The Cardiologist

ISSN: 2691-5685

Case Report

Intestinal Necrosis Due to Beta-Blocker Intoxication

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Abstract

Case report about lethal, acute intestinal necrosis, induced by low-flow during beta-blocker intoxication in a multimorbid patient with chronic arteriosclerotic occlusion of the coeliac trunk, diagnosed by CT.

Knowledge about this rare, albeit serious condition and awareness of the risk factors and the clinical picture are of the utmost importance in order to diagnose and treat intestinal ischemia and necrosis in due course.

Keywords: Intestinal necrosis; Low-flow and splanchnic vasoconstriction; Beta-blocker intoxication; Diagnostic CT; Symptoms and treatment

Introduction

Life-threatening intestinal ischemia and necrosis result from inadequate blood flow through the mesenteric vessels. It may be due to (a) occlusive vascular disease, caused by atherosclerosis and/or thromboembolism, and/or (b) non-occlusive vasoconstriction [1-8].

The condition is primarily found in the elderly and at least 17.5% of this part of the population exhibits stenotic or occluded, arteriosclerotic mesenteric arteries, usually asymptomatic [7,9]. Vasospasm exacerbates under-perfusion and ischemia and increases the risk of intestinal necrosis [3,4,7,10].

Splanchnic vasoconstriction occurs as a direct effect of medications such as digoxin and/or indirectly and reflexive to compensate for systemic circulatory failure and shock [4,5,10], hypovolemia, low-flow, hypotension, and bradycardia, as may be the case in beta-blocker intoxication [1,2,8,11].

Acute intestinal necrosis is a rare diagnosis, even in cardiologic patients with predisposing risk factors and comorbidities. It is probably often overlooked and undoubtedly increasing in incidence due to the aging population [5,7,8].

Intestinal necrosis typically gives rise to pain out of proportion to physical findings, diarrhoea, nausea, and vomiting [2,7]. Diagnosing the condition in its early stages is critical. Following the initial abdominal pain, the patient may experience an interval without symptoms, before the development of peritonitis, and when the pain returns saving the patient is often too late [1-5,8,12].

Case Presentation

A 72-year old female was admitted to the hospital from her nursing home, unconscious, hypotensive, anuric, cold and with bradycardia.

EKG two months previously showed sinus rhythm 66 beats/minute, PR 134 ms, QRS 100 ms, and QTc 450 ms (Figure 1), whereas EKG upon arrival showed sinus rhythm 26 beats/minute, PR 218 ms, LBBB 160 ms, and QTc 474 ms (Figure 2).

The patient was administered isoprenaline, loading dose followed by infusion; she immediately improved and became hemodynamically stable with pulse 50 and systolic blood pressure >110 mm Hg, and a normal echocardiography. She regained consciousness and was able to tell her story.

She had been diagnosed with diabetes mellitus many years previously and had been administered multi-substance therapy against arterial hypertension; and she was known with widespread arteriosclerosis, including occlusion of the coeliac trunk with collaterals from the superior mesenteric artery. In addition to this, she had cervical spondylarthrosis and severe spinal stenosis, and a few months earlier, she had suffered a fall followed by tetraparesis and analgesia.

Twenty hours before being admitted, by mistake, she happened to take twice her usual dosage of beta-blockers. For unknown reasons, she was being treated with both propranolol 80 mg × 2 and metoprolol 50 mg × 2. A few hours later, she felt increasingly ill, dizzy, tired, and nauseous, with severe vomiting, no pains. She felt improvement for a short time, but then rapidly deteriorated.

Upon admittance, the patient was found to have a distended abdomen and a resounding percussion note. Blood test showed severe metabolic acidosis and S-lactate 20 mmol/l, S-creatinine 220 micromoles/l and S-K 6.6 mmol/l (blood tests had been normal two weeks previously). She received intensive care and was administered calcium chloride, sodium bicarbonate, adrenaline, isoprenaline, but clinically remained on a downhill slope.

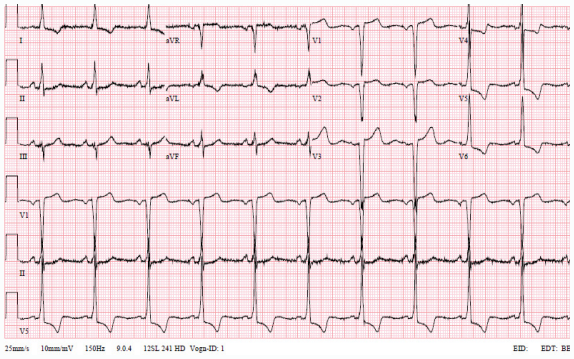

Figure 1: EKG two month before admission.

Citation: Olesen LL. Intestinal Necrosis Due to Beta-Blocker Intoxication. Cardiologist. 2020;1(2):1006.

