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Case Report

Amphetamine-induced small bowel ischemia – A case report ☆

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ABSTRACT

Nonocclusive mesenteric ischemia is most common in elderly patients with multiple comorbidities. Nevertheless, there are some reports of acute bowel ischemia in young patients with a history of recreational drug abuse. We describe the case of a 33-year-old patient who presented with acute abdominal pain following amphetamine consumption. Multidetector computed tomography showed nonocclusive segmental ischemia of the distal ileum, and the patient underwent emergency surgery with ileocecal resection. The patient recovered quickly and was discharged without any postoperative complications. An early and precise diagnosis of patients with intestinal ischemia having a history of amphetamine abuse is of utmost importance for prompt and proper treatment. Especially in younger patients, multidetector computed tomography should be tailored to use with less radiation. A single portal venous scan proved sufficient in our case.

Introduction

The use of recreational drugs (cannabis, cocaine, Methylendioxymethylamphetamin - MDMA, and amphetamines) was found to be more widespread at the municipal level. In selected Austrian cities, studies of illicit stimulants and their metabolites in wastewater were conducted. The estimated last-year prevalence of amphetamine uses in young adults (15-34 years) in Austria was 0.9%, with treatment entrants of 5.5% [1].

The abuse of amphetamine may result in a diverse array of side effects involving almost any body organ, including intracranial hemorrhage, cerebral ischemic stroke, posterior reversible encephalopathy syndrome (PRES), cerebral edema, vasculitis, myocardial infarction, cardiac arrhythmias, cardiomyopathy, pulmonary edema. All these are well documented [2]. In contrast, the literature about visceral ischemia following amphetamine consumption is scarce [3–5].

Given this, we describe a case of non-occlusive mesenteric ischemia due to amphetamine abuse.

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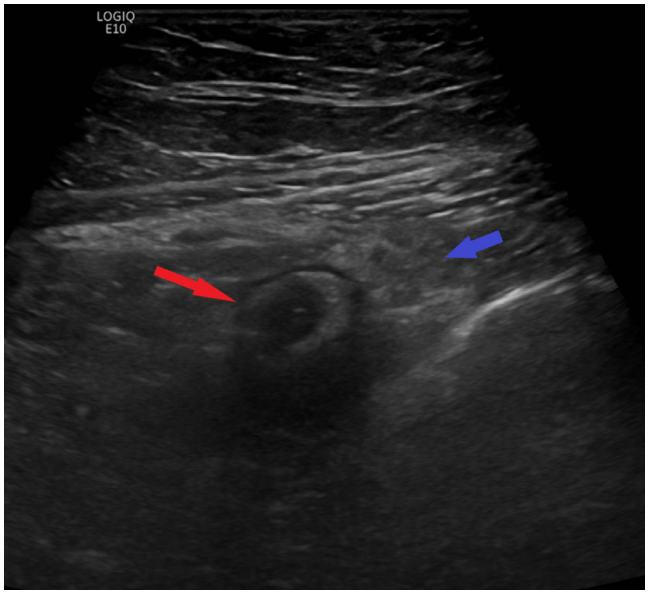

Fig. 1 – Ultrasound of the right lower abdomen (transverse view) showing mural thickening of the terminal ileum (red arrow) and surrounding mesenteric edema (blue arrow). (Color version of figure is available online.)

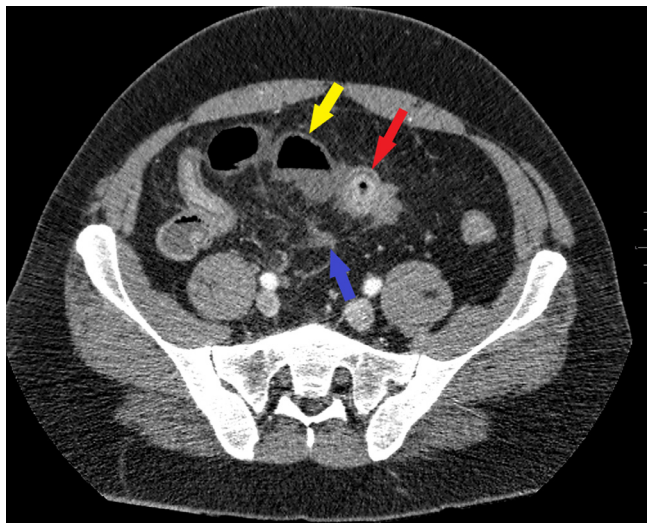

Fig. 2 – Axial contrast-enhanced CT (CECT) at portal venous phase demonstrates terminal ileum with mucosal hyperenhancement (red arrow) and dilatation of a nonenhancing segment (yellow arrow) as well as mesenteric fat stranding (blue arrow). (Color version of figure is available online.)

