Case Report

Nonocclusive Mesenteric Ischemia after Aortic Surgery in a Hemodialysis Patient

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We report a case of nonocclusive mesenteric ischemia (NOMI), which developed after grafting for a descending thoracic aortic aneurysm in a hemodialysis patient. On postoperative day 5, acute increases in serum enzyme levels developed. Emergency angiography revealed severe vasoconstriction in the superior mesenteric artery (SMA) and other splanchnic arteries. Therefore an infusion of papaverine hydrochloride was started into the SMA. Although serum enzyme levels decreased, metabolic acidosis occurred the next day. An emergency laparotomy revealed segmental diffuse necrotic small intestine and colon. Despite a resection of the small intestine and sigmoid colon, the patient died of septic shock several days later. NOMI is uncommon, but it is a catastrophic event that can occur after cardiovascular surgery. If intestinal gangrene is suspected, prompt mesenteric angiography and vasodilator therapy followed by exploratory laparotomy should be performed without delay. (Ann Thorac Cardiovasc Surg 2008; 14: 129–132)

Key words: nonocclusive mesenteric ischemia, hemodialysis, aortic surgery

Introduction

Nonocclusive mesenteric ischemia (NOMI) following cardiovascular surgery is a rare but usually lethal complication. Many studies have shown that selective mesenteric angiography with the concomitant perfusion of papaverine hydrochloride is an effective diagnostic and therapeutic tool, and surgery plays a secondary role in the early stage of NOMI.[1–7] However, laparotomy should not be delayed out of fear of intervening after cardiovascular surgery in critical situations. We present a case of NOMI, which developed after grafting for a descending thoracic aortic aneurysm in a hemodialysis patient.

Case

A 69-year-old man who had been undergoing hemodialysis for end-stage renal disease was admitted to our hospital with back pain. Computed tomography and angiography demonstrated a descending thoracic aortic aneurysm above the level of the diaphragm with a maximum diameter of 65 mm (Fig. 1). An elective operation was then performed. Under femoro-femoral cardiopulmonary bypass (CPB), prosthetic graft replacement with the reconstruction of two pairs of intercostal arteries was performed through a lateral thoracotomy. There was no visceral or renal ischemic time. The postoperative hemodynamic condition was stable with a low dose of an inotrope under continuous hemodiafiltration. The patient regained consciousness several hours later and was extubated on postoperative day (POD) 1. The patient experienced nausea and vomiting without abdominal distention or pain on POD 3. The serum levels of glutamic-oxaloacetic transaminase (GOT), lactate dehydrogenase (LDH), and creatine phosphokinase (CPK) were significantly elevated on POD 5 (GOT: 5,247 IU/L; LDH: 17,501 IU/L; CPK: 10,263 IU/L), and emergency angiography was performed (Fig. 2).

Angiography revealed severe vasoconstrictions of the superior mesenteric artery (SMA) and other splanchnic arteries (Fig. 3). The selective SMA angiogram demon-

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Received August 28, 2006; accepted for publication May 26, 2007
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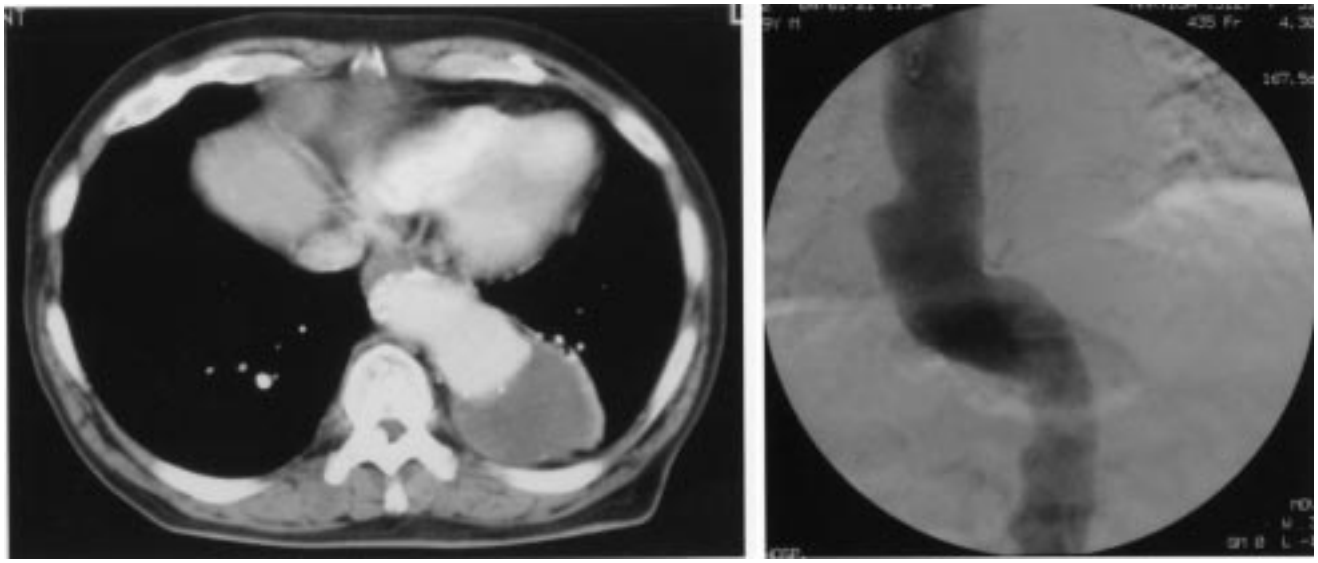