Publisher Name: Medtext Publications LLC

Manuscript compiled: Mar 20th, 2020

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CT thorax and abdomen showed marked contrast-delay; and occlusion of the coeliac trunk (known in advance), and stenotic superior mesenteric artery (Figure 3A). In addition, the inferior mesenteric artery was severely arteriosclerotic with a proximal stenosis and sparse contrast in the distal part (Figure 3B). There was pronounced oedema of the wall of the colon (Figure 3C), but no pneumatosis. Corresponding to the affection of the three mesenteric arteries and their supply areas, ventricular retention and increased wall thickness were observed in the small intestine, the descending colon, and rectum (Figure 4). Ascites and pleural effusions underlined the advanced condition.

There was no treatment to offer and the patient died about 24 hours after onset of symptoms, 8 hours after admittance. No autopsy was performed.

Discussion and Conclusion

This tetraplegic patient felt no pain but otherwise exhibited symptoms of intestinal necrosis. Initially, the symptoms did not lead to the proper diagnosis although she had several risk factors for arteriosclerosis, known affection of the mesenteric arteries and aggravation of mesenteric low-flow due to excess ingestion of beta-blockers with classic EKG changes, severe bradycardia and hypotension, and reactive mesenterial vasospasm due to autoregulation [4,5].

Diagnosing Acute Mesenteric Ischemia (AMI) involves a combination of clinical suspicion and imaging. Laboratory tests are typically nonspecific. As the disease progresses, leucocytosis develops, and lactate levels rise late in the course due to bacterial metabolism and ongoing anaerobic metabolism suggestive of an ischemic process [1,3,7,12]. This patient had an excessively elevated S-lactate, which is consistent with colon-ischemia, but not specific. The most important test is CT angiography, and this patient offered changes characteristic of extensive intestinal ischemia and necrosis (Figures 3 and 4) [1-8].

As soon as suspicion of intestinal ischemia arises, it is imperative to initiate supportive care involving oxygen, fluid, broad-spectrum antibiotics, anticoagulation medication, and pain control while at the same time consulting a vascular surgeon and a gastric surgeon. For Non-Occlusive Mesenteric Ischemia (NOMI), vasodilatation should be considered and treatment with papaverine infusion. The treatment should restore the blood flow. The options available vary depending on the underlying cause, and early recognition and rapid treatment are critical to reduce mortality. The prognosis is severe, and when diagnosis is delayed AMI and NOMI are usually lethal [1-5,8,12].

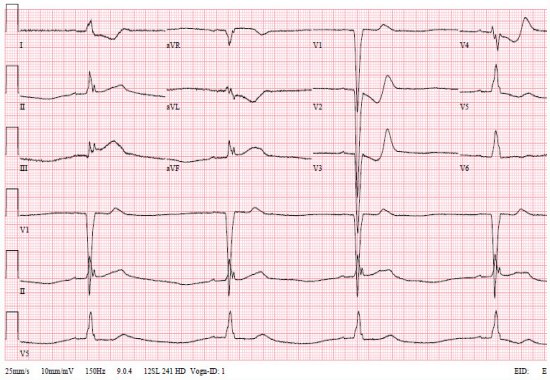

Figure 2: Sinus rhythm 26 beats/min and LBBB 160 ms - upon arrival, intoxicated with beta-blockers.

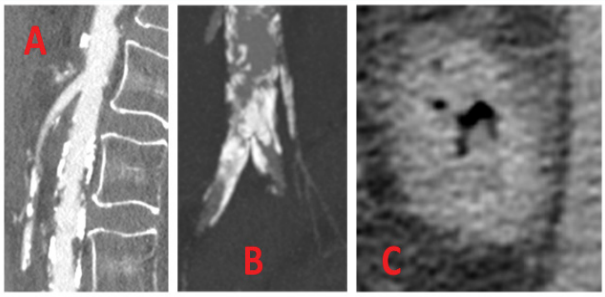

Figure 3: (A) Occlusion of the coeliac trunk and stenotic superior mesenteric artery; (B) The inferior mesenteric artery, arteriosclerotic with a proximal stenosis and sparse contrast in the periphery; (C) Pronounced oedema of the wall of the colon.

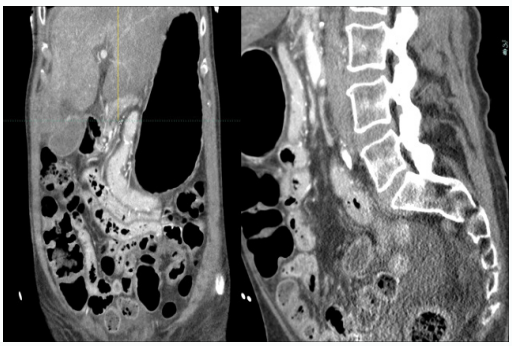

Figure 4: Ventricular retention and increased wall thickness in the small intestine, the descending colon, and rectum.

Acknowledgement

I want to thank the Department of Radiology, Roskilde, DK, to provide Figure 3 and Figure 4.

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