Non-Occlusive Mesenteric Ischemia

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Introduction

Non-occlusive mesenteric ischemia (NOMI) is an acute mesenteric circulatory disorder that, in contrast to mesenteric arterial occlusion induced by blockage of blood flow by emboli and thrombi, is not caused by organic occlusion of blood vessels.[1] Good outcomes in NOMI are observed with early recognition and treatment.[1-2] The early symptoms and characteristics of NOMI, however, are unclear. In many cases, the disease has advanced to an irreversible stage before a definite diagnosis is made.

Case Report

A 59-year-old female presented with congestive heart failure, secondary to ischemic heart disease. She reported a two-day history of profuse watery diarrhea with mild cramping abdominal pain starting 30 minutes after eating and improving intermittently between meals. She had complained over four months of nausea and vomiting that had increased in frequency and of a 20-pound weight loss. She has been compliant with her medications and no new changes have been made within the last six months.

On physical exam, she was hypotensive with mild diffuse abdominal tenderness. The laboratory investigation showed a high white blood count of 25,000 with a bandemia of 22%.

Mesenteric ischemia was suspected and a CT scan of her abdomen showed diffuse thickened small bowel loops (Figure 1). A CT angiogram of her abdomen revealed patent mesenteric vessels (Figure 2).

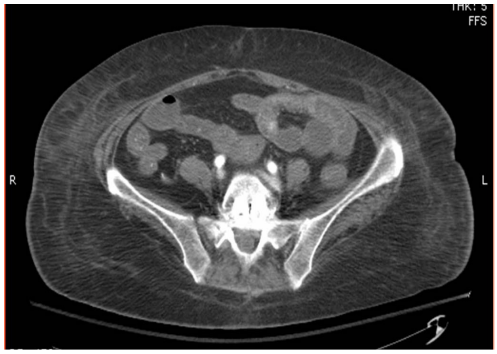

Figure 1. CT scan of the abdomen showed diffuse thickening of small bowel loops.

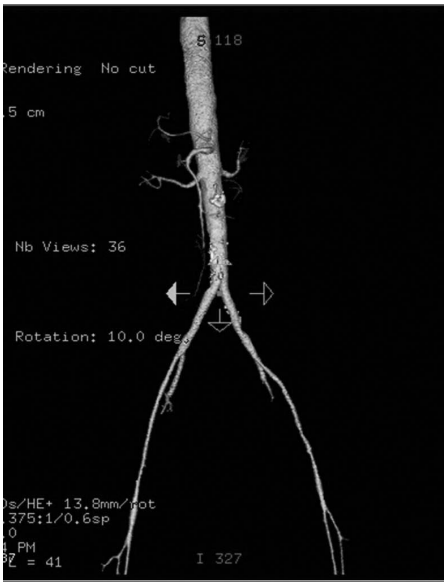

Figure 2. CT angiogram with 3-dimentional reconstruction showed patent mesenteric vessels.

A colonoscopy (Figure 3) showed necrosis from the anal margin to the left splenic margin, necrosis of the cecum and terminal ileum with preserved mucosa of the transverse and right colon consistent with a diagnosis of NOMI.

The patient went into septic shock and expired after one day.

