J Surg Med. 2023;7(9):623-627.
DOI: 10.28982/josam.7979

Research article

Journal of Surgery and Medicine
e-ISSN: 2602-2079
https://jsurgmed.com/

The prevalence and impact of sarcopenia in myeloproliferative neoplasms

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Ethics Committee Approval
The study was approved by the Clinical Research Ethics Committee of Dr. Lütfi Kırdar State Hospital (decision date: December 29, 2021, decision number: 2021/514/216/5).
All procedures in this study involving human participants were performed in accordance with the 1964 Helsinki Declaration and its later amendments.

Conflict of Interest
No conflict of interest was declared by the authors.

Financial Disclosure
The authors declared that this study has received no financial support.

Published
2023 September 29

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Abstract

Background/Aim: Rapid identification of patients with myeloproliferative neoplasms (MPNs) is crucial for clinical decision-making and healthcare management. Sarcopenia is characterized by muscle loss and increases the risks for adverse outcomes; there is limited information in the literature regarding possible links between sarcopenia and MPNs. This study evaluated the frequency of sarcopenia in patients with MPNs and investigated whether biochemical or clinical features were associated with the development of sarcopenia.

Methods: Fifty-six BCR-ABL1-negative patients were included in this randomized prospective cohort research study. Muscle strength was measured using a handgrip dynamometer. Muscle mass was evaluated using a bioelectrical-impedance analyzer, and physical performance was evaluated via gait speed in a 6-minute walking test.

Results: The mean handgrip strength of the cohort was 27.7 kg, and 13 patients (23.2%) tested positive for low muscle strength. Mean muscle mass was found to be 7.58 (1.17) kg/m^2, and seven patients (12.5%) exhibited low muscle mass. Three patients (5.4%) had low muscle quality. Nine patients (16.1%) were diagnosed with probable sarcopenia, and four patients (7.1%) were diagnosed with severe sarcopenia. There was no difference between the groups in terms of clinical features (P>0.05), nutritional assessment (macro and micronutrients) (P=0.959), comorbidities (P=0.476), or laboratory measurements (P>0.05).

Conclusion: There was a high prevalence of sarcopenia among patients with MPNs, which indicates that periodic measurements of muscle strength, body composition and physical performance may contribute to the management of MPNs.

Keywords: sarcopenia, prevalence, myeloproliferative neoplasms, muscle strength, muscle mass

How to cite: Ipek Y, Tuna M. The prevalence and impact of sarcopenia in myeloproliferative neoplasms. J Surg Med. 2023;7(9):623-627.

Introduction

The term "myeloproliferative neoplasms" (MPNs) refers to a class of clonal malignancies formed from hematological stem cells that are distinguished by clonal proliferation of myeloid, erythroid, and megakaryocytic lineages in bone marrow [1]. There are various MPN subtypes that have been classified by the World Health Organization (WHO); the most important include primary myelofibrosis (PMF), secondary myelofibrosis (SMF), essential thrombocythemia (ET), and polycythemia vera (PV) [2]. Myeloproliferative neoplasms are considered diseases of older age and share a common pathophysiology with clonal hematopoiesis and chronic systemic inflammation. They have been linked to a four-fold greater risk of vascular events, a higher incidence of non-MPN solid cancers, and a shorter lifespan [3].

Muscle loss is a symptom of the disorder known as sarcopenia, which frequently develops as people age [4]. In addition to an elevated risk of unfavorable outcomes such as declines in both function and appearance, poor quality of life, socioeconomic burden, and death, sarcopenia is characterized by a generalized and progressive loss of muscle strength and function [5]. While primary sarcopenia is a common occurrence of aging, secondary sarcopenia may be associated with chronic diseases, inflammation, physical inactivity, malnutrition, and cancers. These factors have also been reported to be common in MPNs [6]. However, there is a dearth of information in the literature pertaining to the frequency of sarcopenia and risk factors in patients with MPNs.

This study focused on identifying the incidence of sarcopenia in individuals with MPNs and determining whether biochemical or clinical characteristics were linked to its development.

