκου.

XOS/ADV1/10.2024 MAT-GR-XOS-2024-00006

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