with higher Hgb (p = -0.022), WBC (0.029), higher ANC (p = -0.008), higher eosinophil count (p = -0.03) and lower bone marrow blast count (p = -0.022). Jak2V617F positivity was also associated with lower LDH, lower TSS and higher PLT count, but this was not statistically significant. The splenomegaly rate didn't differ between groups (Jak2 positive – 94.44% and Jak2 negative – 84.62%; p = 0.203). Median follow-up was 34.61-months. Although statistically insignificant, Jak2V617F negative patients seems to have better OS than Jak2V617F positive patients (p = -0.644). Median OS didn't reach in Jak2 negative group vs. 155-months in Jak2 group (Fig. 1). **Conclusion:** Comparison of clinical and laboratory data between Jak2 positive and negative groups in patients with primary myelofibrosis in Azerbaijan has been performed. In our cohort, Jak2V617F positive have significantly higher Hgb, Wbc, ANC, bone marrow blast and eosinophil counts, also higher PLT, lower LDH and Total Symptom burden (TSS), but it's not statistically significant. Similar to our study, article by Vannucchi A.M and colleagues published in the journal Leukemia in 2008, the authors showed that JAK2 V617F mutations in PMF are associated with older age, higher HB level, leukocytosis, and lower platelet count.[1] How Jak2V617F mutation affects the OS in PMF remains controversial. Although it's not statistically significant, we found that Jak2V617F negative patients have a better median OS than positive patients in our cohort. Unlike this, in a multicenter study of 152 patients, Campbell PJ et. al. showed that in PMF, the presence of JAK2V617F was associated with inferior survival despite the fact that mutated patients were less likely to require red blood cell transfusions during follow-up.[2] On the contrary, in a series of 117 PMF patients from a single center, Tefferi et al. reported no significant impact of V617F presence on either survival or leukemic transformation.[3] But we didn't have the exact rate of the CALR and the MPL mutation rate in the Jak2-negative group in our cohort, so we didn't know how this mutation was affecting our study results. So the small number of patients in the comparison groups and the lack of testing for ASXL1, lower number of CALR, MPL mutation is a limitation of our study. There is a need for prospective, large studies with comprehensive genetic testing to learn exactly how genetic mutations affect survival in our PMF patients.

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Table 1 Patient characteristics.

Variable	Jak2V617F positive (n = 91)	Jak2V617F (n = 32)	p-value
Age	58.0 (IQR = 14.0)	54.0 (IQR = 13.5)	0.527
Gender			0.25
Male	40 (43.96%)	18 (58.06%)	
Female	51 (56.04%)	13 (41.94%)	
Stage	n = 74	n = 25	>0.801
Pre-PMF	21 (28.38%)	8 (32.0%)	
Overt PMF	53 (71.62%)	17 (68.0%)	
Bone marrow blast	0.2 (IQR 0.8)	0.4 (IQR 0.8)	0.022
Hgb × 10^9/L	12.4 (IQR 4.4)	10.8 (4)	0.016
WBC × 10^9/L	15.15 (IQR 15.0)	11.42 (IQR 10.25)	0.029
ANC × 10^9/L	10.81 (IQR 11.57)	7.39 (IQR 6.7)	0.008
ALC × 10^9/L	2.08 (IQR 1.24)	1.96 (IQR 1.32)	0.25
PLT × 10^9/L	438.0 (IQR 404.0)	366.0 (IQR 525.5)	0.778
LDH U/L	478.15 (IQR 478.0)	515.0 (IQR 267.0)	0.846
Eosinophil × 10^9/L	0.3 (IQR 0.6)	0.15 (IQR 0.28)	0.03
Basophil × 10^9/L	0.02 (IQR 0.16)	0.007 (IQR 0.068)	0.363
Spleen size, cm	18.85 (IQR 4.6), n = 72	18.4 (4.98), n = 26	0.645
Liver size, cm	15.85 (IQR 2.45), n = 72	16.35 (IQR 2.85), n = 26	0.156
MPN TSS, initial	5.0 (IQR 10.0), n = 9	11.0 (IQR 13.5), n = 31	0.347