Fig. 1. Computed tomography and angiography demonstrate a descending thoracic aortic aneurysm above the level of the diaphragm with a maximum diameter of 65 mm.

strated severe vasoconstriction of the distal SMA and all of its branches, including a spasm of mesenteric arcades and reflux into the aorta, all of which are representative of NOMI. With angiographic evidence of NOMI, the inserted catheter was used for an immediate intra-arterial infusion of papaverine hydrochloride into the SMA at a dose of 60 mg/h. On the next day, though the serum levels of GOT, LDH, and CPK had decreased, the metabolic acidosis occurred and the patient was then reintubated. Arterial blood gases indicated that pH and base excess had been exacerbated from 7.403 and –2.1 mEq/L on POD 5 to 7.276 and –10.2 mEq/L on POD 6.

An emergency laparotomy was performed on POD 6, which revealed segmental diffuse necrotic changes of the small intestine, extending from the region of the jejunum 90 cm distal to Treitz's ligament to the region of the ileum 10 cm proximal to the terminal ileum, and also to the sigmoid colon (Fig. 4). We performed a wide resection of the small intestine with end-to-end anastomosis of the residual small intestine and resection of the sigmoid colon with creation of a colostomy. Unfortunately, however, the patient died of septic shock on POD 22.

Histopathologically, the resected specimen showed hemorrhagic necrosis in the mucosa and submucosa, but no thrombus formation was seen in the SMA and vein.

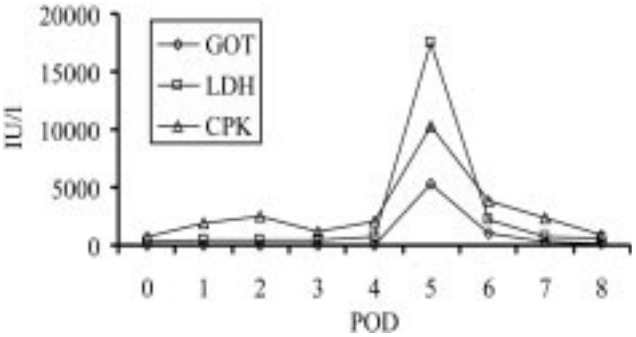
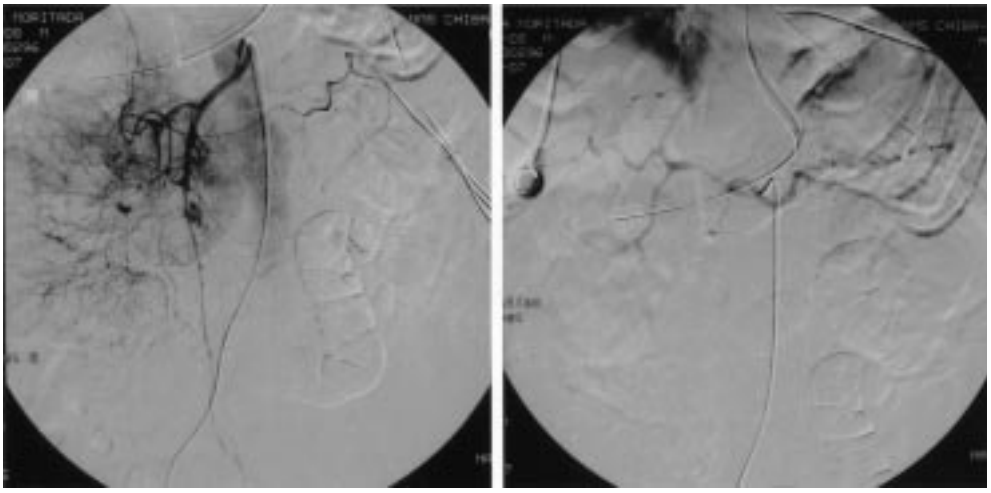


Fig. 2. The serum levels of glutamic-oxaloacetic transaminase (GOT), lactate dehydrogenase (LDH), and creatine phosphokinase (CPK) are significantly elevated on postoperative day (POD) 5 (GOT: 5,247 IU/L; LDH: 17,501 IU/L; CPK: 10,263 IU/L) and decrease on POD 6.

Discussion

NOMI, which is defined as diffuse intestinal ischemia in the presence of a patent arterial trunk, often results in intestinal gangrene. Since the first description of NOMI by Ende in 1958, its incidence after cardiovascular surgery has been reported to be about 0.05% to 0.1%.[1–5] Unfortunately, the prognosis of NOMI resulting in intestinal infarction after CPB has remained dismal with a mortality rate varying from 60%–100%.[1–8] Without prompt intervention, the natural history of NOMI is a progression from localized mucosal ischemia to transmural infarction, ultimately resulting in perforation, peritonitis, and death.