Case presentation

A 33-year-old male patient presented with acute abdominal pain in the right lower quadrant with a gradual onset over 8 hours. Self-medication with 3 g acetaminophen (Mexalen 500 g) and 0.8 g ibuprofen (Ibuprofen 400 mg) had no effect. The pain was associated with nausea and diarrhea, but not with emesis or fever. On clinical examination, the abdomen was distended with tenderness in the right lower quadrant. On detailed examination, the patient was found to use amphetamines and alcohol 4 hours before the onset of symptoms. His medical history was remarkable for previous appendectomy and recent cholecystectomy (3 months before the presentation).

At the time of admission, laboratory investigations revealed elevated Leucocytes 15.57 $\times$ 103/μL (4.40-11.31), C-reactive protein (CRP) 50.5 mg/L (0.0-5.0 mg/L) and an elevated level of Lactate 3.08 mmol/L (0.46-2.47 mmol/L). A drug screening test was not performed.

The abdominal ultrasound did not reveal the underlying cause of segmental ileal wall thickening and mesenteric free fluid (Fig. 1). Moreover, the sonographic evaluation of mesenteric vessels was limited by the patient's body habitus.

Apart from the possibility of an inflammatory cause as infectious enteritis or terminal ileitis, small bowel ischemia was yet to be ruled out. Therefore, the patient underwent multidetector computed tomography (MDCT) using a 64-slice scanner. He received 100 mL of nonionic contrast agent at a rate of 3 mL/s. A conventional portal venous phase was acquired 40 seconds after bolustracking. Standard axial, coronal, and sagittal reformations with 2 mm slices were automatically generated.

As seen on ultrasound, a long segment of the terminal ileum (approximately 30 cm) demonstrated abnormal wall thickening and mucosal hyperenhancement. Additionally, there was a 10 cm long segment of dilated, non-enhancing small bowel distal to the area of segmental wall thickening. A small volume of free fluid was present in the right lower quadrant (Fig. 2).

The scan demonstrated patency of the celiac trunk, superior mesenteric artery, inferior mesenteric artery as well as their central branches. There were no signs of atherosclerosis, fibromuscular dysplasia, or any other vessel wall pathologies in the large and middle-sized arteries. The large mesenteric veins had no thrombus or filling defect. (Fig. 3A, Fig. 3B). No solid organ infarction, perforation, pneumatosis intestinalis, superior mesenteric or portal venous gas were noted. However, the CT findings were highly suggestive for NOMI: the dilated, thin-walled, hypoenhancing segment was interpreted as being necrotic, and the wall thickened segment as partially ischemic or initially ischemic with subsequent reperfusion [6–8] (Fig. 4). Therefore, the patient was referred for urgent surgery. The imaging findings were confirmed, and ileocecal resection was performed with an end-to-end ileoascendostomy.

The pathological examination revealed hemorrhagic necrosis of the short-dilated segment of the terminal ileum with accompanying peritonitis. These findings are required for the definite diagnosis of NOMI [9].

After being admitted in the ICU for 1 day, the patient was transferred back to the surgical ward.

