

Non-Occlusive Mesenteric Ischemia During the Course of Heart Failure

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Abstract

An 89 year old female with a history of myocardial infarction, chronic heart failure, hypertension, and atrial fibrillation was transferred to our institution with circulatory insufficiency. The patient had been undergone treatment for in-stent restenosis of the middle left ascending artery 1 week back. Her systolic blood pressure was 70 mm Hg with evident cyanosis over the whole body surface. Arterial blood gas analysis revealed severe metabolic acidosis and the patient exhibited severe hypovolemia. Initiation of dopamine therapy and transfusion ameliorated the circulatory failure. Three days after admission, she discharged massive bloody stools without absence of abdominal pain; colonoscopy revealed segmental pattern of mucosa necrosis in the sigmoid colon. Enhanced abdominal computed tomography revealed neither thrombus nor embolus in the mesenteric artery. Blood flow to the sigmoid colon was evident, prompting a diagnosis of non-occlusive mesenteric ischemia. Although emergency surgery was recommended, the patient declined and died of panperitonitis following conservative therapy. In addition to circulatory insufficiency caused by heart failure in this case, the intestinal ischemia also resulted from hypovolemia, diuretics and digitalis therapy, and arteriosclerosis.

Introduction

Acute mesenteric ischemia comprises acute obstructive mesenteric ischemia and acute non-obstructive mesenteric ischemia. Acute non-obstructive mesenteric ischemia is further divided into ischemic colitis and Non-Occlusive Mesenteric Ischemia (NOMI). Ischemic colitis is defined as an irreversible mucosal injury induced by intestinal ischemia without organic occlusion of the mesenteric artery. Marston et al. [1] classified ischemic colitis based on its severity: transient, reversible ischemia; ischemic ulcers with structuring; or gangrenous ischemic colitis. Subsequently, gangrenous ischemic colitis was removed from the classification of ischemic colitis as the progress of intestinal necrosis is irreversible. Conversely, NOMI is defined as intestinal ischemia arising from mesenteric vasospasm due to low cardiac output and circulatory insufficiency. NOMI has the highest mortality rate among mesenteric artery ischemias which results due to its delayed diagnosis of nonspecific clinical symptoms.

We describe the case of a female who developed NOMI during the course of heart failure, thereby suggesting an association between NOMI and circulatory insufficiency, diuretics or digoxin therapy, and atrial fibrillation.

Case Presentation

An 89 year old female with a history of myocardial infarction, chronic heart failure, hypertension, and atrial fibrillation was admitted to our hospital with worsening heart failure. She had been taking antiplatelet agent, anticoagulant agent, digitalis, diuretics, and angiotensin receptor blockers. Coronary angiography performed after recovery from heart failure revealed in-stent restenosis of the middle left ascending artery. Percutaneous coronary intervention was performed. One week after discharge, the patient was transferred to our hospital with circulatory insufficiency. Systolic blood pressure was 70 mm Hg, and respiratory rate was 40 breaths / min and pansystolic murmurs were heard on auscultation. Cyanosis was evident over the whole body surface. Blood chemistry analyses revealed inflammation (white blood cell count: 17,500/ μ l; C-reactive protein: 1.3 mg/dl), liver dysfunction (total bilirubin: 2.2 mg/dl; aspartate aminotransferase: 45 IU/l), renal dysfunction (serum creatinine: 2.66 mg/dl; serum urea nitrogen: 49.3 mg/dl), and coagulation dysfunction (prothrombin time: 28%;

international normalized ratio: 3.01; activated partial thromboplastin time: 46.3 s). Blood gas analysis revealed severe metabolic acidosis, based on reduced bicarbonate ion concentration. Chest radiography revealed cardiomegaly, with a cardiothoracic ratio of 77.3%, and protrusion of the right first and second, and the left third and fourth arches. Electrocardiography revealed atrial fibrillation and a heart rate of 66 beats/min. A synergy, with severe hypokinesia at the ventricular septum, moderate mitral regurgitation, and aortic regurgitation, were also evident. As the patient exhibited severe hypovolemia, transfusion and dopamine treatment were initiated; subsequently systolic pressure elevated to 100 mm Hg. On achieving circulatory stabilization 2 days after admission, intravenous dopamine infusion was discontinued. Three days after admission, she discharged massive, bloody stools without absence of abdominal pain. Colonoscopy (Figures 1a and 1b) revealed segmental pattern of mucosal necrosis in the sigmoid colon that was black in colour, suggesting gangrenous ischemic colitis. Enhanced abdominal computed tomography (Figures 2a and 2b) detected no thrombus or embolus of the mesenteric artery. Blood flow to the sigmoid colon was evident; therefore NOMI was diagnosed. Therefore, conservative treatment was continued; although emergency surgery was recommended, the patient declined and died on day 11 of admission due to panperitonitis. Pathological autopsy with the consent of her family found 700 ml of ascites with stools, and a necrotic sigmoid colon with three perforation sites. Microscopic examination (Figures 3a-3c) revealed inflammatory cell infiltration, submucosal