Materials and methods

The investigation was conducted at Kartal Lutfi Kirdar City Hospital in Istanbul, Turkey between January 2022 and March 2022. It was designed as a randomized prospective cohort research study. We included 56 individuals with BCR-ABL1-negative MPNs according to the WHO classification [2].

Patient selection and study design

We included patients aged 18 years and older. The study excluded participants with a history of pregnancy, rheumatological conditions, severe kidney illness, and other types of cancers besides MPNs. Patients with acute diseases, such as acute lymphoblastic leukemia (ALL) or acute myeloid leukemia (AML), and patients who had received an allogeneic stem cell transplant were also excluded from the study. The ethics committee of the Dr. Lütfi Kırdar State Hospital approved the study. The subjects furthermore provided verbal and written consent to participate in the study.

Diagnoses

A diagnosis of PV was made according to four criteria [7]: (1) a hemoglobin value above 16.5 g/dL in men or 16.0 g/dL in women or a hematocrit value above 49% in men or 48% in women or an expanded red cell mass; (2) hypercellularity in a bone marrow biopsy examination with pan-lineage growth with erythroid, granulocytic, and megakaryocytic proliferation; (3) the existence of JAK2 mutations (V617F or exon 12); and (4) a low serum erythropoietin (EPO) level. A diagnosis was made of when criteria (1), (2), and (3) were satisfied or when criteria (1), (2), and (4) were satisfied.

A diagnosis of ET was made according to five criteria [2]: (1) a platelet count above 450 x 109/L, (2) a bone marrow biopsy demonstrating proliferation largely of the megakaryocyte lineage with elevated numbers of enlarged hyper lobulated mature megakaryocytes, (3) a non-detection of other myeloid neoplasms or MPNs according to WHO criteria, (4) the presence of JAK2 or CALR or MPL mutations, and (5) the absence of evidence for reactive thrombocytosis. A diagnosis is made when criteria (1), (2), (3), and (4) were satisfied or when criteria (1), (2), (3), and (5) were satisfied. Primary and secondary myelofibrosis were also diagnosed according to the 2016 WHO criteria [2].

Data collection

Clinical and demographic characteristics were obtained from the patients' files. To evaluate the nutritional status of the patients, a mini-nutritional assessment (MNA) was performed. A total MNA score less than 8, between 8 and 11, and above 11 defined malnutrition, a risk of malnutrition, and the absence of malnutrition, respectively [8]. Sarcopenia was assessed in all cases. In 2018, the revised European consensus recommended three primary characteristics for the diagnosis of sarcopenia: low muscular strength, weak muscle quantity or quality, and subpar physical performance [4]. A digital hand grip dynamometer (Takei TKK 5401 model, Takei Scientific Instruments Co. Ltd, Tokyo, Japan) was used to quantitatively measure muscle strength. Values below 16 kg for females and 27 kg for males were considered to correspond to low muscle strength. We assessed physical performance using the typical gait speed for a 6-minute walk; a gait speed below 0.8 m/s was considered to indicate low muscle function. Appendicular skeletal muscle mass (ASMM) was estimated using a bioelectrical impedance analyzer (Tanita Body Composition Analyzer TBF-300 model, Tanita Co., Tokyo, Japan) using the Sergi equation, which was modified by the patient's body surface area to yield the ASMM index [4]. Low muscle mass was defined as an ASMM index below 5.5 kg/m^2 for women and below 7.5 kg/m^2 for men. Only when low muscle strength was found was sarcopenia considered likely. When reduced muscle strength, weak muscle quantity/quality, and subpar physical performance were observed, sarcopenia was deemed to be severe. The same doctor performed each measurement and evaluation, and strict guidelines were followed to guarantee the accuracy of the results.

Biochemical evaluation

After the patients fasted for 24 hours, blood was taken from the antecubital vein. Complete blood counts, including white blood count (WBC), platelet count, mean corpuscular volume (MCV), hemoglobin, and hematocrit, were analyzed using a Mindray BC-6800 autoanalyzer (Mindray Electronics Co. Ltd., Shenzhen, China). Creatinine, alanine aminotransferase (ALT), aspartate aminotransferase (AST), lactate dehydrogenase (LDH), total protein, albumin, and C-reactive protein (CRP) were studied using photometric methods with an Abbott Architect c8000 analyzer (Abbott Laboratories, Abbott Park, IL, USA). A chemiluminescent enzyme immunoassay was conducted using a UniCelDxI 800 (Beckman Coulter Inc., Brea,

CA, USA) to examine vitamin B12 and vitamin D levels. Real-time polymerase chain reaction and mutation studies of JAK2, CALR, JAK2 EXON12, and MPL were carried out at genetic laboratories. All of the blood samples were evaluated within an hour of being collected.

