Multifactorial intestinal ischemia: a rare and undervalued diagnosis of abdominal pain

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ABSTRACT

A 71-year-old woman was admitted to the hospital after consuming food due to acute malnutrition and abdominal pain. Reduced patency of all jejunoileal branches and the superior mesenteric artery was observed in the abdominal computed tomography scan. Abdominal angina was caused by atherosclerosis, the frequent use of triptans for recurrent migraines, the *JAK2* mutation, and a high platelet count. The patient's complete parenteral nutrition was not spared, despite the development of a collateral circulation preventing fatal acute ischemia.

Introduction

Abdominal angina, caused by atherosclerotic disease of the mesenteric vessels, represents 1/1000 admissions for abdominal pain. Although rare, it must be taken into consideration in the diagnostic process, in particular, if abdominal pain occurs after eating.

Rapid diagnosis is essential, especially in elderly patients with multiple comorbidities that could complicate the diagnosis.[1]

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Case Report

A 71-year-old woman, (weight: 32 kg; height: 152 cm; body mass index: 13,85 kg/m^2) was hospitalized for severe protein-energy malnutrition due to abdominal pain and diarrhea alternated with stypsis, following food and liquid intake. Because of this, she had to repeatedly refer to an emergency room, obtaining only a diagnosis of psychosomatic problems.

On admission, the physical examination was irrelevant. Her medical history documented arterial hypertension, multidistrict atherosclerosis, colonic diverticulosis and frequent use of triptans for a recurrent migraine.

Hematic tests (Table 1) showed leucocytosis with neutrophilia, high platelet count, C-reactive protein, procalcitonin, lactate dehydrogenase, and fibrinogen values. Low levels of prealbumin, iron, transferrin, pseudocholinesterase, glycemia, and albumin testified to a high grade of malnutrition.

Extended stool cultures (Shigella, Salmonella, Yersinia, Campylobacter, E. Coli) and parasitological examination, rectal swab for carbapenem-resistant germs, Widal-Wright serodiagnosis and A, B, C hepatitis virus antibodies were all negative. Lipoprotein a, homocysteine, and lipid profile were normal. The autoimmunity panel and the thrombophilia screening were negative.

The supra-aortic trunk, bilateral carotid, and lower limb arterial and venous color Doppler ultrasounds showed non-hemodynamically significant atherosclerosis. The cardiac color Doppler ultrasound showed mild mitral insufficiency and aortic root dilation with normal left ventricular function.

The *JAK2* gene V617F mutation was found, thus explaining the high levels of platelets; for this reason, the hematologist consultant prescribed oncocarbide 500 mg 1 cp x 2/days.

Abdominal computed tomography (CT) scan showed: multiple diverticular formations and a faecaloma; ectasia (28 mm) and extensive atheromasia of the thoracoabdominal

aorta; atherosclerotic plaques at the emergence of the celiac tripod with reduced patency, filiform caliber, and retrograde flow. In addition, reduced patency of the superior mesenteric artery and all jejunoileal branches were detected. The left colonic artery appeared dilated and tortuous but normally patent; lastly, the subcapsular circulation of the liver was hypertrophic (Figure 1). Consequently, angina abdominis was confirmed. Considering the CT picture, double antiplatelet therapy with acetylsalicylic acid 100 mg and clopidogrel 75 mg was prescribed along with the already prescribed anticoagulant therapy with enoxaparin 2000 UI/day.

Due to severe malnutrition and inadequate intestinal function, the patient underwent total parenteral nutrition through a peripheral inserted central venous catheter.

Table 1. Laboratory values during hospitalization.

Analyte	Normal range	Assessed value					
		At entry	4th day	8th day	11th day	17th day	24th day
Hb (g/dl)	12.0-15.5	14.20	12.80	14.80	13.60	12.10	12.00
RBC (×10⁶/ul)	4.0-5.0	4.65	4.41	4.84	4.60	3.99	3.73
HCT (%)	35-48	43.90	38.50	44.70	40.90	37.00	34.70
MCV (fl)	80.0-97.0	94.40	87.30	92.30	88.90	92.70	93.00
PLT (×10³/ul)	150-450	633	667	799	735	531	296
WBC (×10³/ul)	4.5-11.0	13.20	11.26	15.50	13.41	8.62	7.88
NEUT (×10³/ul)	1.8-7.0	9.99	7.98	10.63	9.14	5.70	4.13
LINF (×10³/ul)	1.0-4.8	1.93	2.06	3.39	2.89	1.66	3.16
CRP (mg/dl)	0.00-0.5	13.90	2.82	0.69	1.23	0.30	-
PCT (ng/ml)	<0.09: negative	0.32	-	0.07	-	-	-
Glucose (mg/dl)	70-110	123	52	66	75	77	86
Cholesterol (mg/dl)	<190	-	117	121	143	130	134
Albumin (g/dl)	3.2-4.6	3.5	2.6	2.9	3.0	-	-
Prealbumin (g/l)	0.2-0.4	0.04	-	-	-	-	-
CHE (U/l)	5320-12,920	-		3351	3653	3120	3264

Hb, hemoglobin; RBC, red blood cell count; HCT, haematocrit; MCV, mean cellular volume; PLT, platelets; WBC, white blood cell count; NEUT, neutrophils; LINF, lymphocytes; CRP, C-reactive-protein; PCT, procalcitonin; CHE, cholinesterase.