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haemorrhage, but no thrombus or embolus in the vasa recta. Severe fibromuscular intimal thickening with marked neutrophils infiltration in the vasa recta was evident. The main cause of death was panperitonitis resulting from colonic necrosis and perforation.

Discussion

Acute mesenteric ischemia is classified as acute occlusive mesenteric ischemia and acute non-occlusive mesenteric ischemia (divided into ischemic colitis and NOMI). NOMI is defined as bowel ischemia or necrosis without organic occlusion by thrombus or embolus of the mesenteric artery. NOMI is diagnosed when [2]:

- no organic occlusion is evident in the intestinal ischemic lesion
- intestinal ischemia or necrosis is segmental and discontinuous
- haemorrhage or necrosis is evident on pathological examination

NOMI accounts for 15% of mesenteric ischemia [3], occurring primarily in elderly patients with atherosclerosis-associated cardiovascular disease [4]. The mortality rate of NOMI is 70%-90% [5]. Conversely, ischemic colitis is defined as intestinal mucosal injury induced by hypoxia and reperfusion due to low intestinal blood flow, and with no organic occlusion of the mesenteric artery. Gangrenous ischemic colitis is the most severe form of injury, because the bowel ischemia is irreversible and causes necrosis. Therefore, the characteristics of gangrenous ischemic colitis are almost identical to those of NOMI. NOMI is treated by first improving hypovolemia and heart failure, which induces mesenteric vasospasm. Second, vasodilators such as papaverine, prostaglandin E1, and nitroglycerin are continuously administered into the mesenteric artery [6,7]. Surgical strategy is determined based on the signs of peritoneal

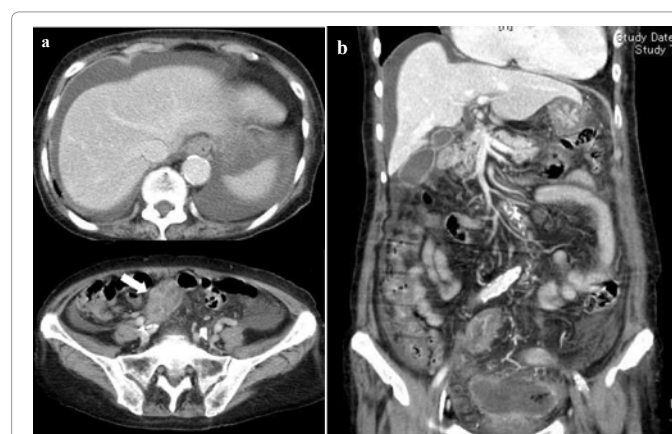


Figure 2: (a) Enhanced abdominal computed tomography Ascites was observed around the liver and spleen. The edematous sigmoid colon was enhanced with contrast injection (white arrow). (b) No thrombus or embolus was evident in the superior and inferior mesenteric arteries.