Statistical analysis

SPSS Statistics for Windows (version 26.0; IBM Corp., Armonk, NY, USA) was used to conduct the analyses. The Kolmogorov-Smirnov test was used to verify the normality of the data. Normally distributed data are presented as means and standard deviations; continuous variables are presented as medians (and minimum-maximum ranges). Categorical variables are presented as frequencies (%). We used the chi-square test to assess categorical variables. Depending on the number of groups, an independent samples t-test or one-way analysis of variance (ANOVA) were used to evaluate the normally distributed variables. Depending on the number of groups, non-normally distributed variables were evaluated using the Mann-Whitney U test or the Kruskal-Wallis test. The statistical significance threshold was set at 0.05.

Results

Table 1 lists the demographic information and clinical features of the patients. Thirty-three patients (58.9%) were male, and the mean age of the cohort was 54.0 years (standard deviation: 15.4 years). The average body mass index (BMI) of the patients was 26.9 kg/m^2 (standard deviation: 4.6 kg/m^2). Twenty-eight patients (50.0%) were diagnosed with ET, 18 patients (32.1%) were diagnosed with PV, 8 patients (14.3%) were diagnosed with primary MF, and 2 patients (3.6%) were diagnosed secondary (postpolycythemia) myelofibrosis. We noted JAK2 mutations in 26 patients (46.4%), and we noted CALR mutations in 3 patients (8.1%). Twenty patients (35.7%) were treated with acetylsalicylic acid, 29 patients (51.8%) were treated with acetylsalicylic acid+hydroxyurea, three patients (5.4%) were treated with hydroxyurea+anagrelide, and four patients (7.2%) were treated with ruxolinitib. The median MNA score was 14. Forty-nine participants walked for physical activity, and 11 patients (19.6%) were smokers. The most common comorbidities of the participants included diabetes (26.8%) and hypertension (33.9%).

The mean skeletal muscle index of the patients was 16.8 kg/m^2 (standard deviation: 3.1 kg/m^2). There was 16 participants (28.6%) in the low skeletal muscle index group. The mean handgrip strength of the cohort was 27.7 kg, and 13 patients (23.2%) exhibited low muscle strength. Muscle mass was calculated as ASMM/height^2 and the mean value was found to be 7.58 (1.17) kg/m^2. Seven patients (12.5%) exhibited low muscle mass. Muscle function was evaluated using a 6-minute walking time, and the mean value was found to be 447.1 seconds (standard deviation: 93.0 seconds). Three patients (5.4%) were assessed as having low muscle function. As a result, nine patients (16.1%) were diagnosed with probable sarcopenia, and four patients (7.1%) were diagnosed with severe sarcopenia. Table 2 lists the data related to the sarcopenia diagnoses.

Based on a diagnosis of sarcopenia, the study population was divided into groups (Table 1). Age (*P*=0.109), age at MPN diagnosis (*P*=0.080), BMI (*P*=0.396), cytogenetic

