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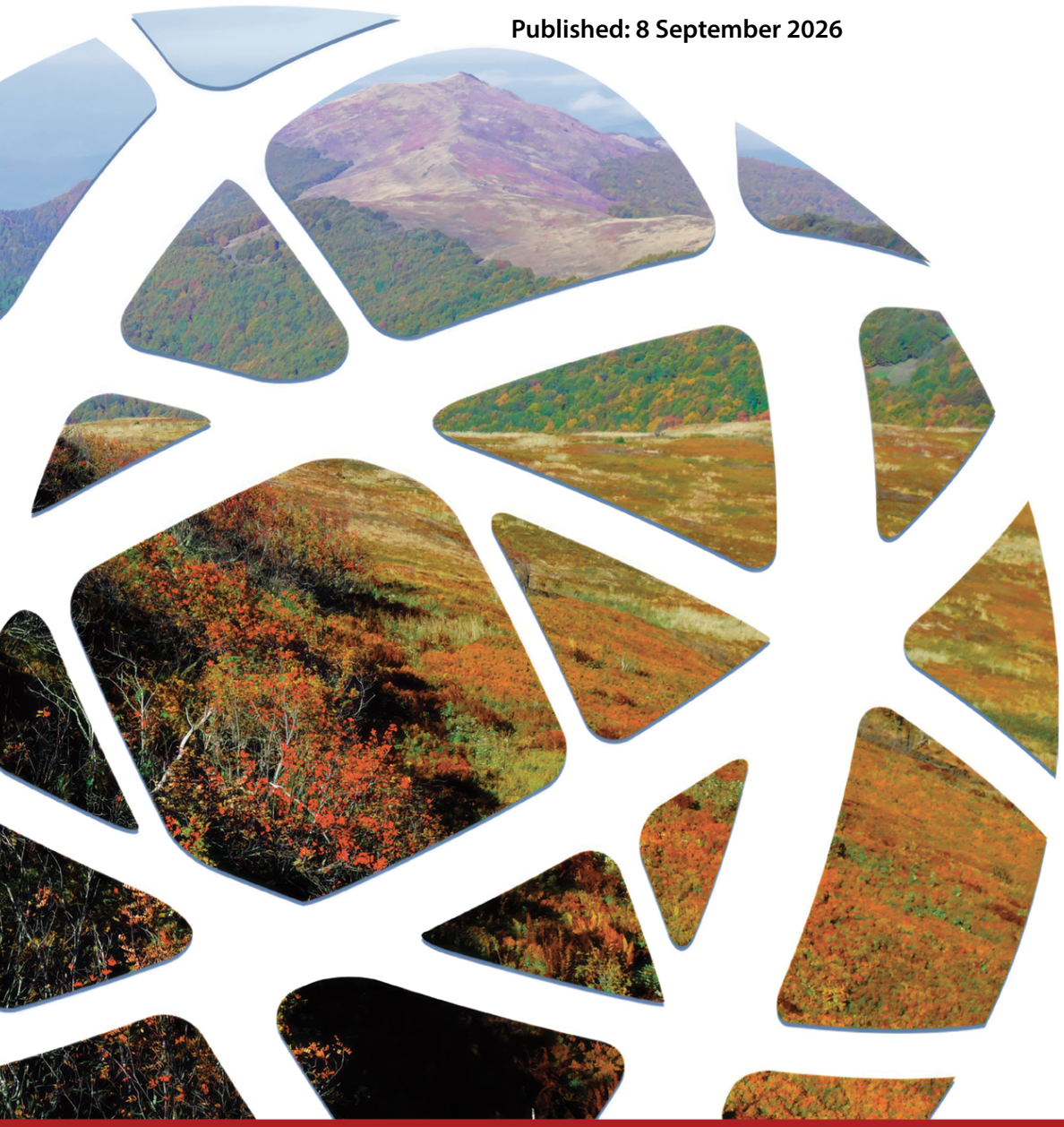

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SPIS TREŚCI

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Topic: Acute Leukemias and Myelodysplastic Syndromes

Title: TERT-related dyskeratosis congenita presenting as apparent acquired aplastic anaemia with cerebral venous sinus thrombosis – a case report

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ABSTRACT

Introduction and aim. Dyskeratosis congenita (DC) is a rare inherited telomeropathy that may present without classical features, leading to delayed diagnosis. The unusual case of TERT-related DC will be presented. A retrospective review of the clinical course was performed using clinical records, laboratory investigations, serial bone marrow biopsies, imaging studies, telomere length assessment, next-generation sequencing (NGS), and whole-exome sequencing (WES).

