Case Report

An unusual case of nonocclusive mesenteric ischemia in a young girl

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Abstract

Nonocclusive mesenteric ischemia is a type of acute mesenteric ischemia with high mortality seen mostly in elderly cardiac patients.We present a 21-year-old healthy student with nonocclusive mesenteric ischemia along with radiological evidence of hepatic portal venous gas and pneumatosis intestinalis, with subsequent fatality. Its significance and its possible etiology are discussed.

Keywords: Enteral feeding, nonocclusive mesenteric ischemia, vasopressin

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Introduction

Nonocclusive mesenteric ischemia (NOMI) is mesenteric ischemia with normal splanchnic vasculature. Although it accounts for 20–30% of all acute mesenteric ischemia, it has more than 70% mortality.[1] It commonly occurs in an elderly person having cardiovascular disease with low-cardiac output.[2] However, our case report describes NOMI in a 21-year-old student, in all probability due to demand–supply imbalance in splanchnic circulation under vasopressor support during septic shock.

Case Report

A 21-year-old college student presented to a tertiary care hospital with history of on and off low-grade fever for 3 months and abdominal pain for 1 week. She had no history of cough with weight loss or facial rashes. On admission, she was conscious but anxious and pale. She had blood pressure on the lower side of normal with distended abdomen. Both tuberculosis and vasculitis like systemic lupus erythematosus (anti-dsDNA, anti-sm-RNA) were ruled out in view of low-grade fever with abdominal pain. Erect X-ray and abdomen ultrasonography were normal. On the third day of hospital admission, she developed septic shock (mean blood pressure <65 mmHg) with altered sensorium for which she was intubated and transferred to the intensive care unit (ICU). Magnetic resonance imaging of head, cerebrospinal fluid study and serum sodium level (137 mmol/L) were all within normal limits. She had leukocytosis (21,000/cmm), anemia (5 gm/dL), thrombocytopenia (20,000/cmm) and deranged liver function tests (prothrombin time 6 s prolonged). Echocardiography showed mild diastolic dysfunction with ejection fraction >60%. She was managed conservatively with fluids, sedation, noradrenaline (0.5–3 µg/kg/min), vasopressin (0.01 U/min) infusion, broad-spectrum antibiotics and platelet and packed cell transfusion. Several blood and endotracheal tube aspirate cultures were sterile. On the fifth day, repeat ultrasonography of the abdomen revealed thickened and dilated nonobstructed bowel loops with coarse hepatic echo-texture. The gastro surgeon refused active management fathoming patient's prolonged shock. Since ICU admission, she had high nasogastric aspirates with feed intolerance despite the use of prokinetics; however, she started accepting nasogastric feeds on day 6. But, after a few hours of

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enteral feed, she developed rapidly rising intraabdominal pressure (>18 mmHg) with sudden deterioration in hemodynamics and increasing metabolic academia and hyponatremia (serum sodium 124 mmol/L). She passed large amounts of fresh blood-mixed loose stools, which was later found to be negative for *Clostridium dificile*. Bedside abdominal ultrasonography suspected presence of air shadows within the hepatic portal vein. Urgent computed tomography (CT) scan abdomen revealed pneumatosis intestinalis of the small bowel with dilated bowel loops [Figure 1] and gas in portal venous system [Figure 2]. In a setting of prolonged septic shock, vasopressor support and metabolic acidosis, the clinical and radiological picture was that of mesenteric ischemia. Exploratory laparotomy, done immediately, showed multiple areas of gangrenous patches and dusky discoloration of the jejunum with air bubbles in the subserosa. The major mesenteric vessels were pulsating and there was no evidence of thrombus, atherosclerosis or visible occlusion. No bowel perforation was detected. In view of extensive distribution of gangrenous patches, the surgical team released the intrabowel pressure by surgical incisions in the wall of the jejunum so as to reduce the increasing distension.

She succumbed within 4 h of operation. Postmortem examination could not be conducted as consent was not available.