Table 1: Demographical and clinical characteristics of participants

Variables	Total	Probable-Confirmed Sarcopenia (n=13)	Non-Sarcopenia (n=43)	*P*-value
Age, years	54.0 (15.4)	61 (25-81)	53 (25-77)	0.109
Age of MPNs diagnosis, years	50.4 (15.3) (22-80)	59 (22-80)	49 (24-74)	0.080
Sex				
Male (n,%)	33 (58.9)	8 (61.5)	25 (58.1)	0.827
Female (n,%)	23 (41.1)	5 (38.5)	18 (41.9)	
BMI, kg/m^2	26.9 (4.6) (19.5-43)	26.6 (20.5-43)	26.9(19.5-40.6)	0.396
JAK2				
Negative, (n,%)	30 (53.6)	8 (61.5)	22 (51.2)	0.511
Positive, (n,%)	26 (46.4)	5 (38.5)	21 (48.8)	
CALR				
Negative, (n,%)	34 (91.9)	3 (75)	31 (93.9)	0.298
Positive, (n,%)	3 (8.1)	1 (25)	2 (6.1)	
Diagnosis				
Polycythemia vera (n,%)	18 (32.1)	8 (61.5)	10 (23.3)	0.071
E.Thrombocythemia (n,%)	28 (50.0)	4 (30.8)	24 (55.8)	
Primary MF, (n,%)	8 (14.3)	0 (0.0)	8 (18.6)	
Secondary MF, (n,%)	2 (3.6)	1 (7.7)	1 (2.3)	
Treatment				
Acetylsalicylic acid, (n,%)	20 (35.7)	3 (23.1)	17 (39.5)	0.491
Acetylsalicylic acid+ Hydroxyurea (n,%)	29 (51.8)	10 (76.9)	19 (44.2)	
Hydroxyurea + Anagrelide (n,%)	3 (5.4)	0 (0.0)	3 (7.0)	
Ruxolinitib (n,%)	4 (7.2)	0 (0.0)	4 (9.3)	
Mini-nutritional assessment				
10, (n,%)	1 (1.8)	0 (0.0)	1 (2.3)	0.959
11, (n,%)	1 (1.8)	0 (0.0)	1 (2.3)	
12, (n,%)	4 (7.1)	1 (7.7)	3 (7.0)	
13, (n,%)	7 (7.1)	2 (15.4)	5 (11.6)	
14, (n,%)	43 (76.8)	10 (76.9)	33 (76.7)	
Physical Activity				
Low physical activity / home-office, (n,%)	1 (1.8)	1 (7.7)	0 (0.0)	0.095
Walking, (n,%)	49 (87.5)	12 (92.3)	37 (86.0)	
Doing sport, (n,%)	6 (10.7)	0 (0.0)	6 (14.0)	
Smoking, (n,%)	11 (19.6)	1 (7.7)	10 (23.3)	0.426
Thrombosis/Emboli (n,%)	5 (8.9)	0 (0.0)	5 (11.6)	0.580
Co-morbidities				
Ischemic heart failure, n (%)	11 (19.6)	2 (15.4)	9 (20.9)	0.476
Hypertension, n (%)	19 (33.9)	6 (46.2)	13 (30.2)	
Diabetes mellitus, n (%)	15 (26.8)	5 (38.5)	10 (23.3)	
PAH, n (%)	3 (5.4)	0 (0.0)	3 (7.0)	
Hypothyroidism, n (%)	8 (14.3)	3 (23.1)	5 (11.6)	
HM, n (%)	13 (23.2)	3 (23.1)	10 (23.3)	

MPNs: Myeloproliferative neoplasms, BMI: Body mass index, MF: Myelofibrosis, E. Thrombocythemia: Essential Thrombocythemia, PAH: Pulmonary arterial hypertension, HM: Hepatomegaly, JAK2: Janus kinase 2, CALR: Calreticulin. Data are given as mean (standard deviation) or median (minimum - maximum) for continuous variables according to normality of distribution, and as frequency (percentage) for categorical variables

profile for *JAK2V617F (P*=0.511), cytogenetic profile for *CALR* (*P*=0.298), current medications (*P*=0.491), MNA score (*P*=0.959, smoking status (*P*=0.426), and the presence of comorbidities (*P*=0.476) did not significantly differ between the groups.

Table 2: Results of Sarcopenia Criteria based on revised European consensus of participants

	Values
Skeletal Muscle Index (kg/m^2)	16.8 (3.1)
Positive (n,%)	16 (28.6)
Negative (n,%)	40 (71.4)
Hand Grip strength, kg	27.7 (10.2)
Positive, (n,%)	13 (23,2)
Negative (n, %)	43 (76,8)
ASM	21.2 (4.7)
ASM/height^2	7.58 (1.17)
Positive, (n,%)	7 (12.5)
Negative, (n,%)	49 (87.5)
6-m walking time (s)	447.1 (93.0)
Positive, (n,%)	3 (5.4)
Negative, (n,%)	53 (94.6)
Diagnosis of sarcopenia	
Probable, (n,%)	9 (16.1)
Confirmed, (n,%)	4 (7.1)