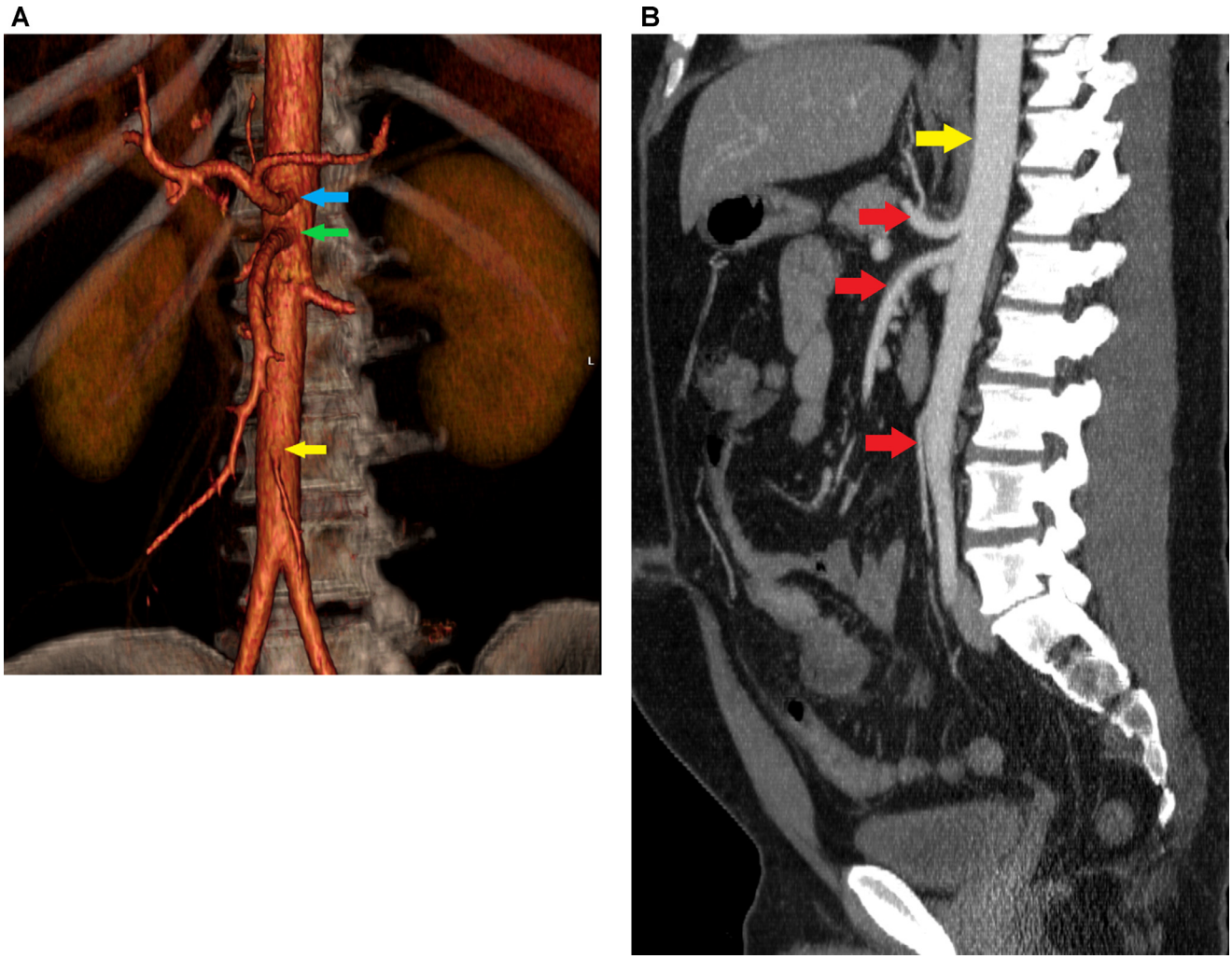

Fig. 3 – (A) Semiautomatic generated coronal 3D volume-rendered image at portal phase with manual trimming of the venous structures shows permeability of the celiac trunk (blue arrow), the superior mesenteric artery (green arrow), and the inferior mesenteric artery (yellow arrow). (B) Sagittal maximum intensity projection (MIP) contrast-enhanced CT (CECT) at portal phase shows no atheromatous plaques and non-atherosclerotic pathologies in the abdominal aorta (yellow arrow), the proximal celiac trunk, superior mesenteric artery and inferior mesenteric artery (red arrows). (Color version of figure is available online.)

To minimize septic complications related to bacterial translocation and peritonitis [10], broad-spectrum antibiotic therapy with piperacillin-tazobactam (Tazonam 4 g/0.5 g) was initiated on the first day postoperatively and continued for seven days. As no postoperative complications or newly developed bowel ischemia were observed, the patient was discharged on the eighth postoperative day. Planned appointments for follow up were uneventful. However, 3 weeks after surgery, the patient presented again for abdominal complaints. He reported a rapid weight loss of 7 kg and diarrhea up to 10 times daily. Based on extended laboratory work-up, colonoscopy, and a capsule endoscopy of the small bowel, any underlying diseases such as vasculitis or Crohn's-Disease were excluded. The newly developed symptoms were attributed to bile acid malabsorption following the recent ileocecal resection. Dietary changes relieved the patient's pain and reduced the frequency of diarrhea.

Discussion

Bowel ischemia can be caused by multiple conditions such as mesenteric arterial or venous occlusion, strangulating bowel obstruction, and hypoperfusion associated with non-occlusive vascular disease [7,8,11].

NOMI was first reported in elderly patients who underwent cardiovascular surgery or dialysis. In these cases, intestinal vasospasm caused by persistent low perfusion induce ischemic disorder due to decreased cardiac output and blood pressure. Digitalis preparations and catecholamines often induce vasospasm, and associations of NOMI with activation of the renin-angiotensin system and vasopressin secretion from the pituitary gland have also been reported [12,13].