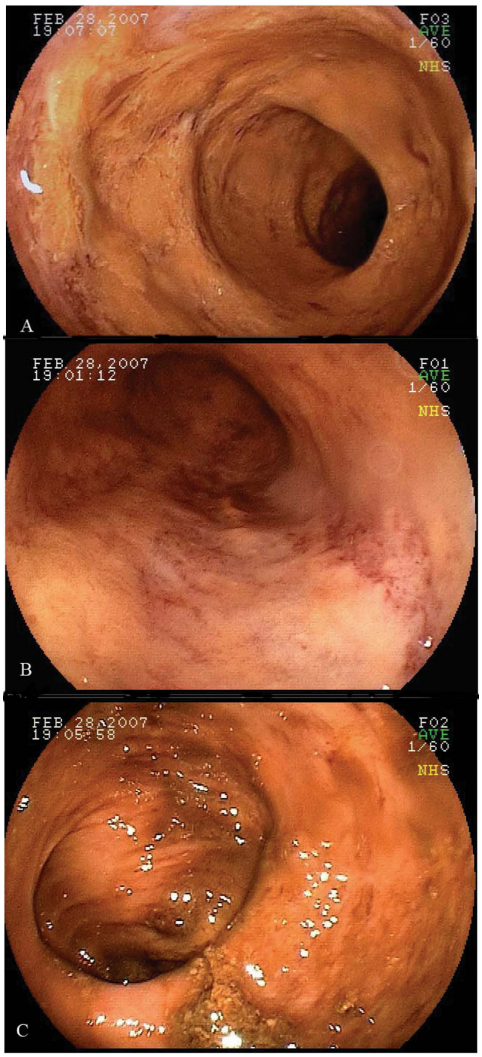

Figure 3. (A - B) Colonoscopy at the level of the sigmoid and splenic flexure showed a pale mucosal with diffuse ischemia, scattered shallow irregular ulcerations, longitudinal and irregular in form with gray-yellow exudates. (C) A diffuse ischemic mucosa of the colon is seen with overlying exudates at the hepatic flexure.

Discussion

NOMI is the result of splanchnic vasoconstriction occurring in response to a variety of systemic insults that diminish mesenteric blood flow.[1,3] The macro-vasculature is patent, but the microvascular blood flow is inadequate to meet intestinal tissue demands leading to gangrene. The consequences are disastrous and the prognosis is very poor, despite the absence of organic obstruction in the principal arteries.[1,4] NOMI accounts for more than 10% to 20% of cases of acute mesenteric circulatory disorders, mainly in elderly patients, with a mortality rate of 70% to 90%.[2-3]

The pathophysiology of NOMI involves low blood flow states such as shock, heart failure, hemodialysis, and direct splanchnic arteriolar vasoconstriction by drugs (e.g., digoxin).[2-3,5] Intestinal vasospasm due to persistent low perfusion is thought to be the inciting factor. NOMI can present with abdominal pain, nausea, vomiting, and ileus, but the characteristic early symptoms and laboratory test results are unclear. Early diagnosis is difficult and during the diagnostic process the disease slowly advances to an irreversible state with extensive intestinal necrosis.[2,4-5]

Angiography is the gold standard for diagnosis. Its invasive nature and potential for contrast nephropathy, however, makes angiography a less than optimal screening tool, thereby missing the opportunity for resolution in many cases.[1-2,5] For definite diagnosis, the absence of organic obstruction of blood vessels distributed in the necrotic intestinal region and segmented discontinuous intestinal and colonic ischemic changes with necrosis on colonoscopy or laparotomy are required.[3-4] However, the time required for definite diagnosis may compromise the chances of survival.[3]

The endoscopic feature in NOMI is segmental distribution with a clear boundary between the injured and uninvolved region. The lesions could range from marked edematous mucosa with loss of clear vascular vessel pattern to scattered shallow irregular ulcerations, longitudinal or irregular in form, with gray-yellow exudates.[8]

The role of colonoscopy is limited to the evaluation of the mucosal severities and the extent of the disease. It may be helpful in predicting clinical status and the prognosis of the patients.[6-8] It is safe and helpful in the early phase but should be performed with great care because increased pressures from insufflations could induce new ischemic lesions.[7-8]

Recently, abdominal contrast multi-detector row computed tomography upon suspicion of NOMI has emerged enabling a rapid definite diagnosis and providing vascular information comparable to that obtained in angiography. It permits subsequent early initiation of therapy and monitoring of disease resolution.[1,9]

The initial treatment is to correct predisposing or precipitating causes. Relief of acute congestive heart failure, correction of unstable or new cardiac arrhythmias, and replacement of blood volume should precede any diagnostic studies.[3-4] The main goal of current therapy for NOMI is reduction of spasm and improved perfusion of the mesenteric artery mainly with continuous administration of vasodilators into the mesenteric artery such as papaverine, prostaglandin E1, and nitroglycerine. The role of surgery is limited to diagnostic laparotomy and excision of irreversibly necrotized intestine.[3,10]

Conclusion

NOMI is increasingly more common due to the aging of the population, but the disease concept has not been established fully. Moreover, NOMI is difficult to diagnose, lacks characteristic symptoms, and is fatal in the advanced stage. Therefore, many patients may not have been diagnosed correctly and consequently may have died without receiving adequate treatment. Prognosis is related to the time of treatment initiation. Early diagnosis in suspected cases and early initiation of treatment may increase survival of NOMI patients.

References

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