Objective: Polycythemia vera (PV) is a chronic myeloproliferative neoplasm characterized by an increase in red blood cell mass. Thrombotic complications are the main cause of morbidity and mortality in PV. Elevated hematocrit and increased blood viscosity are crucial risk factors for thrombus formation. The aim of our analysis is to evaluate the biochemical alterations in red blood cells (RBCs) and the hemoglobin structure in patients with PV that may be associated with thrombotic complications. **Methodology:** Blood samples were taken from 20 PV patients and 16 healthy individuals. The isolated RBCs were examined using Raman spectroscopy. **Results:** We found a larger contribution of ferrous heme iron, which is a molecular state typical for deoxyhemoglobin in PV samples compared to the control samples. Furthermore, a significant increase in the Fe II/Fe III ratio in PV samples was correlated with a higher hematocrit (Hct) to hemoglobin (Hgb) ratio. A positive trend between a higher Fe II/ Fe III ratio and a higher RDW-SD and RDW-CV was observed in PV samples. In RBCs collected from PV patients we observed a less stable hemoglobin structure. **Conclusion:** Higher values of RDW-SD and RDW-CV may reflect a higher Fe II/ Fe III and be a simple indicator of biochemical alterations in RBCs. A higher Hct/ Hgb ratio could indicate higher clonal myeloproliferative potential and be associated with shorter time to thrombosis in patients with PV. Our future analysis will focus on correlating the above observations with the prothrombotic activity to demonstrate a possible link between the biochemical alterations of RBCs and the thrombotic complications in PV.

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OP 03

SECONDARY SOLID CANCER FREQUENCY AND RISK FACTORS IN PHILADELPHIA- NEGATIVE CHRONIC MYELOPROLIFERATIVE NEOPLASMS

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Objective: Philadelphia chromosome-negative myeloproliferative neoplasms (Ph- MPNs) are characterized by clonal myeloproliferation and somatic mutations. Major complications of Ph-MPNs are thrombosis, bleeding, transformation to myelofibrosis and leukemia. One important concern in the course of Ph-MPNs is risk of development of secondary solid cancers (SSC). In a large cohort of Turkish Ph-MPN patients, we aimed to determine the types and frequencies of SSC, to identify risk factors for SSC including role of cytoreductive therapies and to study impact of SSC on survival in Ph-MPNs. **Methodology:** 1013 patients diagnosed with Ph-MPN from 1995 and 2022 under follow up at adult hematology sections of Istanbul Bakırköy Dr Sadi Konuk Hospital and Istanbul University Medical Faculty were included in this retrospective study. **Results:** Of the 1013 Ph- MPN patients enrolled in our study, 65, 46 and 37 patients were diagnosed with essential thrombocythemia (ET), polycythemia vera (PV) and primary myelofibrosis (PMF), respectively. Patient clinical and laboratory characteristics are summarized in Table 1. Sixty-seven patients (6.6%) developed SSC, predominantly carcinoma (64.2%), non-melanoma skin cancer (23.9%), sarcoma (4.5%), and melanoma (3%). Median time to SSC diagnosis was 80.03 ± 60.5 months with no significant difference among Ph-MPN subtypes. Compared to patients with no diagnosis of SSC, patients with SSC were older at time of Ph- MPN diagnosis (63 vs. 54 years; $p<0.001$) and included a higher proportion of males ($p=0.025$). Ph- MPN patients with SSC and without SSC showed no significant difference for complete blood count parameters, spleen size, Ph-MPN diagnosis groups, driver mutation frequencies and follow-up time. Arterial thrombosis frequency was higher in patients with SSC (37.3% vs. 25.3%; $p=0.030$). SSC rates were 5.7% in patients not exposed to cytoreductive treatment and 5.3%, 4% and 2.1% with exposure to ruxolitinib, anagrelide, and interferon (IFN), respectively. A trend toward lower SSC rates was noted with IFN therapy (3% vs. 97%; $p=0.066$). SSC incidence was significantly higher in patients exposed to hydroxyurea (HU) as first-line monotherapy compared to other treatment groups (7.8% vs. 4.6%; $p=0.046$). Median OS in patients with SSC and patients with no diagnosis of SSC group were 273 months and 195 months, respectively. PV patients, who developed SSC, had significantly worse median OS compared to PV patients without SSC (Figure-1). **Conclusion:** The strengths of our study are that it enrolls a larger patient population, includes PV, ET and PMF subgroups, separately examines development of SSC after MPN, has a long follow-up period and has multicenter design. In MPN patients, malignancy screening gains more importance for those aged ≥65 and males. Our study evaluated with data from previous studies suggest that increased risk of developing SSC in MPN patients may be associated with cytoreductive therapy. Further studies with more pateints are needed

to determine whether Ph- MPN patients are predisposed to development of SSC independent of cytoreductive therapy, to better assess risk of HU or RUX in promoting SSC development in MPNs, and to elucidate the potential protective effect of IFN.