Besides the severity of the disease, delay in the diagnosis of NOMI is the main cause of the high mortality



A | B

Fig. 3. Angiography demonstrates severe vasoconstriction of the SMA (**A**) and other splanchnic arteries (**B**).
The selective SMA angiogram reveals severe vasoconstriction of the distal SMA and all of its branches, including spasms of mesenteric arcades and reflux into the aorta, all of which are representative of NOMI.

rate.[1–8] Therefore a high index of clinical suspicion is essential. Significant risk factors identified for the development of NOMI after cardiac surgery include emergency CPB, the use of an intra-aortic balloon pump, failed percutaneous transcoronary angioplasty resulting in emergency CPB, prolonged pump time, hemodialysis, and advanced age.[2–4,8,11,12] In consideration of these risk factors, a heightened suspicion of mesenteric ischemia may decrease the diagnostic delay and allow for a more effective use of all therapeutic options. Nevertheless, physical findings may be nonspecific and laboratory parameters, are of limited value.[3–8] Radiographic studies, including plain abdominal films or computed tomography scans, are not helpful in diagnosing NOMI.[4–7] Angiography remains the gold standard for the early diagnosis of NOMI.[1–8] However, an indication of angiography for NOMI is not apparent. Klotz et al. demonstrated that the indication for selective mesenteric angiography is established if at least one of four possible indications for mesenteric ischemia is present: (i) no defecation later than 3 days after surgery, despite maximal laxative treatment, (ii) severe abdominal bloating with a considerably distended belly, (iii) clinical and radiologic signs of paralytic ileus, or (iv) borderline or elevated serum lactate.[5] However, this indication of mesenteric angiography is often impractical in cases of abdominal complications following cardiovascular surgery.

There is no definitive guideline to treat patients with

Fig. 4. An emergency laparotomy reveals segmental diffuse necrotic changes of the small intestine, extending from the region of the jejunum 90 cm distal to Treitz's ligament to the region of the ileum 10 cm proximal to the terminal ileum, and also to the sigmoid colon.

NOMI after cardiovascular surgery. A prospective, randomized study is not likely to be performed in view of the paucity of the patients and fatality of the disease. In general, there are two types of management for NOMI after cardiovascular surgery. Patients who have signs of peritonitis should undergo laparotomy, whereas patients who have no signs of peritonitis should have prompt di-

agnostic angiography and subsequent infusion therapy. In recent years, some studies have reported that selective mesenteric angiography with the concomitant perfusion of papaverine hydrochloride is an effective diagnostic and therapeutic tool and should be performed in all patients with NOMI.[4–7] Moreover, some authors have shown that laparotomy should be reserved for patients with acute arterial thrombosis, mesenteric arterial embolism, or persistent symptoms despite intra-arterial papaverine.[6,7] However, laparotomy should not be delayed for fear of intervening after cardiovascular surgery, particularly because missed intestinal ischemia results in a 100% mortality rate.[3] Boley et al. reported only a 20% survival rate among patients with NOMI who had peritoneal signs and were treated initially with intra-arterial papaverine.[1] They suggested, however, that the extent of bowel resection could be markedly reduced if laparotomy was attempted following infusion therapy in patients with peritonitis, and that mortality rates were correlated closely to the extent of bowel resection.[1] Difficulties in the diagnosis of peritonitis after NOMI are attributed to ventilator support and heavy sedation, making communication and physical examination difficult.[3] Metabolic acidosis and a rise in the serums GOT, LDH, or CPK occur late in the course of intestinal ischemia and often signify irreversible ischemia or infarction, though these markers are not specific during conditions of low cardiac output syndrome.[2–4] If there is the slightest suspicion of necrosis or peritonitis in critical situations such as our patient, in whom serum markers were already elevated without peritoneal signs under sedation, a combination of intra-arterial pharmacologic treatment followed by diagnostic laparotomy and bowel resection might ultimately yield the best results.[3]

Another striking aspect of this case is that angiography demonstrated severe vasoconstriction in the SMA and other splanchnic arteries. Most previous studies on NOMI have focused on vasoconstriction in the SMA alone.[9,10] Some authors have reported a successful resolution of NOMI by a selective infusion of vasodilators into the SMA, but unsuccessful cases are rather more common.[1–8] In this critical situation in which angiography reveals extensive severe vasoconstriction, angiographic control and monitoring of serum markers should be performed deliberately, referring patients to laparotomy when there is no reaction to vasodilator infusion or if serum markers suggest necrosis or peritonitis.

Conclusion

NOMI is uncommon, but it is a catastrophic event that can occur after cardiovascular surgery. If intestinal gangrene is suspected, prompt mesenteric angiography and vasodilator therapy followed by exploratory laparotomy should be performed without delay.

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