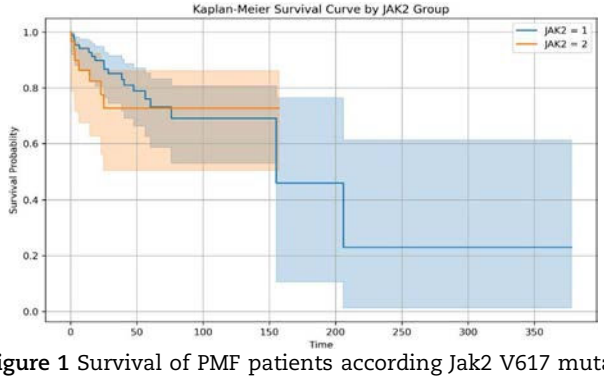

Figure 1 Survival of PMF patients according Jak2 V617 mutational status.

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OP 11_Case report

TREATMENT OF BLAST PHASE MYELOPROLIFERATIVE NEOPLASM WITH THE COMBINATION OF AZACITIDINE, VENETOCLAX AND RUXOLITINIB

Fidan Khalilova, Azer Kerimov, Gulnar Kerimova

National Center of Hematology and Blood Transfusion, Vietnam

Objective: In the development of Myeloproliferative Neoplasm (MPN), transformation to the Blast Phase (BP) is often noted. Thus, the incidence of BP in Primary Myelofibrosis (PMF) is -9%–13%, in Essential Thrombocythemia (ET) -1%–4%, and in Polycythemia Vera (PV) -3%–7%. As a result of the development of ET and PV, transformation to Myelofibrosis (MF) can also be noted. In this case, differentiation of PMF from post-ET-MF and post-PV-MF can be difficult. In the treatment of these diseases, an individual approach according to the history and comorbidity, increases the effectiveness of treatment. **Methodology:** Patient U.T., born in 1955, was registered at the NCHBT in June 2019 with a diagnosis of PMF. At

the time of initial admission, the patient complained of abdominal distension and severe weight loss over the past 6-months. During examinations, a splenomegaly (204 × 85 mm) was found. In hemogram: Hb – 151 g/L, WBC – 43 × 10^9/L, PLT – 779 × 10^9/L were. Histological examination of the bone marrow revealed that the bone cavities were filled with fibrotic stroma, no fat cells were detected. Hematopoietic cells were diffusely scattered, the cellular composition consisted of granulocytic and megakaryocytic orders. A reduction in the erythroid order was noted. The number of megakaryocytes was increased, acute polymorphism was noted, atypical forms were abundant. Megakaryocytes formed dense and sparse clumps (up to 6-cells) and layers, their paratrabecular localization was noted. Areas of coarse-fiber collagen fibrosis were noted. During molecular genetic examination, the allelic load of the JAK2V617F mutation was 92.694%. The patient was treated with Hydrea (HY)from June 2019 to September 2019. Since the hemogram did not show positive dynamics, Interferon (IFN) 3 million units was administered intramuscularly 3 times a week from September 2019. After this administration, a relative decrease in spleen size was noted. Starting from March 2020, the patient's condition deteriorated again. In hemogram: Hb – 161 g/L, WBC – 34 × 10^9/L, PLT – 343 × 10^9/L were. The spleen size was 200 × 84 mm on Ultrasound Scan (USS). The patient was prescribed HY 1000 mg p/day along with IFN. Positive dynamics were achieved as a result of treatment with HY+IFN. Hemogram: Hb – 120 g/L, WBC – 4 × 10^9/L, PLT – 476 × 10^9/L; spleen in palpation was +4 cm. Treatment with HY+IFN was continued until April 2021. From April 2021, treatment was continued with HY alone. In May 2024, the patient's condition worsened. Morphological examination of the bone marrow showed 16% blasts, histological examination showed 20% blasts, blasts were of myeloid type. Transformation of the disease to the BP was recorded. The patient was prescribed 2 courses of low-dose Cytosar. Since no positive dynamics were noted and blasts in the bone marrow increased to 78.6%, treatment with Azacitidine (AZA) + Venetoclax (VEN) was initiated in July, and after the 2nd course, clinical-hematological remission was recorded (blasts on myelogram were 0.8%). Although the patient's hemogram and bone marrow results showed Morphological Leukaemia-Free State (MLFS), Ruxolitinib (RUX) 15 mg was added to the treatment with AZA+VEN as a result of the recent sharp increase in spleen size (197 × 78 mm) and abdominal discomfort. As a result of the treatment, the patient's spleen size decreased, abdominal discomfort disappeared. During the examination of the patient, a complete blood count and histological examination of the bone marrow were performed. To confirm myelofibrosis, reticulin stroma examination was performed using the Gomori method, and first- and second-degree fibrosis (M1–MF2) was detected (scale 0–3). In assessment with the Dynamic International Prognostic Scoring System (DIPSS)-2 points-intermediate-1 risk group was formed. The patient's complaints were assessed with Myeloproliferative Neoplasm Symptom Assessment Form Total Symptom Score (MPN-SAF-TSS). Molecular-genetic examination of the JAK2V617F gene was performed using the real-time PCR method of peripheral blood. Spleen size was assessed with USS. AZA was prescribed subcutaneously at a dose of 100 mg for 7 days per course. 6 courses have been conducted so far. VEN was increased according to the scheme and prescribed at a dose of 400 mg; depending on cytopenia's, the dose was reduced by 200 mg, and the number of days of administration varied from 28 to 14 days. RUX was prescribed at a dose of 15 mg daily. **Results:** After transformation of PMF to BP, the patient did not achieve remission despite 2 courses of low dose cytosar treatment. After treatment with AZA+VEN, the patient achieved MLFS. After some time, due to the growth of the spleen, RUX was added to the AZA+VEN treatment protocol, and the spleen's size decreased. **Conclusion:** The use of the AZA+VEN protocol was effective in BP-MF. The subsequent addition of RUX to the treatment further increased the effectiveness of the treatment and led to an improvement in the patient's general condition and a decrease in complaints.