Table-1 Ph- MPN clinical and laboratory characteristics

	MPN (n=1013)	PV (n=380)	ET(n=419)	PMF(n=214)	p**	PV vs ET*	PV vs PMF*	ET vs PMF*
Gender Female, n (%) Male, n (%)	497 (49.1%) 516 (50.9%)	122 (32.1%) 258 (67.9%)	266 (63.5%) 153 (36.5%)	109 (50.9%) 105 (49.1%)	<0.001	<0.001	<0.001	0.002
Age at MPN diagnosis, median (range) <65, n (%) ≥65, n (%)	54 (12-88) 736 (72.7%) 277 (27.3%)	55 (17-84) 284 (74.7%) 96 (25.3%)	51 (12-88) 312 (74.5%) 107 (25.5%)	57.5 (21-84) 140 (65.4%) 74 (34.6%)	<0.001 0.028	0.029 0.926	0.008 0.016	<0.001 0.017
JAK2V617F n (%)	730 (72.1%)	305 (80.3%)	269 (64.2%)	156 (72.9%)	<0.001	<0.001	0.039	0.019
CALR n (%)	71 (7%)	.	58 (13.8%)	13 (6.1%)	.	.	.	0.003
MPL n (%)	4 (0.4%)	.	3 (0.7%)	1 (0.4%)	.	.	.	1.000
Triple negative n (%)	136 (13.4%)	.	90 (21.5%)	46 (21.5%)	.	.	.	0.996
WBC at MPN diagnosis, median (range)	10400 (2300-94000)	10795 (2510-34300)	9900 (4200-51400)	11350 (2300-94000)	<0.001	<0.001	<0.001	<0.001
HB at MPN diagnosis, median (range)	14.7 (5.5-24.5)	17.8 (11.4-24.5)	13.6 (6.7-17.1)	11.4 (5.5-19.5)	<0.001	<0.001	<0.001	<0.001
HCT at MPN diagnosis, median (range)	44.5 (14-85)	54 (36-85)	41 (21-55.5)	35.4 (14-62.7)	<0.001	<0.001	<0.001	<0.001
PLT at MPN diagnosis, median (range)	636000 (28000-2786000)	(40600-1818000)	853000 (110000-2786000)	425500 (28000-230	<0.001	<0.001	0.204	<0.001
Spleen size at MPN diagnosis, median (range)	120 (70-340)	120 (87-260)	120 (75-301)	178 (70-340)	<0.001	0.113	<0.001	<0.001
CV Risk n(%)	716(70.7%)	300(78.9%)	278(66.3%)	138(64.5%)	<0.001	<0.001	<0.001	0.640
Thrombosis, n (%) Arterial, n (%) Venous, n (%)	356 (35.1%) 264(25.1%) 92(12.3%)	144(37.9%) 110(28.9%) 42(11.1%)	138(32.9%) 107(25.2%) 48(11.5%)	74(34.6%) 47(22%) 34(15.9%)	0.335 0.168 0.184	0.143 0.279 0.857	0.421 0.064 0.090	0.678 0.321 0.116
Cytoreductive Therapy, n (%) Hydroxyurea, n (%) IFN, n (%) RUX, n (%)	871(86%) 831(82%) 94(9.3%) 95(9.4%)	314(82.6%) 311(81.8%) 16(4.2%) 15(3.9%)	355(84.7%) 327(78%) 59(14.1%) 5(1.2%)	202(94.4%) 193(90.2%) 19(8.9%) 75(35%)	<0.001 <0.001 <0.001 <0.001	0.425 0.181 <0.001	<0.001 0.006 0.02 <0.001	<0.001 <0.001 0.06 <0.001
Secondary Solid Cancer n (%)	67 (6.6%)	31 (8.4%)	26 (6.2%)	10 (4.7%)	0.236	0.284	0.108	0.431