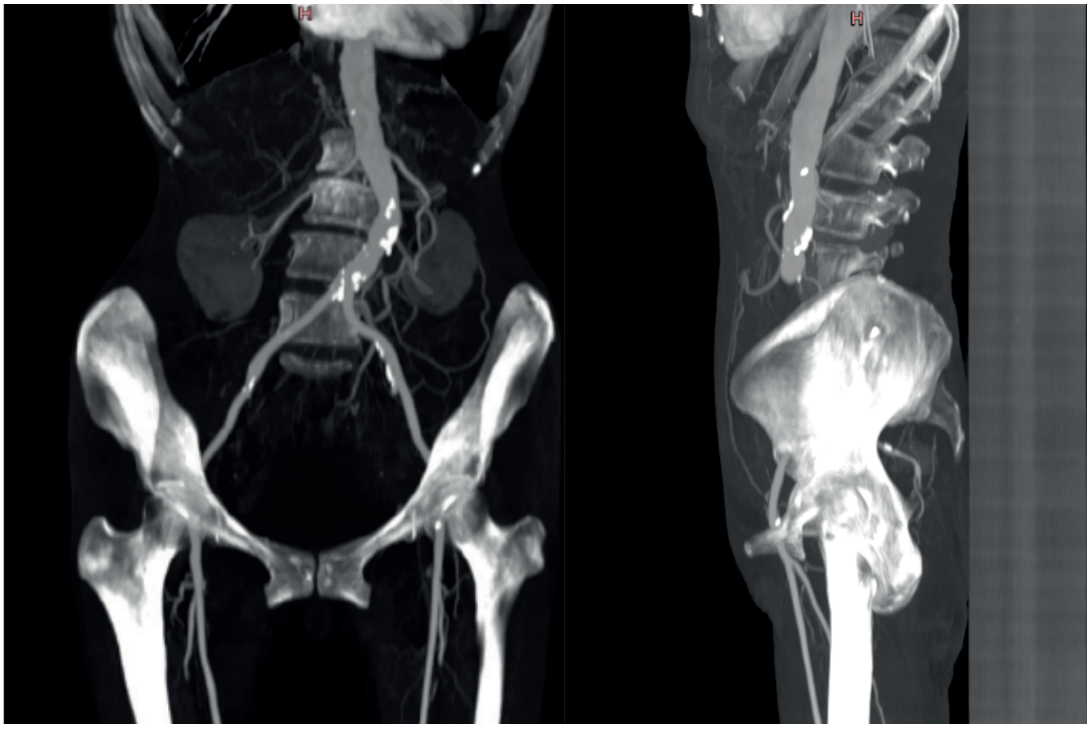

Figure 1. Patient's abdominal computed tomography scan. Frontal and sagittal view showing extensive atheromasia of the aorta; lack of opacification of the celiac tripod, superior and inferior mesenteric arteries.

Discussion

Mesenteric ischemia may be caused by embolic occlusion (45-50% of cases), thromboembolism of a previously stenotic vessel (20-35%), vessel dissection, or arteritis (5%). 90% of the causes of chronic mesenteric ischemia are due to atherosclerotic disease affecting the wall of the aorta, involving the proximal segments of the splanchnic vessels. Finally, 5-15% of cases of ischemia are caused by primary or idiopathic venous thrombosis, related thrombophilia, trauma, or local inflammation.[2]

The mesenteric circulation is supplied by three main vessels (celiac, superior and inferior mesenteric arteries) that branch off into a thick network guaranteeing organ perfusion even in the case of a main vessel occlusion.[3] Acute occlusion of the superior mesenteric artery can lead to severe ischemia. In chronic forms, the development of collateral and vicarious vessels can delay the onset of symptoms until sub- or total occlusion of two or more main vessels.

In elderly patients with pulmonary or heart failure, the physiological reserve is insufficient for the organ oxygen metabolic demand, leading to chronic vasodilation.[1] However, prolonged ischemia results in reflex vasoconstriction. Ischemia worsening can cause injury to the mucosa, submucosa, and intestinal barrier system, stimulating the release of inflammatory cytokines. These inflammatory mediators further worsen the vasospasm and extend the ischemic area to the entire intestinal wall. Without a timely intervention, the damage can lead to intestinal infarction, resulting fatal.

The abuse of vasoconstrictive drugs such as triptans, can contribute to the development of mesenteric ischemia. Triptans are anti-migraine vasoconstrictive drugs, acting on the 5-HT1 receptors of the brain and systemic circulation, determining vasoconstriction not only of the brain circulation, but also of other vascular beds, such as the mesenteric one, potentially causing ischemia. Therefore, their use should be avoided in patients with a predisposition to enteric ischemia.

Conclusions

In this case report, the abuse of triptans for migraine treatment, together with the presence of the *JAK2* gene mutation worsened the chronic intestinal ischemia.[4-6]

The collateral loops allowed the patient not to have acute ischemia, nonetheless, this did not save the patient from home parenteral nutrition.

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