Description of the case. A healthy 16-year-old girl presented with pancytopenia and non-severe aplastic anaemia. During evaluation, she developed transient facial nerve palsy, a suspected cerebellopontine tumour, and extensive cerebral venous sinus thrombosis despite profound thrombocytopenia. Repeated bone marrow biopsies excluded myelodysplastic syndrome and malignancy. Investigations excluded autoimmune and infectious disorders, thrombophilia, myelodysplastic syndrome, haematological malignancy, and inherited red cell disorders. Markedly shortened telomeres (3.99 kb) together with biallelic TERT variants confirmed the diagnosis of dyskeratosis congenita. A concurrent ETV6 variant was also identified. Following treatment with thrombopoietin receptor agonists and danazol, the patient's haematological parameters remained clinically stable during follow-up.

Conclusion. Inherited telomere biology disorders should be considered in young patients with unexplained bone marrow failure, particularly when atypical features or unexpected complications occur. Early telomere length testing and genetic analysis are essential to establish the diagnosis, guide management and haematopoietic stem cell transplantation.

Keywords. aplastic anaemia, bone marrow failure, cerebral venous sinus thrombosis, dyskeratosis congenita, telomere biology disorder

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Topic: Acute Leukemias and Myelodysplastic Syndromes

Title: A case of a rare KMT2A gene abnormality in B/myeloid mixed phenotype acute leukemia

Authors: Jakub Łączak [1], Marta Szarawarska [2], Dominika Dudycz [3], Karolina Bieńko [3], Jarosław Grzyb [1], Tomasz Skoczylas [1], Beata Blajer-Olszewska [1], Agnieszka Kopacz [1], Mirosław Markiewicz [1]

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ABSTRACT

Introduction and aim. Mixed-phenotype acute leukemia (MPAL) is a rare hematologic malignancy characterized by ambiguous lineage differentiation and poor clinical outcomes. We present the case of a 62-year-old patient diagnosed with B-cell/myeloid MPAL and a rare KMT2A abnormality identified during diagnostic evaluation.

Description of the case. The disease had an aggressive clinical course, with central nervous system involvement, severe coagulation abnormalities, secondary hemophagocytic syndrome, and recurrent infectious complications leading to progressive multiorgan failure. Despite intensive chemotherapy and supportive treatment, the patient developed septic shock and died on the 25th day of hospitalization.

Conclusion. Despite intensive treatment, the disease followed a highly aggressive course with multiple complications and progressive treatment failure, ultimately resulting in the patient's death. KMT2A alteration in MPAL is a very high risk factor associated with adverse outcomes.

Keywords. cytogenetic abnormality, KMT2A, mixed-phenotype acute leukemia, MPAL, risk factors

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Topic: Acute Leukemias and Myelodysplastic Syndromes

Title: Artificial intelligence in measurable residual disease assessment in acute leukemias – current applications and future directions

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ABSTRACT

Introduction and aim. Measurable residual disease (MRD) is a key prognostic and predictive biomarker in acute leukemias. Artificial intelligence (AI) offers new opportunities to improve MRD assessment by enhancing data analysis, standardization, and reproducibility. This review summarizes current evidence on AI applications in MRD assessment and discusses future clinical directions.

Literature search. A narrative review of peer-reviewed PubMed literature published between 2024 and 2026 was performed to identify studies evaluating AI-based approaches for MRD assessment, including flow cytometry, molecular diagnostics, and integrated data analysis.

Analysis of the literature. AI algorithms improve automated multiparametric flow cytometry, support molecular MRD assessment, and facilitate interpretation of complex diagnostic data. Recent evidence demonstrates excellent diagnostic performance of AI-assisted flow cytometry, improving workflow efficiency, reducing observer-dependent variability, and enhancing reproducibility. Integration of flow cytometry, molecular techniques, and clinical data may further improve risk stratification and treatment monitoring.

Conclusion. AI is a promising tool for MRD assessment in acute leukemias. Although prospective validation and standardization remain necessary, AI is expected to become an important component of future laboratory hematology, complementing expert interpretation rather than replacing it.