Table-2 Clinical and laboratory characteristics of patients with secondary solid cancer

	SSC (n=67)	Non-SSC (n=946)	P.
Gender Female n (%) Male n (%)	24 (35.8%) 43 (64.2%)	473 (50.0%) 473 (50.0%)	0.025
Age at MPN diagnosis, median (range) <65 n (%) ≥65 n (%)	63 (37-78) 24 (35.8%) 43 (64.2%)	54 (12-88) 24 (35.8%) 43 (64.2%)	<0.001 0.001
WBC at MPN diagnosis, median (range)	10160 (3900-57260)	10400 (2300-94000)	0.457
HB at MPN diagnosis, median (range)	15.6 (5,8-21)	14.6 (5.5-24.5)	0.734
HCT at MPN diagnosis, median (range)	45.12 (19-69.5)	44,40 (14-85)	0.882
PLT at MPN diagnosis, median (range)	621000 (80000-2786000)	645500 (28000-2631000)	0.803
Spleen Size (mm) at MPN diagnosis, median (range)	120 (102-320)	120 (70-340)	0.658
Diagnostic Group PV n (%) ET n (%) PMF n (%)	31 (46.2%) 26 (38.8%) 10 (14.9%)	349 (36.9%) 393 (41.5%) 204 (21.6%)	0.236
Driver mutation JAK n (%) CALR n (%) MPL n (%) Triple Negative n (%)	49 (73.1%) 7 (10.4%) 1 (1.6%) 10 (14.9%)	681 (84.9%) 65 (8.1%) 3 (0.4%) 53 (6.6%)	0.201
Thrombosis n (%) Arterial n (%) Venous n (%)	30 (44.8%) 25 (37.3%) 6 (9.0%)	326(34.5%) 239 (25.3%) 118 (12.5%)	0.069 0.03 0.396
Cytoreductive Therapy None Cytoreductive Therapy Hydroxyurea Hydroxyurea monotherapy Interferon Therapy Interferon monotherapy Ruxolitinib Ruxolitinib monotherapy Anagrelide Anagrelide monotherapy	8(5.7%) 58(7.0%) 49 (7.8 %) 2 (2.1%) 1 (4.5 %) 5 (5.3%) 0 (0.0 %) 4 (4.0%) 0 (0.0 %)	133 (94.3%) 773 (93.0%) 576 (92.2%) 92 (97.9%) 21 (95.5%) 90 (94.7%) 7 (100.0%) 96 (96.0%) 4 (100.0%)	0.628 0.317 0.046 0.066 1,000 0.578 1,000 0.268 1,000

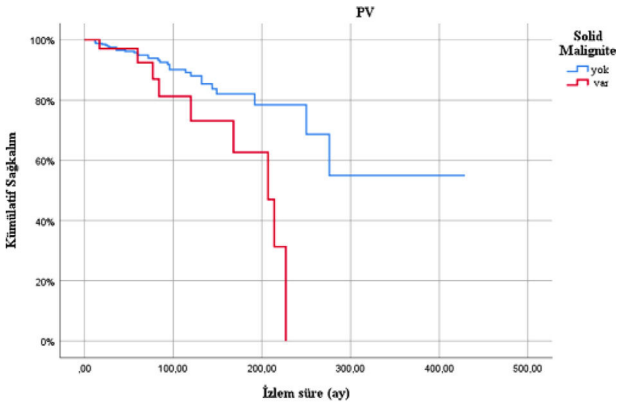

Figure-1 Overall Survival PV patients

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OP 04

EFFECT OF HEREDITARY THROMBOPHILIA ON ARTERIAL THROMBOSIS

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Objective: Screening for hereditary thrombophilia is recommended for venous thrombosis, but there is conflicting information about the causal relation with arterial thrombosis. In this study, in order to clarify these conflicting results and recommendations, it was aimed to determine whether there is a relation between arterial thrombosis and hereditary thrombophilia tests, to determine whether the treatment plan changes according to the test results of patients with hereditary thrombophilia panel, and t **Methodology:** In this single-centre, non-intervention, retrospective cohort study, 200 patients over the age of 18 who were performed hereditary thrombophilia tests by various clinics between 12/02/2019 and 01/07/2022 were included. The patients had no history of disease predisposing to thrombosis, no rheumatological disease, negative antiphospholipid antibodies, and arterial thrombosis. As a control group, 50 patients without arterial and venous thrombosis were included. **Results:** When the