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Adult Hematology Abstract Categories

Multiple myeloma

OP 12_Case report

PROGNOSTIC IMPLICATIONS OF HIGH-RISK GENETIC MUTATIONS IN MULTIPLE MYELOMA PATIENTS UNDERGOING SECOND AUTOLOGOUS STEM CELL TRANSPLANT

Taner Tan, Ahmet Umur Topcu, Ümit Barbaros Üre, Emre Osmanbasoglu, Sinem Civriz Bozdağ, Olga Meltem Akay

Koç University Hospital, Istambu, Turquia

Objective: High-risk genetic mutations significantly influence prognosis in multiple myeloma. Although autologous Hematopoietic Stem Cell Transplantation (auto-HSCT) is a cornerstone of multiple myeloma treatment, the prognostic impact of genetic abnormalities in patients undergoing a second auto-HSCT warrants further investigation. This study aims to evaluate the prognostic significance of high-risk genetic mutations in multiple myeloma patients undergoing a second auto-HSCT. **Methodology:** This retrospective analysis evaluated 26 multiple myeloma patients who underwent a second auto-HSCT between May 5, 2017, and December 10, 2024. Detailed analysis was conducted on 19 patients with available pre-transplant Fluorescence In Situ Hybridization (FISH) data. Among these, 9 patients underwent tandem transplantation, and 10 underwent a non-tandem second auto-HSCT. Prognostic analyses focused on genetic abnormalities detected by FISH. **Results:** The analyzed cohort included 10 males (52.6%) and 9 females (47.4%), with a mean age of 56.79-years (SD = 10.39, range 34–69). Median follow-up post-second transplantation was 31-months (IQR 18–54). Median intervals between two transplantations were 16-months (IQR 4–72.5) overall and 62-months (IQR 31–93) excluding tandem cases. High-risk genetic mutations were detected in 11 of 19 analyzed patients (57.9%): deletion 17p and amplification 1q (each 26.3%), t(4;14) (15.8%), deletion 1p (10.5%), and t(14;16) (5.3%). Patients with high-risk mutations