UNUSUAL CASE OF INTESTINAL ISCHEMIA AFTER VENTRICULAR ASSIST DEVICE IMPLANTATION: A CASE REPORT

Harveen K. Lamba, Kevin Chen, Yakov Elgudin
Division of Cardiothoroacic Surgery, Luis Stokes Cleveland Veterans Affairs Medical Center, Cleveland, USA

Ventricular assist device (VAD) technology is being increasingly used for circulatory support and refractory heart failure. Known complications of VAD technology including bleeding and thrombosis. We report a rare case of acute mesenteric ischemia in a VAD patient, who was successfully treated with segmental small bowel resections.

Key Words: acute mesenteric ischemia; ventricular assist device; heart failure

Introduction

Advanced heart failure is a growing epidemic with a significant public health burden. The rise in the number of patients who develop progressive heart failure is thought to be due to the reduction in deaths from acute coronary syndromes, improved survival with most cardiovascular interventions and the use of implantable cardioverter defibrillators [1]. While medical management for end stage heart failure has made significant advances over the years, heart transplantation is the only therapy known to improve functional capacity and life expectancy [2]. However, because of shortages of heart donors and a growing number of patients who are not eligible for transplantation but still require cardiac support, an increasing number of patients undergo implantation of a left ventricular assist device (VAD). VADs can serve as bridge to transplantation, bridge to myocardial recovery, or as end-stage therapy. Although infectious and bleeding complications after VAD implantation are common, intestinal ischemia has been rarely reported [3]. To our knowledge, this is the first report describing VAD associated acute mesenteric ischemia.

Case Report

A 67-year-old male with severe ischemic cardiomyopathy was admitted to our cardiovascular unit for advanced refractory heart failure. He had a history of percutaneous coronary intervention (PCI) with bare metal stents on three separate occasions after suffering a myocardial infarction (MI) in 2007. His past medical history was also significant for left hemicolectomy for adenocarcinoma of the colon. During his admission he underwent implantation of HeartMate II left VAD as end-stage therapy. VAD parameters were stable (flow 4.5 to 5.6 L/min, speed 9000 to 9400 rpm, and power of

5.5 W) and he was weaned off of ionotropes by POD 2. He was closely followed by Infectious disease (ID) service closely for persistent low grade fever and leukocytosis and started on prophylactic antibiotic coverage with vancomycin, piperacillin-tazobactam, and fluconazole.

On POD 3 he developed mild abdominal pain and abdominal distension with decreased bowel sounds. Over the next day his condition quickly deteriorated. He was febrile to 39.2 °C, with worsening abdominal tenderness and distention, delirium and hemodynamically instablity requiring epinephrine and vasopressin (blood pressure, 70/50 mmHg; pulse, 100 bpm, central venous pressure 14 mmHg, cardiac index 1.5 L/min·m2). VAD paramaters remained unchanged (flow 5.0 L/min and speed 9400 rpm). Laboratory findings included increasing leukocytosis to 22,000/mm3 and compensated metabolic acidosis with mildly elevated lactate level of 1.5 mmol/L. Sepsis was suspected and CT scan of chest abdomen and pelvis was performed. He had pneumatosis intestinales involving a substantial portion of the small intestine, portal venous gas, and thickening of the intestinal wall, indicative of significant bowel ischemia. The patient was immediately taken to the operating room for an emergent exploration laparotomy, which revealed multiple areas of full thickness ischemia of the small intestine without perforation. Intraoperative colonoscopy demonstrated a dilated colon with viable mucosa. Consequently, two small bowel resections were performed. The bowel

was left in discontinuity and a temporary abdominal closure was enacted. After three days, he returned to the operating room for a second look. An additional area of bowel ischemia on the proximal small bowel segment was resected and the bowel was re-anastomosed in a stapled end-to-end fashion. The abdomen was closed.

The patient initially progressed well but unfortunately, within four days again developed signs of sepsis (fever of 38 °C and leukocytosis of 14,000/ mm3) and respiratory distress requiring re-intubation. Another exploratory laparotomy was performed, during which 5.5L of enteric fluid was suctioned out. A small 1cm anastomotic defect at the staple line was found at the more proximal small bowel anastomosis and was hand repaired. Due to lack of gross bowel necrosis and risk of developing short gut syndrome, no additional bowel resection was performed and the abdomen was left open. On second look at 48 hours, there was no evidence of additional bowel ischemia or perforation. The abdomen was closed and he was admitted to the surgical intensive care unit. He had a prolonged hospital course complicated by persistent gastrointestinal bleed requiring interventional radiology guided embolization and recurrent sepsis secondary to intra-abdominal abscess, requiring percutaneous drainage. He progressed slowly and was discharged to home on POD 129.

Histopathology of surgical specimens demonstrated acute ischemic necrosis with ulcerated mucosa and inflammatory infiltrates. The submucosal vessels were congested but no thrombi were seen.

Discussion

Gastrointestinal complications following cardiac surgery are seen in 0.4 to 2.9% of cases and include mesenteric ischemia, pancreatitis, gastroduodenal ulceration, acute choleycystitis, hollow viscus perforation and hepatic failure [4]. Of these, acute mesenteric ischemia (AMI) carries the highest mortality rate (40 to 94%), making it a rare but fatal complication [5].

Clinical presentation of AMI can be vague, insidious and non-specific, making early diagnosis and treatment difficult. This delay contributes to the poor outcomes associated with AMI. Traditional CT angiography (CTA) and Magnetic resonance angiography (MRA) are the gold standard for diagnosis [6]. In our patient the clinical onset of AMI, as evidenced by abdominal distension and tenderness, fever, leukocytosis, and acidosis, was quick and thought to be the result of sepsis. Treatment of AMI was delayed until the final results of CT abdominal scan demonstrating portal venous gas and pneumatosis intestinalis, signs highly suggestive late findings of AMI [7].

AMI can result from arterial embolism, arterial thrombosis, venous thrombosis, or hypoperfusion. Hypoperfusion or non-occlusive mesenteric ischemia (NOMI) is the most frequent cause of AMI after cardiac surgery. It is related to reduction in splanchnic blood flow from low cardiac output and is aggravated by inotropic support, preexisting atherosclerosis, and ventilator dependence [8]. In our patient, the presence of a ventricular assist device must also be considered for its role in AMI. The Heartmate II VAD is an axial flow, implantable pump with blood-immersed bearings. The major complications associated with axial flow VADs are thromboembolism and hemorrhage. However, the risk from thromboembolism and pump thrombosis is reported to be quite low in the HeartMate II population [9]. In our case, there was no evidence of pump thrombosis as demonstrated by pump flow rate, speed, and power. Additionally, the distribution of ischemia in our patient did not demonstrate an embolic pattern in which the distal small bowel and proximal jejunem and transverse colon are involved. Rather, it was patchy and segmental as is typically seen in NOMI [10]

Conclusion

Ventricular assist devices are becoming increasingly common for the treatment of heart failure. While acute mesenteric ischemia remains a rare complication it is associated with significant morbidity and mortality, necessitating early diagnosis and treatment. In a patient with septic condition after cardiac surgery, especially from an unknown origin, the diagnosis of mesenteric ischemia from NOMI and or embolism should be considered.

Conflict of Interests

None of the authors have any conflict of interest to declare.

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For correspondence:
Lamba Harveen K.
Address: 10701 E. Blvd, Cleveland, OH 44106
Tel. (202) 316-2146, E-mail: hkl12@case.edu