Splenic infarction as a rare cause of left upper abdominal quadrant pain in an elderly woman

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ABSTRACT:

Polycythemia vera (PV) is a chronic myeloproliferative neoplasm resulting in increased red blood cell mass. It can lead to various symptoms caused by blood hyperviscosity and an elevated number of red blood cells. Both thrombosis and bleeding complications can occur in PV. Splenic infarction is a rare complication with an unknown incidence usually presenting with pain in the upper left quadrant of the abdomen. The treatment is usually conservative, but there are some complications requiring surgery. We describe an elderly female patient who presented to the emergency department with left costal margin pain which was caused by splenic infarction.

KEYWORDS: Splenic Infarction; Polycythemia vera; Abdominal Pain

SAŽETAK:

Infarkt slezene kao rijedak uzrok boli u lijevom gornjem abdominalnom kvadrantu u starije žene Polycythemia vera (PV) je kronična mijeloproliferativna neoplazma koja rezultira povećanom masom crvenih krvnih stanica. Može dovesti do različitih simptoma uzrokovanih hiperviskoznošću krvi i povećanim brojem crvenih krvnih stanica. U PV se mogu pojaviti i tromboza i komplikacije krvarenja. Infarkt slezene rijetka je komplikacija s nepoznatom učestalošću koja se obično manifestira bolom u gornjem lijevom kvadrantu abdomena. Liječenje je obično konzervativno, ali postoje neke komplikacije koje zahtijevaju operaciju. Opisujemo stariju pacijenticu koja se javila u Hitnu s bolom lijevog rebra uzrokovanom infarktom slezene.

KLJUČNE RIJEČI: infarkt slezene; Policitemija vera; Bol u trbuhu

INTRODUCTION

Polycythemia vera (PV) is a chronic myeloproliferative neoplasm resulting in increased red blood cell mass (1). Symptoms of the disease are non-specific and related to blood hyperviscosity and to elevation in the number of blood cellular elements. Patients suffering from PV are at risk of both arterial and venous thrombosis, but also bleeding complications. The major risk factors linked to thrombosis are age and previous medical history of thrombosis. Other significant contributing factors are increased haematocrit and leukocytosis (2). Splenic infarction is a rare consequence of PV with an unknown incidence.

CASE REPORT

A 70-year-old woman presented to the Emergency Department with sharp left costal margin pain that is constant, radiates to the back, worsens on manual palpation, coughing, and deep inspiration. Pain is described as 10/10 on the visual-analogue scale (VAS). She has been taking non-steroid anti-inflammation medications in high doses for weeks without resolution of symptoms.

She had been vaccinated with a booster dose of Pfizer 3 weeks prior. Her previous medical history includes polycythemia vera with JAK2 mutation, diabetes mellitus, and arterial hypertension. In her chronic medical therapy she was using hydroxycarbamide, tramadol, ibuprofen, vidagliptin, metformin, gliclazide, acetylsalicylic acid, and pantoprazole. The physical exam was unremarkable except for pain under the left costal margin. Significant lab results were leukocytes 20 x 10°/L, haemoglobin 141 g/L, haematocrit 0,446, thrombocytes 10001 x 109/L, lactate-dehydrogenase 833 U/L, C- reactive protein 18 mg/L. Differential diagnoses in this patient include gastritis, pancreatitis, pulmonary embolism, pneumonia, nephrocolic and aortic dissection. Point-of-Care Ultrasound (POCUS) of the abdomen showed an inhomogeneous spleen with localized pain in that area (Fig. 1.). Suspected splenic infarction was confirmed with a computed tomography (CT) scan, showing multiple hypovascular zones (Fig. 2.). An accidental finding is a splenunculus, measuring 26 mm. She was admitted to the haematology department and was treated conservatively.

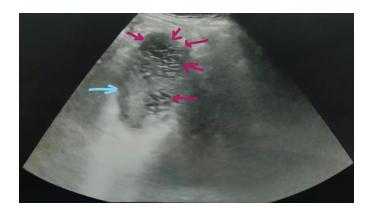


Figure 1. Point-of-Care Ultrasound (POCUS) of the abdomen showing an inhomogeneous spleen.



Figure 2. Computed tomography scan showing multiple hypovascular zones in the spleen.

Supplementary Table S1. Additional data about study participants

	DIAPHYSEAL FRACTURE GROUP (N=6)	METAPHYSEAL FRACTURE GROUP (N=10)	
Age in years mean (±SD)	43.17 (±13.92)	45.8 (±10.84)	
Male N (%)	3 (50%)	6 (60%)	
Female N (%)	3 (50%)	4 (40%)	
Surgical treatment N (%)	6 (100%)	6 (60%)	
Conservative treatment N (%)	0 (0%)		

Supplementary Table S2. Values of mean fold change and standard deviation of each analysed cytokine per subject (or subject group). Fold change was calculated from values of the control group.