Keywords. acute leukemia, artificial intelligence, flow cytometry, measurable residual disease, molecular diagnostics

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Topic: Acute Leukemias and Myelodysplastic Syndromes

Title: A case of acute myeloid leukemia presenting divergence of morphologic and immunophenotypic features

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ABSTRACT

Introduction and aim. Heterogeneous nature and overlapping features of acute leukemias of uncertain lineage evoke difficulties in both diagnosis and treatment outcome. We present a study of an acute leukemia showing divergence in diagnostic tests results.

Description of the case. Multiple diagnostic procedures were performed, including histologic and cytologic evaluation of bone marrow as well as multiparameter flow cytometry analysis.

Bone marrow cytology revealed population of small blast cells constituing 22% of marrow cellularity. Immunophenotypic evaluation showed two aberrant blast cell populations: first with B/myeloid features presenting the following antigen pattern: CD45+/CD34+/CD117+/MPO-/CD19+/CD10-/cyCD22+/-/cyCD79a+/TdT+/-/HLA-DR+/CD33+/-/CD13+/CD71-/CD41a-/CD42b-; and second with myeloid differentiation features presenting antigen pattern: CD45+/CD34+/CD117+/MPO-/CD19-/CD10-/cyCD22-/cyCD79a-/TdT-/HLA-DR-/CD33-/+/CD13+/-/CD71-/CD41a-/CD42b-. Since both populations did not express MPO, criteria for mixed phenotype acute leukemia presented by the WHO 2022 could not be followed and EGIL score system for BAL diagnosis was applied. Score results were as follows: 3 points for myeloid lineage, 5.5 points for B lineage, suggesting biphenotypic leukemia.

Histological evaluation of bone marrow indicated diagnosis of myelodysplastic syndrome/myeloproliferative neoplasm with fibrosis and transformation to acute myeloid leukemia with features of megakaryocytic differentiation.

Conclusion. Presented study shows complexity of acute leukemia diagnostic workflow and confirms the need for detailed and coordinated results analysis for accurate interpretation.

Keywords. acute myeloid leukemia, differential diagnosis, immunophenotype

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Topic: Acute Leukemias and Myelodysplastic Syndromes

Title: The case of a patient with blastic transformation of primary myelofibrosis and the role of IDH1 mutation

Authors: Dominika Strzała [1,2], Aleksandra Połom [2], Wojciech Homenda [2,3], Izabela Zawlik [4]

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ABSTRACT

Introduction and aim. Acute myeloid leukemia (AML) resulting from myelofibrosis (MF) accounts approximately 8% of all AML cases and has a poor prognosis. Median survival time after transformation is approximately 2.6 months. The aim of this paper is to highlight the prognosis of patients with post-MF AML and an IDH1 mutation in the era of targeted therapies.

Description of the case. Here, we present a 68-year-old female with JAK2-positive MF who developed AML with an IDH1 mutation and began treatment with ivosidenib. The case is based on medical records from 2022 to 2026. We also summarized the latest published literature on diagnosing and treating post-MF AML, focusing specifically on the role of IDH1 mutations. The IDH1 mutation can occur in the chronic phase of MF but is detected more frequently in the blastic phase. This indicates its driving influence on blastic, rather than fibrotic, progression. Targeted therapies, including ivosidenib, have been available for patients with this mutation for several years. However, clinical data evaluating their effectiveness in this group remain significantly limited.

Conclusion. Using IDH1 inhibitor therapy in patients with post-MF AML may positively impact their historically poor prognosis and improve survival. Emerging data suggest potential benefits of utilizing IDH1 inhibitors already in the accelerated phase. However, large-scale studies on post-MF AML patients treated with ivosidenib are still lacking.

Keywords. IDH1 mutation, ivosidenib, myelofibrosis, secondary acute myeloid leukemia

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Topic: Coagulopathies and Hemolysis

Title: Diagnosis and treatment of congenital thrombotic thrombocytopenic purpura in the pregnant woman

Authors: Beata Blajer-Olszewska [1], Katarzyna Kalandyk-Osinko [2], Agnieszka Kopacz [1], Sylwia Kot [3], Adrianna Włodyka-Pękosz [3], Dagmara Olszewska [3], Joanna Zdziarska [4], Teresa Iwaniec [5], Tomasz Kluz [2], Mirosław Markiewicz [1]

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ABSTRACT

Introduction and aim. Although cTTP results from a congenital mutation of the ADAMTS13 gene, in some cases the manifestation of the disease depends on an additional triggering factor, and the diagnosis is made in adulthood. In pregnancy, it is a life-threatening disease for both the mother and fetus.