ASM: Appendicular Skeletal Muscle Mass. Data are given as mean (standard deviation) or median (minimum - maximum) for continuous variables according to normality of distribution, and as frequency (percentage) for categorical variables

Data pertaining to the biochemical analyses of the participants are listed in Table 3. The mean results of the biochemical tests were within the normal physiological reference value for age and sex. Biochemical results such as vitamin D (*P*=0.528), vitamin B12 (*P*=0.489), creatinine (*P*=0.278), AST (*P*=0.465), ALT (*P*=0.156), LDH (*P*=0.756), total protein (*P*=0.281), albumin (*P*=0.167), CRP (*P*=0.426), WBC (*P*=0.318), hemoglobin value (*P*=0.826), platelet count (*P*=0.313), hematocrit ratio (*P*=0.930), and MCV (*P*=0.793) were similar between the groups in terms of probable/confirmed sarcopenia.

Table 3: Biochemical analysis of participants according to sarcopenia diagnosis

	Total	Probable-Confirmed Sarcopenia (n=13)	Non-Sarcopenia (n=43)	*P*-value
Vitamin D (ng/mL)	20.9 (13.0)	14.95 (8.9-39.6)	20.1 (5.8-59.7)	0.528
Vitamin B12 (pg/mL)	327.1 (111.2)	362 (205-471)	338 (108-595)	0.489
Creatinine (mg/dL)	0.83 (0.26)	0.87 (0.53-2.14)	0.79 (0.45-1.14)	0.278
AST (U/L)	19.5 (6.4)	18 (13-31)	20 (10-54)	0.465
ALT (U/L)	19.8 (11.1)	14 (9-29)	19 (5-78)	0.156
LDH (U/L)	269.1 (133.5)	221 (131-447)	231 (134-722)	0.756
Total protein (g/dL)	7.03 (0.47)	6.8 (5.9-7.6)	7 (5.8-7.9)	0.281
Albumin (mg/dL)	4.62 (0.25)	4.6 (4.2-4.9)	4.7 (4.1-5.2)	0.167
CRP (mg/L)	1.34 (0.92)	1 (0.32-3.56)	1.26 (0.11-3.29)	0.426
WBC (x10^6/L)	9014.8 (3616.2)	7420 (4440-17330)	8420 (3300-21200)	0.318
HGB (g/dL)	13.7 (1.6)	13.9 (11.9-15.9)	13.5 (10.5-17.1)	0.826
HCT (%)	43.1 (5.4)	42.9 (35.9-51)	43.2 (31.4-58.7)	0.930
MCV (fL)	92.7 (13.4)	86 (77.8-118.4)	90.4 (64.9-126.9)	0.793
PLT(x10^9/L)	503.5 (270.6)	426 (208-786)	450 (123-1285)	0.313

AST: Aspartate aminotransferase, ALT: Alanine aminotransferase, LDH: Lactate dehydrogenase, CRP: C-reactive protein, WBC: White blood cell, HGB: Hemoglobin, HCT: Hematocrit, MCV: Mean corpuscular volume, PLT: Platelet. Data are given as mean (standard deviation) or median (minimum - maximum) for continuous variables according to normality of distribution, and as frequency (percentage) for categorical variables

Discussion

The purpose of the study was to evaluate the incidence of sarcopenia in individuals with various MPN subtypes and to ascertain whether biochemical or clinical traits had an effect on the onset of the disease. The importance of identifying sarcopenia has been identified for treating aging patients and individuals with chronic illnesses [6]. In this study, sarcopenia was identified as being probable in 16.1% of patients with MPNs and severe in 7.1% of patients with MPNs. While 13 patients presented with low muscle strength, seven patients had weak muscle mass, and three patients exhibited weak muscle function. We found similar biochemical and clinical characteristics between the groups with and without sarcopenia.

