Delayed Presentation of Massive Cocaine-Induced Enteropathy: Report of a Fatal Case

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Abstract: Cocaine-induced tissue injury is frequently seen, important health issue, especially presented as myocardial ischemia and infarction. Cocaine-induced gastrointestinal complications are fatal as well as unexpected. The toxic effects of cocaine causes thrombosis by damaging endothelium cells and leading to intimal hypertrophy or atherosclerosis. Thus substance-abuse may cause vascular complications and end organ ischemia. Acute mesenteric ischaemia (AMI) is a vascular emergency in the setting of inadequate blood supply to the intestine causing tissue necrosis. We presented the ultrasonography (USG) and computed tomography (CT) findings of a 30-year-old male with AMI accompanying extensive thrombosis in mesenteric vascular structures after substance abuse.

Keywords: Ischemia, substance abuse, cocaine-induced enteropathy

1. INTRODUCTION

Cocaine-induced tissue injury is frequently seen, important health issue, especially presented as myocardial ischemia and infarction (1). Cocaine-induced gastrointestinal complications are fatal as well as unexpected. Acute mesenteric ischaemia (AMI) is a vascular emergency in the setting of inadequate blood supply to the intestine causing tissue necrosis (2) and usually affect senior population. In the setting of AMI in young adults, underlying factors should always investigate such as inherited thrombophilies, various rheumatologic diseases, vasculitis and toxicological evaluation. We presented the ultrasonography (USG) and computed tomography (CT) findings of a 30-year-old male with AMI accompanying extensive thrombosis in mesenteric vascular structures after substance abuse.

2. CASE REPORT

A 30-year-old patient with a history of substance-abuse presented to the emergency department with extensive abdominal pain. On his initial laboratory finding serum aspartate aminotransferase level was:1650 units/L (references: 40 units/L), alanine amino transferase level was:1919 units/L (references: 45 units/L), white blood count was 32x10⁹ (references: 4 × 10⁹/L and 1.1 × 10¹⁰/L).

There was a widespread tenderness in the abdomen on physical examination with the palpation. Plain radiograph was non specific(Figure 1).

Figure1: Non specific plain radiograph findings is seen.
Abdominal USG was performed. Liver parenchyma was heterogeneous and extensive, echogenic foci with shadowing in portal vascular structures was seen on USG (Figure 2). Hypoechoic intestinal wall was seen due to oedema. Contrast enhanced CT revealed multiple, irregular, hypointense areas corresponding to acute enfarct on left and right lobe of liver, left kidney and spleen (Figure 3). Extensive gas densities within portal vascular structures was also noted in CT.

Localised intramural gas within intestinal wall and mesenteric oedema was presented (Figure 3). Findings was corresponding to pneumatosis portalis and intestinalis. Thrombus materials obstructing almost entire vessel lumen were observed in the celiac artery, superior mesenteric artery, splenic artery and left renal artery level of aorta on CT angiography. The patient expired after an urgent operation. Autopsy confirmed radiological diagnosis.

Figure 2: (a) On subcostal sonographic view of liver parenchyma; heterogenous and extensive, echogenic foci with shadowing in portal vascular structures is seen (white arrows). (b) On axial CT image of liver; extensive gas densities within portal vascular structures is demonstrated (yellow arrows).

Figure 3: On abdominal axial contrast enhanced CT images, abdominal aorta (a) and superior mesenteric artery (b) hypodense intraluminal filling defects consistent with thrombus (white arrows) is shown. (c) Jejunal intestinal segments are dilated and intramural air densities compatible with necrotizing enterocolitis are observed within the walls (yellow arrow). (d) Hepatic artery, splenic artery and left renal artery thrombosis leads to infarct in the liver, spleen and left kidney parenchyma seen as hypodense areas (red arrows). Air densities due to necrotizing enterocolitis are observed in the intrahepatic biliary tract (red arrow).
3. DISCUSSION

Pneumatosis portalis and intestinalis is an ominous, rare radiological findings characterized with localised intramural gas within intestinal wall and in portal venous structures due to intestinal necrosis and ischemia. Detected gas in portomesenteric structures has been reported to be associated with mortality above 75% (3). The toxic effects of cocaine causes thrombosis by damaging endothelium cells and leading to intimal hypertrophy or atherosclerosis(4). Thus substance-abuse may cause vascular complications and end organ ischemia.

Elramah et al. reported that in a significant number cases with gastrointestinal complications had delayed presentation more than 72 hours; which make it difficult to diagnose(5). In our patients; we could not obtain the proper information about drug-abuse, after our patient reinterrogated, history of cocain use was noted 5 days before the clinical picture. AMI is a rare, fatal complication of an important and common health issue; since substance abuse is a taboo, it increases the importance of trust in the patient physician relationship. AMI patients usually presented with severe abdominal pain and rectal bleeding. Urgent radiologic diagnosis is necessary as prompt surgical intervention is crucial. USG, CT and CT anjiography are the chose of modalities. Definitive diagnosis can be obtain using anjiography by demonstrating trombosis of mesentheric vascular structures. Sonographically hypoechoic intestinal wall, hypointense areas corresponding to acute enfarct on solid organ parenchyma and echogenic foci with shadowing in portal vascular structures can be seen. Gas in the portomesentheric structures and within the intestinal wall may be detect on abdominal CT. CT anjiography may reveal etiology by demonstrating thromboembolism in mesenteric artery and veins. Submucosal oedema may produce low density and thickening of the watershead areas of the intestinal wall can be monitored, free intraperitoneal gas can be detect if perforated. Differential diagnosis of AMI includes infectious colitis, ulcerative colitis, chron disease and diverticulitis.

In conclusion; cocaine-induced gastrointestinal complications are fatal as well as unexpected. Delayed presentation should be challenging in young adults and radiologist play a key role in the diagnosis of this severe clinical picture.

REFERENCES


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