Description of the case. We present the case of a 30-year-old woman with a history of ischemic strokes and one miscarriage, in whom at the 24th week of her second pregnancy extensive hemolytic anemia developed and was finally diagnosed as cTTP.

After a complicated initial treatment with FFP, patient received replacement therapy with recombinant ADAMTS13. Due to preeclampsia diagnosed on the 37th day of treatment, the pregnancy was terminated by cesarean section, without complications.

Conclusion. Prompt diagnosis of cTTP and quick start of replacement therapy with rADAMTS13 is essential for successfull termination of pregnancy enabling birth of a healthy child and an uncomplicated postpartum course.

Keywords. congenital thrombotic thrombocytopenic purpura, pregnancy, recombinant ADAMTS13

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Topic: Coagulopathies and Hemolysis

Title: Combined paroxysmal nocturnal hemoglobinuria therapy with ravulizumab-danicopan – first experiences of the center

Authors: Katarzyna Warzybok [1], Agnieszka Kopacz [1], Beata Blajer-Olszewska [1], Mirosław Markiewicz [1]

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ABSTRACT

Introduction and aim. Combined therapy for paroxysmal nocturnal hemoglobinuria (PNH), using ravulizumab, a C5 inhibitor, and danicopan, an oral Factor D inhibitor, targets both intravascular and C3-mediated extravascular hemolysis. We report the first center experience with this approach.

Description of the case. A 59-year-old previously untreated patient was admitted urgently with hemolytic anemia and suspected PNH. Bone marrow aspiration suggested myelodysplastic syndrome, requiring interpretation alongside laboratory findings, while trephine biopsy showed no bone marrow pathology. Flow cytometry confirmed a GPI defect in 94.71% of neutrophils and a PNH phenotype in 53.87% of erythrocytes. Considering the patient's age, general condition and fatigue (FACIT score 30), ravulizumab was initiated on 11 July 2024 within the drug program "Management of patients with paroxysmal nocturnal hemoglobinuria." During treatment, hemoglobin remained at 8.0–9.0 g/dL, LDH was >1.5 x ULN and one blood transfusion was required. A positive DAT test suggested extravascular hemolysis. On 18 April 2025, danicopan was added through early access and later continued within a drug program. Hemoglobin increased to 11.5–12.8 g/dL, LDH decreased to <1.5 x ULN and the patient became transfusion-independent.

Conclusion. Adding danicopan to ravulizumab improved control of intravascular and extravascular hemolysis, resulting in stable hemoglobin increase and transfusion independence.

Keywords. complement inhibitors, hemolysis, paroxysmal nocturnal hemoglobinuria

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Topic: Lymphoproliferative Disorders

Title: Efficacy of zanubrutinib in the rare concurrent presentation of Waldenström's macroglobulinemia and myeloproliferative neoplasm

Authors: Jarosław Grzyb [1], Marzena Wojtaszewska [1], Marta Szarawarska [1], Mirosław Markiewicz [1]

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ABSTRACT

Introduction and aim. Waldenström's macroglobulinemia (WM) concurrent with myeloproliferative neoplasms (MPNs) is extremely rare. While Bruton's tyrosine kinase inhibitors (BTKis) like zanubrutinib are highly effective in WM, their impact on MPNs remains unexplored. This study presents a unique patient with concurrent WM and MPN-NOS treated with zanubrutinib.

Description of the case. A 61-year-old male presented with severe fatigue, night sweats, and weight loss. Diagnostics confirmed concurrent malignancies: WM (IgM 25.12 g/L, *MYD88 L265P*) and MPN-NOS (platelets 789x10^9/L, bone marrow reticulin fibrosis grade 2+, *JAK2 V617F*). The patient received zanubrutinib (320 mg/day). Hydroxycarbamide was initially co-administered for cytoreduction. After 6 months, a very good partial response (VGPR) for WM was achieved, featuring complete IgM normalization and *MYD88 L265P* eradication from bone marrow. Concurrently, MPN parameters regressed: platelet counts persistently decreased below 500x10^9/L, allowing complete hydroxycarbamide discontinuation. Reticulin fibrosis reduced to grade 1+, and *JAK2 V617F* variant allele frequency stabilized at 18% without clonal expansion. No significant adverse events were recorded.

