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Objective: Anemia is a common complication in patients with Chronic Kidney Disease (CKD), particularly in those not receiving dialysis. Roxadustat, a Hypoxia-Inducible Factor Prolyl Hydroxylase Inhibitor (HIF-PHI), has been investigated as a therapeutic option for anemia management in this population. This study aimed to evaluate the efficacy of Roxadustat compared to control interventions in Non-Dialysis-Dependent CKD (NDD-CKD) patients. **Methodology:** A comprehensive literature search was conducted in Cochrane CENTRAL, Ovid Medline, PubMed, and Web of Science up to December 14, 2024. Randomized Controlled Trials (RCTs) directly comparing Roxadustat with a control group were included. Data were pooled using an inverse variance-weighted random-effects model. The primary efficacy outcome was the change in Hemoglobin (Hb) levels at weeks 24–28 and during follow-up. Subgroup analyses were performed based on the type of control intervention (Erythropoiesis-Stimulating Agents [ESAs] vs. placebo) and prior ESA use. **Results:** A total of six RCTs, including 5,330 patients, from 520 unique records from the databases were included. Roxadustat significantly increased Hb levels during follow-up compared to the control group (Mean Difference [MD = 1.21 g/dL], 95% confidence interval [95% CI 0.45 to 1.97], $I^2 = 99\%$, $p = 0.0017$). However, at weeks 24–28, the increase in Hb levels was not statistically significant (MD = 0.86 g/dL, 95% CI -0.11 to 1.83, $I^2 = 99.4\%$, $p = 0.0833$). Iron-related parameters showed mixed results. Roxadustat was associated with a significant reduction in ferritin levels (MD = -38.54 ng/mL, 95% CI -68.21 to -8.87, $I^2 = 84.1\%$, $p = 0.0109$). Conversely, Total Iron-Binding Capacity (TIBC) was significantly increased with Roxadustat treatment (MD = 20.33 μg/dL, 95% CI 1.15 to 39.51, $I^2 = 98.5\%$, $p = 0.0377$). No significant difference was observed in serum iron (MD = 3.1 μg/dL, 95% CI -0.39 to 6.6, $I^2 = 93.1\%$, $p = 0.0820$) and Transferrin Saturation (TSAT) levels (MD = -1.08%, 95% CI -2.42 to 0.26, $I^2 = 40.1\%$, $p = 0.1151$) between the two groups. Subgroup analyses revealed that in placebo-controlled trials, Roxadustat significantly increased Hb levels at both weeks 24–28 and during follow-up. However, in trials comparing Roxadustat with ESAs, the changes in Hb levels were not significant at either time point. **Conclusion:** Roxadustat reduced ferritin but increased TIBC without significantly affecting free iron and TSAT levels compared to the control group in patients with NDD-CKD.

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Adult Hematology Abstract Categories

Myeloproliferative Neoplasms

OP 10

Genetic profile of primary myelofibrosis patients in Azerbaijan

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Objective: Primary myelofibrosis is a clonal myeloproliferative neoplasm characterized by atypical myeloid proliferation and significant symptom burden. Activation of the Jak-STAT signaling pathway plays a central role in the pathogenesis of this disease. Approximately 90% of patients have one of three genetic mutations: Jak2V17F, CALR and MPL. The Jak2V617F mutation is the most common mutation and has been found in 60%–65% of patients. Last year in SOHO 2024 annual meeting we first demonstrated genetic mutations of primary myelofibrosis patients in Azerbaijan. However, in our study only a small number of patients underwent genetic testing. Here we have updated the data of our cohort. The main goal of our study was to know the genetic profile of primary myelofibrosis patients in Azerbaijan. **Methodology:** We retrospectively analyzed 123 patients with primary myelofibrosis who underwent genetic testing. We created 2 groups according to JAK2 levels. Group comparability was assessed by comparing baseline demographics and follow-up time between groups. Normality and heteroscedasticity of continuous data were assessed using the Shapiro-Wilk and Levene tests, respectively. Continuous outcomes were compared using unpaired Student *t*-test, Welch *t*-test or Mann-Whitney *U* test, depending on the data distribution. Discrete outcomes were compared using Chi-Squared or Fisher's exact test, respectively. The alpha risk was set at 5% and two-tailed tests were used. **Results:** A total of 123 patients underwent genetic testing. Jak2V617F was positive in 91 (74%), CALR was positive in 3 (2.4%), MPL was positive in 1 (0.8%) patient. 32 (26%) patients were Jak2 negative. The median allele burden was 68.21% (IQR = 46.16). Median age was 58.5-years, 58 (47.2%) patients were male. We separated patients to groups according to Jak2 mutations and compared their clinical laboratory characteristics (Table 1). There was no difference between two groups according to IPSS: Low – 27 (31.03%), INT1 – 42 (48.28%), INT2 – 17 (19.54%), High – 1 (1.15%) in Jak2 positive (n = 87) vs. Negative (n = 31) Low – 11 (35.48%), INT1 – 12 (38.71%), INT2 – 7 (22.58%), High – 1 (3.23%). Jak2V617F positivity was significantly associated

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

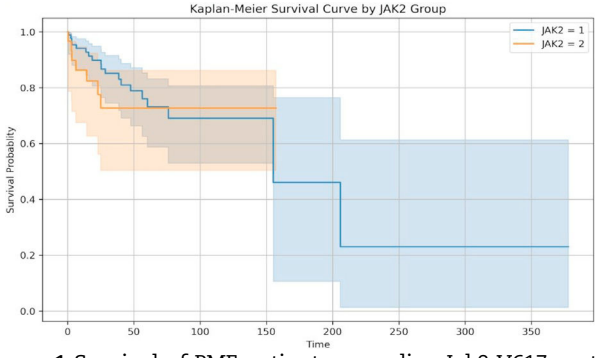

Figure 1 Survival of PMF patients according Jak2 V617 mutational status.

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OP 11_Case report

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

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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