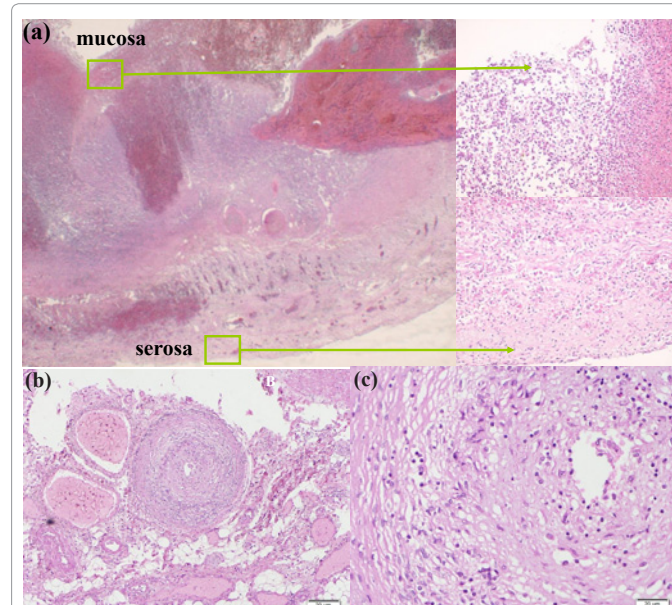


Figure 3: Pathological examination marked edema, inflammatory cell infiltration and haemorrhage were revealed in the submucosa. No thrombus or embolus was evident in the vasa recta. The structure of the colon wall was destroyed (a). Severe fibromuscular intimal thickening of the vasa recta was revealed (b) and neutrophils infiltration in the thickened intima (c) were observed.

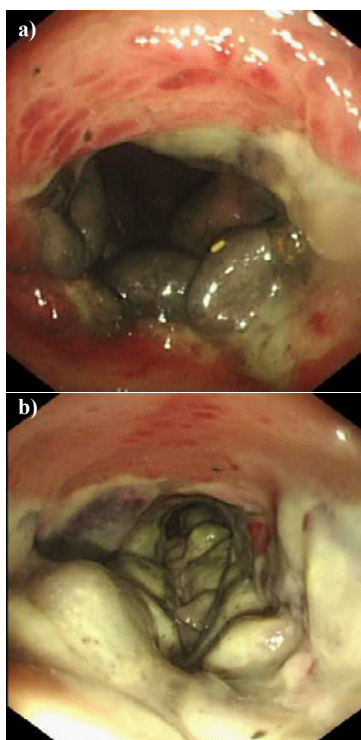


Figure 1(a,b): Colonoscopy examination Segmental patterns of the colonic mucosa (black in colour) were evident in the sigmoid colon, suggesting severe gangrenous ischemic colitis.

irritation. In cases with intestinal necrosis, intra-arterial vasodilator infusion promotes the spread of toxic substances throughout the whole body; therefore, surgery is recommended as soon as possible. Almost all the part of the small and large intestine are susceptible to ischemia: in the large intestine, the ileocecal region, splenic flexure of the colon, sigmoid colon, and rectosigmoid colon are particularly susceptible because anastomotic communications between the straight arteries are limited at these sites. Intestinal necrosis tends to distribute discontinuously and segmentally. As the clinical course of NOMI is slow, specific symptoms are often elusive. Although sudden, severe abdominal pain occurs with mesenteric thromboembolism, abdominal pain is often absent with NOMI [8]. Therefore, diagnosis of NOMI is often delayed and the clinical course of NOMI is usually fatal. In

our case, severe acidosis and peripheral circulatory dysfunction at admission could have resulted in intestinal ischemia. Dopamine is known to cause mesenteric vasoconstriction via an alpha-stimulating effect. Intravenous dopamine administration after admission could have worsened the intestinal necrosis. Fogarty and Fletcher [9] reported congestive heart failure, digitalis toxicity, and hypovolemia as the three main causes of NOMI. Some authors [10,11] report cardiovascular disease, use of digitalis and/or diuretics, arrhythmia, burn injuries, acute severe pancreatitis, gastrointestinal bleeding, and haemodialysis to be the risk factors for NOMI. Circulatory insufficiency induced by low cardiac output, and vasopressin or angiotensin stimulation due to reduced blood plasma volume cause vasospasm of the mesenteric artery. Consequently, prolonged vasospasm causes intestinal ischemia [5]. In a histological study of the straight arteries [12], arterial stenosis was identified in >80% of cases. Furthermore fibromuscular intimal thickening of the vasa recta was evident in 94.7% of cases, while luminal stenosis with neutrophils or plasma cell infiltration was observed in the tunica intima, media, and adventitia in some cases [13]. In our case, NOMI was associated with fibromuscular intimal thickening and inflammation of the straight segment in the necrotized intestine along with low cardiac output and reduced blood plasma volume.

Disclosure

We present the case of a female patient who developed NOMI during the course of heart failure. Acute mesenteric ischemia usually accompanies severe abdominal symptoms such as abdominal pain and/or vomiting; however, clinical symptoms in NOMI are often elusive. Elderly patients with chronic heart failure, using digitalis and/or diuretics, or with atrial fibrillation should be carefully monitored for intestinal ischemia.

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