Conclusion. Zanubrutinib monotherapy induced a profound, simultaneous response in both lymphoid (WM) and myeloid (MPN-NOS) compartments. This unprecedented case underscores the pleiotropic efficacy of BTK inhibition in managing concurrent lymphoproliferative and myeloproliferative disorders, providing a strong rationale for further investigations in MPN therapy.

Keywords. BTK inhibitor, concurrent malignancies, JAK2 V617F, myeloproliferative neoplasms, Waldenström's macroglobulinemia, zanubrutinib

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Topic: Lymphoproliferative Disorders

Title: Beyond single biomarkers: a hierarchical, phase-specific model of chronic lymphocytic leukemia driven by BCR architecture and genomic context

Authors: Anna Sobczyńska-Konefał [1,2], Robert Mroczek [1], Anna Jaśkowiec [2], Marta Lemieszewska-Gajek [2], Magdalena Kaczmarek [2], Natalia Wiski [2], Paweł Gliński [2], Klaudia Gontarz [2], Monika Jasek [1], Lidia Karabon [1], Jarosław Dybko [2,3], Dagmara Michałowska [2], Emilia Jaskuła [1,2]

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ABSTRACT

Introduction and aim. Risk stratification in CLL relies on biomarkers, yet clinical behavior diverges from predictions. We hypothesized that disease kinetics are shaped by hierarchical interaction between B-cell receptor architecture and genomic alterations. This study aimed to evaluate whether CLL dynamics are driven by phase-specific interplay between BCR structure and genomic lesions, and to define a model in which BCR architecture establishes the primary program while genomic events act in a context-dependent manner.

Material and methods. We conducted immunogenetic, cytogenetic, and genomic profiling in 215 CLL patients. *IGHV* status and BCR features (V(D)J usage, CDR3 structure) were assessed in 204 cases. Cytogenetic abnormalities and mutations were analyzed using FISH and NGS. Clinical endpoints included TTFT, TTST, PFS. Multivariable Cox models evaluated context-dependent effects.

Results. *IGHV* status was the determinant of early disease behavior, with U-CLL showing shorter TTFT (HR2.6, $p<0.0001$). U-CLL exhibited a distinct BCR phenotype (IGHV1-69 usage, longer CDR3, higher aromatic content). Prognostic effects of mutations were IGHV-dependent: *NOTCH1* was adverse only in U-CLL, while *SF3B1* impacted M-CLL. Biological clusters refined risk, including a BCR-driven hub with poor TTFT/TTST and a DNA damage hub (*ATM*/del(11q)). Post-treatment outcomes were dominated by *TP53* disruption and *XPO1* mutations.

Conclusion. CLL follows a hierarchical, phase-specific model where BCR architecture drives early disease, while genomic lesions modulate progression and resistance, supporting integrated risk stratification.

Keywords. B cell receptor (BCR) architecture, CDR3 structure, chronic lymphocytic leukemia (CLL), genomic alterations, mutated CLL (M-CLL), unmutated CLL (U-CLL)

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Topic: Myeloproliferative Neoplasms

Title: An unusual case of 59-Year-Old man with Ph(-) *BCR*::*ABL*1(+) chronic myeloid leukemia and complex variant translocation between chromosomes 9, 11 and 22

Authors: Agata Szymańska-Szura [1], Marta Szarawarska [1], Marzena Wojtaszewska [2,3], Kamila Pawlik[1], Jakub Łączak [1], Mirosław Markiewicz [1,3]

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ABSTRACT

Introduction and aim Chronic myeloid leukaemia (CML) is characterised by fusion of *BC*R and *ABL1* genes. In ~90% of patients it results from classic t(9;22) translocation, leading to Ph chromosome. The remaining 5–10% of cases involve variant translocations, sometimes masked. We present case of Ph(-) CML with rare variant rearrangement leading to atypical localisation of BCR::ABL1 fusion gene.

