

Nonocclusive mesenteric infarction: a rare complication of continuous ambulatory peritoneal dialysis

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Abstract

This report describes an uncommon condition, nonocclusive mesenteric ischemia (NOMI) in a patient with end-stage renal disease on continuous peritoneal dialysis therapy. The patient presented with nonspecific abdominal symptoms and leucocytosis which later progressed to an acute abdomen due to generalized peritonitis of unknown origin. She had been hospitalized with an episode of hypotension four days before surgery. At emergency laparotomy, we detected an unusual form of ileal infarction that was characterized by multiple, variable sized and patchy-looking necrotic areas, of which a few were perforated. We resected the infarcted ileal segment, and did a proximal ileostomy. The patient was as well as she could be in relation to her chronic renal disease. We emphasize the need for surgeons to consider NOMI as a cause of peritonitis in patients with renal failure.

Keywords

Peritonitis; nonocclusive mesenteric ischemia; continuous ambulatory peritoneal dialysis; emergency laparotomy.

Introduction

Nonocclusive mesenteric ischemia (NOMI) is a rare condition in which unrelenting microvascular vasoconstriction results in bowel ischemia in the presence of pulsatile macroscopic arterial blood flow^[1]. Apart from a variety of known risk factors and associated conditions, NOMI has been repeatedly reported as a rare cause of abdominal emergency in hemodialysis patients and more recently in the continuous ambulatory peritoneal dialysis (CAPD) patients^[2-5]. We describe an end stage renal failure (ESRF) patient who underwent surgery for an unusual episode of acute abdomen secondary to NOMI while she was on a CAPD program.

Case report

A 45-year-old woman with ESRF due to unknown etiology on CAPD was admitted to the Emergency department with intense sharp pain in the hypogastrium. Her heart rate was 113/min, blood

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pressure 50/30 mmHg and respiration rate 20/min. Abdominal examination revealed generalized tenderness with guarding but no rebound. The blood leukocyte count was 12800/mm³. The leukocyte count at the abdominal lavage was 440/mm³.

We hospitalized her with an initial diagnosis of peritonitis presenting with abdominal pain and increased white cell count in peritoneal fluid, and switched to hemodialysis because of the drainage problems. During this period, she received IV fluid and antibiotics (metronidazole qid 500 mg IV, ceftazidime 1.5 g once every two days). Over the next 3 days, her general condition improved and her abdominal pain subsided. Culture of the peritoneal fluid at the time of presentation was negative and a diagnosis of culture negative peritonitis was made. The leukocyte counts at the peritoneal lavage fluid were around 1400–4300/mm³. Resistance to initial empirical therapy was suspected. Abdominal ultrasound revealed localized fluid collections among dilated aperistaltic small bowel loops.

The sudden onset of intense abdominal pain together with guarding and rebound recurred on fifth day. The leukocyte count in the peritoneal lavage fluid rose to 12 400/mm³, and fluid drained through the CAPD catheter revealed intestinal contents. She had an urgent laparotomy, at which generalized peritonitis characterized by oedematous, soiled and fibrin-covered peritoneal surfaces was demonstrated. The intestines were dilated and oedematous. There were numerous, irregular shaped and sized infarcted areas in the terminal ileum within 10 cm of the ileocaecal valve, of which a few were already perforated and leaking intestinal contents. The infarcted areas had a characteristic patchy appearance. We resected the infarcted segment, and brought up the proximal end as an ileostomy, and irrigated and drained the abdominal cavity prior to closure of the wound. Histopathology demonstrated a variable inflammatory cell infiltrate in all layers of intestine, and mucosal ulceration along with epithelial necrosis and submucosal exudation. There was also segmental serosal and muscle necrosis with perforation.

The patient had an uneventful recovery in the early postoperative period. Her blood pressure was low in the first two days, but rose from 110/70 to 120/80 mmHg levels thereafter. She was discharged on the 30th day. She was well at her 3-month follow up. We intend to close her jejunostomy at 6 months postoperative.

Discussion

NOMI makes up a small percentage of all the causes of acute mesenteric infarction^[6]. In their retrospective review Howard *et al.* identified 113 patients with acute mesenteric ischemia, of whom 13 (12%) were nonocclusive in nature^[7]. Reports have shown with increasing frequency that nonocclusive bowel infarction is one of the many causes of morbidity and mortality in hemodialysis patients^[1-3, 8]. Patients on CAPD may be at risk from various gastrointestinal complications^[9]. NOMI is a rare one of these complications, however its incidence is not yet known^[4, 5]. This was the first case of NOMI we diagnosed among our 75 cases on the CAPD program.

In a patient who is on hemodialysis or CAPD, an accurate preoperative diagnosis of NOMI requires a high index of suspicion. Howard *et al.* has grouped the NOMI patients into early and late presenters, of which early presenters were women younger than 50 years of age, and had vague symptoms leading to a delay in diagnosis^[7]. Previous studies have identified some characteristics of NOMI in dialysis patients. In almost all, hemodialysis-induced hypotension was the most frequent finding, which should probably be termed as an aetiological factor for NOMI development^[1, 5]. In their retrospective study on 29 NOMI cases John *et al.* pointed out that the majority of patients experienced abdominal pain more than 24 h before admission^[2]. The additional risk factors in their study included hypertension (83%), diabetes (55%) and atherosclerosis (38%). 66% of their patients had had a leucocytosis on admission. Dahlberg *et al.* emphasized that all of their 6 dialysis patients with NOMI had had leukocyte counts of greater than 12 000 on admission, and 4 had stools positive for occult blood^[10]. The clinical presentation of our case coincided well with those reported before. She had a period of resistant hypotension prior to the onset of vague pain, which lasted quite a long time before proceeding to an acute abdomen.

The pathophysiological mechanisms of NOMI remain unknown. Diamond *et al.* suggest the possible role of volume reduction and of splanchnic vasoconstriction induced by drugs like digitalis, vasopressors, or beta-blockers^[1]. Our case did not have any history of drug treatment that might have predisposed her to NOMI. Dueymes *et al.* demonstrated volume contraction resulting from excessive fluid loss in 10 of 15 episodes that they have observed^[8]. However, in 5 cases, neither abnormal fluid loss nor splanchnic vasoconstriction was present. The records of the present case did not reveal any significant factor regarding the pathogenesis of NOMI. The

early diagnosis of NOMI is most unlikely among frequent episodes of CAPD-associated peritonitis, and vague symptoms. The early diagnosis of NOMI is most unlikely. Emphasis should be given to dialysis-induced hypotension followed by nonspecific abdominal symptoms and leucocytosis. NOMI should be considered in the differential diagnosis of peritonitis in patients resistant to initial empirical antimicrobial therapy.

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