Besides amphetamine, methamphetamine and cocaine also induce hypoperfusion due to mesenteric arterial vaso-

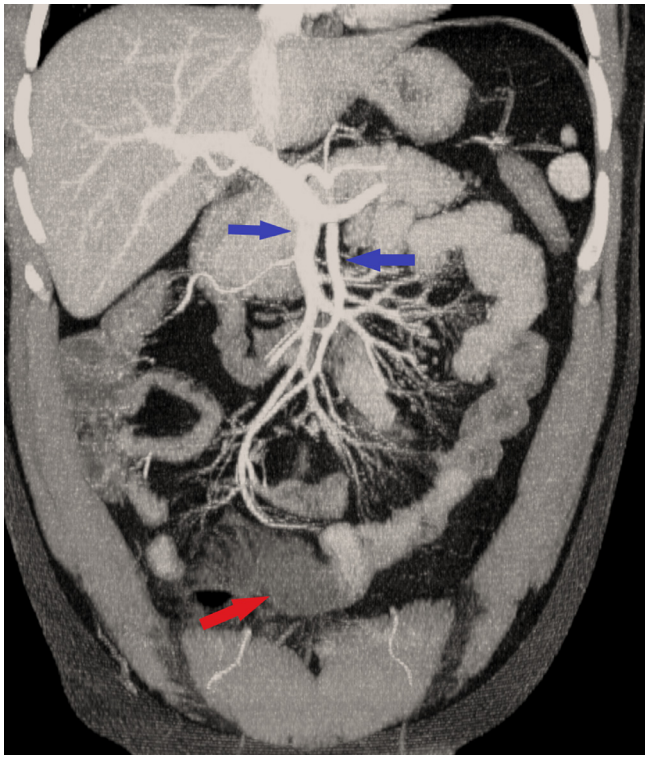

Fig. 4 – Paracoronal maximum intensity projection (MIP) CECT at portal phase shows no filling defects in the mesenteric veins and arteries (blue arrow) and necrotic dilated, thin-walled, hypoenhancing segment (red arrow). (Color version of figure is available online.)

constriction [2]. The plasma half-life of amphetamine ranges from 5 to 30 hours, depending on urine flow and pH [14,15]. Other authors reported delayed bowel necrosis and ischemic colitis associated with amphetamine and methamphetamine abuse [16–18].

Given our patient's history, the most probable cause of intestinal ischemia is amphetamine-induced vasoconstriction. Contrast-enhanced CT (CECT) showed no occlusion in mesenteric circulation. Furthermore, the patient had no hypovolemia, hypotension, cardiac failure, or no administration of digoxin, which are the most common causes of mesenteric arterial vasoconstriction with a reduction in mesenteric blood supply [7,8,19]. Although Doppler mode ultrasound can demonstrate proximal mesenteric vessel stenosis or occlusion with a sensitivity and specificity of 85%-90% [20], this was inconclusive in our obese patient.

A definite diagnosis of NOMI is still a challenge despite the advances in laboratory diagnosis and imaging techniques. Before the establishment of MDCT, an immediate angiographic examination upon the suspicion of NOMI was the first step in the diagnosis [13]. Direct vascular access allows the administration of vasodilative agents. Digital subtraction angiography, in contrast to MDCT, however, is not able to identify intestinal necrosis or perforation [20].

The short time between consumption and onset of symptoms, as well as the CECT appearance of the mesenteric vessels without evidence of emboli or thrombi, supports this etiology. The patient's gangrene was localized at the ileum.

The final diagnosis in the presented case was NOMI caused by amphetamine-induced splanchnic vasoconstriction.

The management of NOMI depends on the clinical and imaging findings. If the diagnosis is made before the onset of bowel wall necrosis and peritoneal signs, the treatment consists of selective intraarterial infusion of papaverine, prostaglandin E1, or nitroglycerine into the superior mesenteric artery [10,13,21].

When CECT findings are consistent with bowel wall necrosis and symptoms of peritonitis are present, expedient surgery should be performed [10,13].

The mortality rates of bowel ischemia caused by hypoperfusion associated with nonocclusive vascular disease range from 30% to 93% [19,21]; therefore, a prompt diagnosis is crucial.

Conclusion

We describe the case of a 33-year-old man who presented with an acute abdominal emergency caused by nonocclusive small bowel ischemia related to amphetamine consumption. A suspicion of intestinal ischemia should be raised when the presence of amphetamine, methamphetamine, or cocaine were found in the patient admitted to the emergency room along with acute abdominal pain.

In the case of our obese patient, abdominal ultrasound was inconclusive.

As radiation dose is a primary concern in younger patients, we performed a single-phase CECT scan. The CT findings, together with an appropriate clinical background, delivered vital information for surgical management decision making.

Patient consent

Written informed consent was obtained from the patient for publication of this case report and accompanying images.

Ethical approval

Ethical approval was not required in this case.

Author's contribution

Elena Ciupilan – wrote the original draft; interpreted radiological findings; Markus Gapp - reviewed and edited the original draft; provided relevant images; Robert Stelzl - approved the final draft; Sigrid Kastl - reviewed and edited the original draft.

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