Material and methods. The diagnosis was based on conventional cytogenetic analysis supplemented by FISH and molecular testing. Thirty metaphases derived from short-term cell cultures were analysed using GTG banding. Probes for chromosomes 9, 11 22, and D-BCR/ABL and ABL1 Break-apart-specific probes were used for FISH analysis. RT-qPCR was used for BCR::ABL1 transcript identification.

Results. Conventional cytogenetics revealed presence of t(11;22)(q11;q11) in all metaphases. FISH analysis, however, revealed presence of only one fusion signal between *ABL1*(9q34) and *BCR*(22q11), located on derivative chromosome der(9), and single free signals of both genes, indicating the insertion of *BCR* gene fragment into *ABL1* on der(9). A classic rearrangement of *ABL1* gene was ruled out, which further supports the insertion of *BCR* gene fragment into *ABL1*. RT-qPCR analysis revealed typical P210 transcript.

Conclusion. Clinical picture confirms diagnosis of Ph(-) BCR::ABL1(+) CML, resulting from a variant translocation involving chromosomes 9, 11 and 22. As only a single gene fusion is present, treatment results monitoring requires application of molecular techniques and is impossible with FISH.

Keywords. chronic myeloid leukemia, masked translocation, BCR::ABL1 fusion, insertion, cytogenetic and molecular diagnostics

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Topic: Plasma Cell Dyscrasias

Title: Cytometric evaluation of TNF-α expression in CD3+ T lymphocytes and its clinical correlations in patients with plasma cell myeloma

Authors: Karolina Basista [1,3], Katarzyna Karwicka [1,2], Arleta Malecha-Jędraszek [2], Marek Hus [1]

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ABSTRACT

Introduction and aim. PCM pathogenesis involves cytokine network deregulation. This study aimed to evaluate spontaneous serum TNF-α concentration and its intracellular expression in activated CD3+ T lymphocytes from PCM patients, analyzing their clinical correlations.

Material and methods. The study involved 40 newly diagnosed PCM patients and 20 healthy volunteers. Peripheral blood mononuclear cells were cultured for 24h (37°C, 5% CO_2); PMA, ionomycin, and protein transport inhibitors were added 4h before harvest, using appropriate controls. Intracellular TNF-α was evaluated by flow cytometry using fluorochrome-conjugated antibodies, and serum concentration by CBA method. Data were analyzed using Statistica 13.0PL.

Results. The TNF-α+/CD3+ [MFI] parameter was significantly lower in patients compared to the control group (mean: 8543.60 ± 5725.28 vs. 11853.50 ± 3634.61; median: 6715.50 vs. 11277.50; p<0.001). No significant differences were found for TNF-α+/CD3+ [%] (p=0.211) and serum TNF-α (p>0.05). TNF-α+/CD3+ [%] correlated positively with age (R=0.325; p=0.041), albumin (R=0.466; p=0.002), and absolute lymphocyte count (R=0.364; p=0.021). The TNF-α+/CD3+ [MFI] parameter showed an association with albumin (R=0.338; p=0.032) and absolute lymphocyte count (R=0.500; p=0.001). Serum TNF-α correlated with erythrocyte count and hematocrit (for both: R=0.323; p=0.042).

Conclusion. Impaired TNF-α secretion indicates functional T-cell exhaustion in the tumor microenvironment. Decreased serum TNF-α, accompanied by lower red blood cell parameters, reflects progressive bone marrow remodeling. Increased intracellular TNF-α expression in older patients manifests physiological immunosenescence.

Keywords. plasma cell myeloma, TNF-alpha, T lymphocytes, flow cytometry, tumor microenvironment, immunosenescence

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Topic: Plasma Cell Dyscrasias

Title: Autologous hematopoietic stem cell transplantation in patients with multiple myeloma – center's experience

Authors: Beata Blajer-Olszewska [1], Agnieszka Kopacz [1], Aleksandra Rejus-Gajdek [1], Sylwia Kot [2], Adrianna Włodyka-Pękosz [2], Katarzyna Warzybok [1], Marcin Klimko [3], Antonina Krzyształowicz [3], Emilia Król [3], Mirosław Markiewicz [1]

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ABSTRACT

Introduction and aim. Plasma cell myeloma (PCM) is the most common indication for autologous stem cell transplantation (ASCT), recommended as first-line treatment. We present results of ASCT in PCM obtained during first three years (03.2023-04.2026) of transplant center's activity.