Discussion

In NOMI, mesenteric blood flow ceases for a while due to an acute splanchnic vasospasm. It tends to occur mostly in the elderly patients who have low-cardiac output.[1,2] Myocardial infarction, ventricular dysfunction or arrhythmias, circulatory or cardiogenic shock, vasoactive drugs and heart failure are the various risk factors.[3,4] Nor-epinephrine and vasopressin can both lead to profound vasoconstriction of the splanchnic circulation.[5] Vasopressin is known to significantly lower blood flow in the superior mesenteric artery.[5,6]

We believe that NOMI in our case could be secondary to excessive circulatory vasopressin as a consequence of altered metabolism with deranged liver function.[7] Initiation of an enteral feed alters the intestinal supply–demand ratio,[8] leading to reperfusion injury[9] mediated by oxygen free radicals and neutrophil adhesion to the endothelium of mesenteric venules, which can result in NOMI.[10,11] The temporal relationship of enteral feeding and the episode suggests that enteral feeding-related supply–demand mismatch is the likely precipitating event.

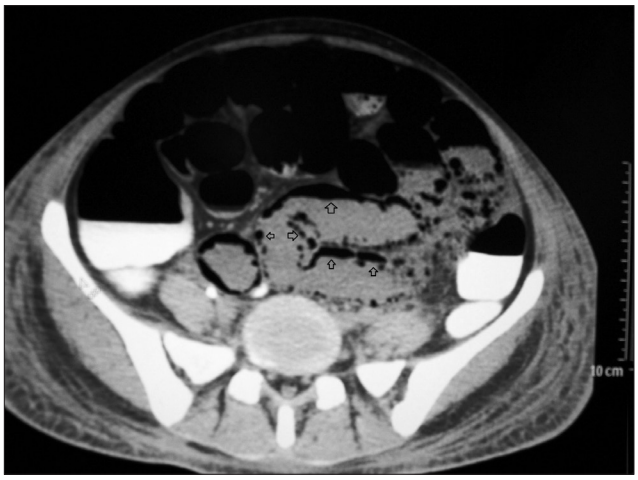

Figure 1: Compter tomography scan of the abdomen showing pneumatosis intestinalis (arrowed)

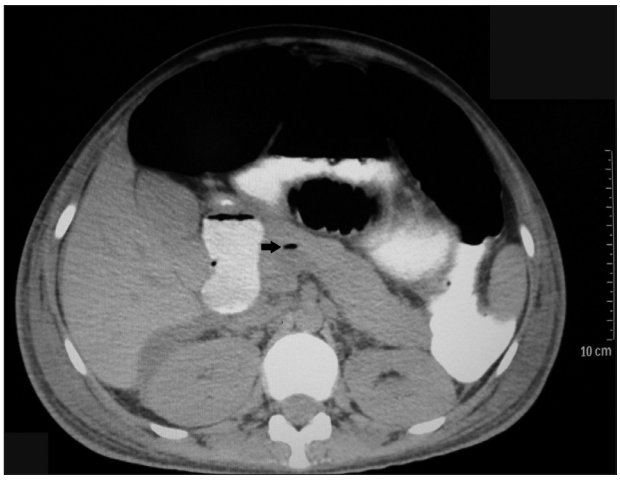

Figure 2: Compter tomography scan of the abdomen showing hepatic portal venous gas (arrowed)

It is a known fact that diagnosis of NOMI requires a high index of suspicion, especially in elderly patients with risk factors.[1,2] However, the index of suspicion must also be high for young patients with prolonged shock requiring vasopressor support. Early diagnosis of altered splanchnic blood flow in the backdrop of prolonged shock and vasopressor support could have averted the fatal outcome of a young patient. Our report shows that findings like increasing gastric aspirates, bowel wall thickening and dilated bowel loops should be investigated with increased vigor and managed accordingly. Gut-based organ failure scoring may help in early diagnosis as well as in monitoring therapy. Routine intraabdominal pressure measurement in septic shock may also help in the early detection of this fatal condition. Definitive diagnosis of NOMI however requires an arteriographic study,[2] which is costly and not widely available. Gastric mucosal tonometry is the only technique available at the bedside to measure splanchnic blood flow, but this technique certainly has

limitations.[8,11] Pneumatosis intestinalis[12] and hepatic portal venous gas[13] are late radiological features of bowel ischemia, and the presence of hepatic portal venous gas in bowel ischemia signifies mortality as high as 83%.[14]

Through this case report, we wish to emphasize the probability of occurrence of NOMI even in young age, especially in patients with prolonged septic shock on vasopressor support. The importance of early enteral feeding even in septic shock is proved, but hindrance in early enteral feeding needs to be timely appreciated and appropriately managed. Overzealous enteral feeding attempts may be harmful in this situation.

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