Sarcopenia is a multifactorial disorder resulting from changes in endocrine function, suboptimal protein intake, activation of inflammatory response, reductions in physical activity, and reductions in the number of alpha motor units in the spinal cord [9]. The determinants leading to sarcopenia in cancer patients are primarily attributable to decreased food intake, side effects of medications, anorexia, and muscle disuse; previous reports have suggested complex interactions between glucose utilization, immune response, inflammation, energy metabolism, and body muscle mass in oncological patients [6,10]. Sarcopenia can also lead to an inability to tolerate optimal cancer managements, resulting in postoperative complications and high chemotherapy toxicity [11]. Furthermore, anticancer therapy seriously influences body composition by causing muscle wasting, cachexia, and loss of bone mass and adipose tissue [12]. Among the elderly, the prevalence of sarcopenia has been reported to be 10% [13]. In hospitalized patients the prevalence is 14.7%, in nursing home residents the prevalence is 59%, in community residents the prevalence ranges from 12.9–40.4%, and in cancer patients the prevalence is 38.6% [13]. The prevalence of sarcopenia was found to be 11.8% among elderly Turkish individuals living in rural areas; it was 21.6% in among Turkish individuals living in urban areas [14]. A meta-analysis of 1578 patients with different hematological malignancies revealed that 39.1% of patients with lower overall survival were sarcopenic [15]. Burkat et al. [16] studied 109 patients with aggressive B cell lymphoma and found that male patients with sarcopenia, unlike female patients, had decreased progression-free survival and overall survival. That finding suggests a role of cachexia in sex-specific prognostic use. The same authors also found that 65 of patients (60%) were identified as sarcopenic; there was furthermore a relationship between cachexia and low serum marker levels of in-glucose utilization (as insulin-like growth factor-binding protein 6), inflammation (as lymphotoxin-like inducible protein), and energy metabolism (as leptin).

A meta-analysis of 1752 patients with hematological malignancies who were in remission showed that sarcopenia was linked to a lower overall survival rate among individuals who had undergone hematopoietic stem cell transplant [17]. Kamiya et al. [18] reported that the prevalence of sarcopenia based on the low mass strength, weak muscle function, and decreased muscle mass was 36% in women and 24% in men among 56 elderly patients with hematological malignancies. In this study, we consistently demonstrated high sarcopenia frequencies: 16.1% of patients had probable sarcopenia, and 7.1% of patients had severe sarcopenia. This finding may be related with the fact that patients with MPNs often suffer from low appetite and weight loss from immunosuppressive drugs, inactivity due to fatigue, and tumor-related inflammation resulting in catabolism and high protein consumption. Our results support the hypothesis that sarcopenia contributes to clinical adverse outcomes of MPNs by reducing quality of life and limiting functionality. Periodic measurements of body composition, muscle strength and performance may contribute to the primary prevention and management of the disease in patients with MPNs.

Sarcopenia is a common disease worldwide, and it is especially prevalent in elderly populations [11,14]. It is also associated with sex, disease status and comorbidities, and the

type and duration of treatment (e.g., high-dose chemotherapy and steroids) [17]. Kurose et al. [19] showed that age, obesity, hypertension, the frequency of daily conversation, and malnutrition were independent predictors of sarcopenia. Wu et al. [20] showed that risk factors for sarcopenia included age, sex, smoking status, and BMI. However, we could not find a relationship between sarcopenia and clinical and biochemical features in patients diagnosed with MPN. This is likely due to the heterogeneity of MPN subtypes with small sample size. Variations in the clinical manifestations of MPNs and heterogeneity in drug use may have also contributed to the disparate results. To clarify the specific association between clinical variables and sarcopenia and to support our findings, additional large-scale prospective investigations that consider various patient clinical statuses and MPN subtypes are required.

Limitation

The study has a number of limitations. First off, it was conducted at a single institution with a cross-sectional design. The range of MPN subtypes considered was furthermore small, and only a limited number of patients were enrolled in the study. Finally, there was an absence of information about sarcopenia or changes in body composition prior to the development of MPNs.

Conclusion

We found that among individuals with MPNs there was a pronounced rate of sarcopenia. This finding suggests that sarcopenia may be important for clinical outcomes and management of MPNs. Our findings highlight the importance of patient-specific strategies in order to reduce symptoms, increase overall survival rates, and enhance quality of life.

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