Material and methods. 27 patients (16-F,11-M) with PCM, aged 60 (38–72) years were transplanted. At diagnosis serum M-protein was 3.86(1.9-6.6)g/dl, i/ni (involved/noninvolved) FLC-ratio 260.11 (2.33–1692.15); t(4;14)-2pts, t(14;16)-2pts, del(17p)-3pts, none of mentioned cytogenetic changes-20pts. In induction, 24pts received 4(4-6)xDVTd cycles, remaining 3pts 6(5-7)xVTd/VCd and 2xKd-PACE/1xVd/1xVTd. At least VGPR was achieved before ASCT in 22pts, PR-4pts, Minimal Response-1pt; serum M-protein was 0.22 (0.0–1.6)g/dl, i/niFLCratio 10.22(0.47-78.22). Conditioning was Melphalan 200/140mg/m^2 in 18/9pts.

Results. At median 5.2(3.1-7.7)x10e6CD34+cells/kg were transplanted. Reconstitution of PLT>50G/l was achieved on day21(17-38), ANC>0.5G/l on day13(11-21). Post-ASCT at least VGPR was achieved in 26pts, PR-1pt; serum M-protein was 0.03 (0.0–0.5) g/dl, i/niFLCratio 2.246 (0.44–7.64). After ASCT, 23pts received maintenance therapy with additional DVTd cycles up to 5(1pt) or 6(21pts), lenalidomide (7pts), second ASCT (5 pts) 6 (3–7) months after the first one. Post-transplant complications were observed in 18(66.6%)pts: diarrhea-12pts, stomatitis-7, CMV reactivation-2, sepsis-2, ITP-2, arrhythmia-1. With median follow-up 19 (2–38) months, PCM progression was noted in 3pts at 20 (16–26) months, and 1pt died after secASCT (CNS bleeding); 26 (96%) patients are alive and 23 (85%) are alive without progression.

Conclusion. This retrospective analysis confirms the effectiveness and safety of ASCT in patients with PCM.

Keyword. autologous stem cell transplantation, first-line treatment, plasma cell myeloma

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Topic: Plasma Cell Dyscrasias

Title: CDI affects nearly half of BeEAM-conditioned patients and dominates early infectious complications after auto-HSCT

Authors: Filip Krzanowski [1], Anna Szymańska [2], Jakub Nowak [3], Aleksandra Czępińska- Myszura [4], Małgorzata Kozioł [5], Jacek Krzanowski [6]

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ABSTRACT

Introduction and aim. Patients undergoing auto-HSCT for multiple myeloma (MM) or lymphoma are at risk of early infectious complications due to conditioning chemotherapy, neutropenia, and mucosal barrier injury. We assessed their spectrum and risk factors, with particular focus on *Clostridioides difficile* infection (CDI), across conditioning regimens.

Material and methods. This retrospective study included 125 patients: 108 (86.4%) with MM received melphalan (MEL-140/200) and 17 (13.6%) with lymphoma received BeEAM conditioning. Independent risk factors were identified by multivariable logistic regression ($p<0.05$).

Results. Infectious complications occurred in 44 patients (35.2%) and were far more frequent after BeEAM than melphalan (70.6% vs 29.6%). CDI was the predominant documented infection (26 patients; 20.8%; 59.1% of infectious episodes). Other pathogens were sporadic: 6 Gram-negative, 5 Gram-positive isolates, and 2 viral infections (BK virus, SARS-CoV-2). Infection was also associated with longer grade IV neutropenia ($p=0.0459$). CDI was more frequent after BeEAM (47.1% vs 16.7%; $p=0.0475$) and associated with prolonged antibiotic therapy (empirical and targeted; $p=0.0034$). On multivariable analysis, duration of antibiotic therapy excluding vancomycin and metronidazole was the only independent predictor of CDI (OR 1.52/day; $p=0.0224$). No CDI-related deaths occurred.

Conclusion. CDI dominates early infectious complications after auto-HSCT. BeEAM-conditioned patients represent a higher-risk group, warranting targeted surveillance and antimicrobial stewardship.

Keywords. auto-HSCT, BeEAM conditioning, CDI, post-transplant infections