	Diaphyseal fracture 1. timepoint	Diaphyseal fracture 2. timepoint	Metaphyse-al fracture 1. timepoint	Metaphyse-al fracture 2. timepoint
GCSF	1 ±0.03	0.98 ±0.05	0.75 ±0.01	0.79 ±0.07
GM-CSF	0.85 ±0.01	0.79 ±0.01	0.71 ±0.04	0.75 ±0
GRO (α, β, γ)	0.95 ±0.03	0.92 ±0	0.76 ±0	0.84 ±0.01
GRO-α	0.84 ±0.04	0.87 ±0	0.71 ±0.01	0.83 ±0.02
IL-1a	1.03 ±0.14	1.16 ±0.02	0.88 ±0.04	0.75 ±0.08
IL-2	1.1 ±0.15	1.01 ±0.08	0.84 ±0.03	0.8 ±0.11
IL-3	1.21 ±0.07	1.03 ±0.08	1.01 ±0.04	1.01 ±0.06
IL-5	1.15 ±0.04	1.06 ±0.01	0.78 ±0.01	0.83 ±0.01
IL-6	1.26 ±0.03	1.27 ±0.02	0.89 ±0.04	0.98 ±0.05
IL-7	1.04 ±0.05	1.12 ±0.09	0.8 ±0.04	0.85 ±0.06
IL-8	1.02 ±0.08	1.02 ±0.01	1.01 ±0	0.97 ±0.02
IL-10	0.89 ±0.03	0.93 ±0	0.78 ±0.01	0.81 ±0.01
IL-13	0.82 ±0.02	1.1 ±0.02	0.82 ±0.1	0.62 ±0.02
IL-15	1.01 ±0.08	1.15 ±0.09	0.85 ±0.04	0.72 ±0.08
IFN-γ	0.92 ±0.05	0.88 ±0.03	0.82 ±0.07	0.76 ±0.1
MCP-1	2.41 ±0.25	2.34 ±0.17	2.25 ±0.18	2.07 ±0.17
MCP-2	1.03 ±0.18	1.15 ±0.04	0.76 ±0.11	0.75 ±0.13
MCP-3	0.94 ±0.1	0.95 ±0.04	0.68 ±0.09	0.68 ±0.13
MIG	0.91 ±0.11	0.99 ±0.08	0.76 ±0.04	0.74 ±0.07
RANTES	1.63 ±0.03	1.8 ±0.01	1.59 ±0.05	1.63 ±0.03
TGF-β1	1.06 ±0.01	1.15 ±0.01	1.03 ±0.04	0.83 ±0.01
TNF-α	1.18 ±0.08	1.13 ±0.11	1 ±0.07	0.84 ±0.1
TNF-β	1.08 ±0.01	1.18 ±0.02	0.82 ±0.13	0.75 ±0.06

DISCUSSION

Polycythemia vera is a chronic myeloproliferative neoplasm characterized by augmented erythropoiesis. It can lead to thrombosis and thromboembolism, among other complications. The aforementioned complications occur in 39-41% of patients suffering from PV (1,3) and can occur years before a diagnosis of the disease. The most important risk factors for thromboembolism in patients with PV are age >65 years and previous history of thromboembolic events. The British Society for Haematology includes the stated age as a defining clinical feature of high-risk PV (4.5).

Leukocytosis and elevated CRP, which were present in our patient, are linked to a higher risk of thromboembolism in PV, however, the correlation with thrombocytosis is unclear and it might even increase the risk for bleeding in some cases (2). The primary endpoint of medical therapy in PV is to reduce the risk for thrombosis mostly by reducing HCT level. European Society for Medical Oncology Clinical Practice Guidelines suggest maintaining a target HCT of < 45% in patients with PV to reduce mortality and morbidity, which was achieved in our patient (5).

The type of thrombosis can be dependent on the patient sex. Tefferi et al observed that arterial thrombosis more commonly occurs in men than in women (18% vs 14%), and venous

thrombosis is more common in women than in men (9,3% vs 5,4%)(6). The most common manifestations of arterial events are acute coronary syndrome and stroke, while venous thrombosis most commonly manifests as deep vein thrombosis, splanchnic vein thrombosis, pulmonary embolism, and superficial venous thrombosis (7,8).

Splenic infarction, as a consequence of PV, is a rare complication with unknown incidence. The symptoms include left upper abdominal quadrant pain, left shoulder pain (Kehr sign), pleuritic chest pain, fever, and nausea. The preferred diagnostic method to diagnose splenic infarction is CT due to its ability to demonstrate any subtle parenchymal changes in the spleen (9). Haemodynamic instability can occur in the cases of subcapsular haemorrhage. Another possible complication is a spleen abscess. Such complications require surgical treatment, otherwise, splenic infarction is treated medically.

Regardless of the very low incidence of splenic infarction, a combination of abdominal pain and haematological disorder or hypercoagulable state should raise a concern about this diagnosis.

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