The Interesting Case

Ischaemic bowel disease in patients on continuous ambulatory peritoneal dialysis

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Introduction

Peritoneal signs in patient on continuous ambulatory peritoneal dialysis (CAPD) are usually due to CAPD-associated peritonitis without or with bowel perforation. Ischaemic bowel disease (IBD), however, is a condition that must be considered in the differential diagnosis. It is an acute mesenteric catastrophe, which can be mistaken for CAPD-associated peritonitis because of their similar clinical features [1]. IBD can be classified as occlusive or non-occlusive [2]. Non-occlusive mesenteric infarction was first described by Ende in 1958 in non-renal patients with cardiac failure [3]. In recent years, however, a haemodialysis-induced hypotensive episode has been a common finding associated with non-occlusive mesenteric infarction [4]. Possibly because of the lower risk of hypotensive episodes, it has rarely been reported in CAPD patients. We observed two uraemic patients on CAPD, who developed IBD.

Cases

Case 1

A 71-year-old female with end-stage renal disease (ESRD) secondary to diabetic nephropathy had been on maintenance CAPD therapy for 14 months. There was a history of coronary artery disease (3-vessels-disease) with myocardial infarction and hypertension. She suffered from congestive heart failure and unstable angina. Her medication included recombinant human erythropoietin (rHuEpo) 4000 U s.c. weekly, aspirin 100 mg q.d., isosorbide dinitrate 10 mg t.i.d., nifedipine (Coracten®) 20 mg b.i.d., and potassium chloride (Slow-K®) 8 mmol b.i.d. When excessive fluid accumulation with facial and leg oedema developed, she had more frequent exchange of high-glucose concentration dialysate (4.25% Dianeal®). She was admitted because of epigastric pain with bloody stool, poor appetite, and general weakness. Her vital signs revealed a blood pressure (BP) of 63/48 mmHg (the baseline BP was around 150/80 mmHg); pulse rate, 70 beats/min; body temperature, 38.7°C, and CVP, −1 cm H₂O. The patient’s abdomen was soft and non-tender with normal bowel sounds. Peritoneal effluent was clear, without leukocytosis, and the culture of drainage fluid grew no pathogens. The exit site was clean without erythema or purulent discharge, there was no tenderness over the tunnel tract. The haemogram showed WBC: 14 000/mm³ with left-shifting, and haemoglobin, 7.5 g/dl. Biochemistry showed serum amylase, 30 U/l; Na, 127 mmol/l; K, 2.1 mmol/l; bicarbonate, 18.7 mmol/l; and arterial blood pH, 7.36. Hypovolaemic shock combined with sepsis was suspected and the patient was treated with saline and antibiotics. Unfortunately high fever and cloudy dialysate set in rapidly. The culture of the dialysate grew Pseudomonas aeruginosa and Klebsiella pneumoniae. The amylase level in peritoneal fluid was 157 U/l. Blood culture yielded P. aeruginosa and oxacillin-resistant Staphylococcus aureus. The antibiotics were changed and intravenous vancomycin, ciprofloxacin, and metronidazole were administered. Abdominal computed tomography (CT scan) showed pneumatosis intestinalis (Figure 1). Exploratory laparotomy revealed infarction extending from jejunum to transverse colon. No vascular occlusive lesion was found and no resection attempted. The patient’s condition rapidly deteriorated and she died on the first postoperative day.

Case 2

A 40-year-old male with ESRD of unknown aetiology, and acquired renal cystic disease, had been well
on CAPD therapy for 7 years. Two episodes of CAPD-associated peritonitis by coagulase-negative Staphylococcus had occurred in February 1990 and March 1997. The peritonitis resolved completely after treatment. In August 1997, the patient suffered from fever, diffuse abdominal pain and tenderness 2 days before admission. Plain film of the abdomen disclosed no subdiaphragmatic free air accumulation. Peritoneal effluent showed pleocytosis and the Gram stain revealed Gram-negative bacilli. CAPD-associated peritonitis was diagnosed, and he was initially treated with intraperitoneal cephalothin (Ulothin®) and gentamycin. Peritoneal signs and cloudy dialysate did not improve after 2 days’ antibiotic therapy. Baseline BP was around 130/80 mmHg and decreased to 70/50 mmHg. Laboratory data showed a WBC count of 5400/mm³ with a shift to the left; serum amylase, 55 U/l; bicarbonate, 21.2 mmol/l; and arterial blood pH, 7.36. The dialysate effluent was cloudy with an amylase level of 30 U/l (serum amylase, 80 U/l). Repeated plain films of the abdomen showed dilated loops of the small bowel with air–fluid levels. The culture of dialysate eventually grew *P. aeruginosa*. Abdominal CT scan showed thickened small-bowel wall (Figure 2). IBD was suspected. During laparotomy, turbid ascites, foul odour and an extensive gangrenous change of the small bowel were found. No resection was performed. The patient died on the first postoperative day.

**Discussion**

The small intestine is supplied by the coeliac and superior mesenteric arteries, and the colon is supplied by the branches of the superior and inferior mesenteric arteries. Because multiple collateral vessels are available throughout the alimentary tract except the small bowel and colon, IBD affects almost always the small bowel and colon, as observed in our cases [5]. The clinical manifestations of IBD range from chronic, mild symptoms to catastrophic events, depending on the segment involved, the degree of involvement, and the rapidity of the process. The most common manifestation of IBD is abdominal pain, which may be mistaken as CAPD-associated peritonitis, as happened in our cases. According to the literature, the duration of abdominal pain prior to the establishment of the correct diagnosis ranges from 12 h to 11 days [5]. Diarrhoea and a positive test for faecal occult blood or even overtly bloody stool are the result of prolonged bowel ischaemia and necrosis [6]. Leukocytosis, fever, and signs of peritonitis develop when ischaemia persists and infarction develops.

IBD may be classified as occlusive or nonocclusive. In approximately one-half of patients with mesenteric ischaemia, one does not find occlusion of a major vessel. This condition is then referred to as ‘non-occlusive ischaemia’ resulting from low blood flow [7]. It is clear that the prevalence of vascular disease is higher in patients with ESRD, because of the high frequency of risk factors, such as diabetic microangiopathy, hypertension, hyperlipidaemia, etc [8]. Non-occlusive mesenteric infarction is usually found in haemodialysed patients, and this is presumably explained by the high frequency of hypotensive episodes [9]. It appears to be rare in patients on CAPD, although patients undergoing CAPD are also at high risk of IBD for several reasons. Firstly, the majority of patients are elderly. Secondly, many patients with ESRD have hypertension and advanced organic heart disease [9]. Thirdly, these patients also routinely undergo the removal of large amount of fluid. Although blood pressure tends to be more stable on CAPD, patients may have severe hypotension [10]. Some factors contribute to hypotension in CAPD patients: hypovolaemia from excessive fluid removal because of inappropriate use of hypertonic dialysate, administration of diuretics, or adherence to a very low
sodium intake [10,11]. Marquez-Julio et al. [11] showed that low aldosterone concentration resulted in decreased colonic absorption of sodium. They proposed that removal of aldosterone in the dialysate exposed the patient to the risk of hypovolaemia.

Factors contributing to IBD include: (i) congestive heart failure, valvular heart disease, cardiac arrhythmia (particularly atrial fibrillation) [12]; (ii) oral oestrogens, possibly due to their thrombogenic properties [13]; (iii) Digitalis treatment also may cause vasocostriction of the splanchnic vessels [14]; (iv) erythropoietin therapy. Epo therapy has a direct vasopressor effect on small mesenteric vessels in vivo [15]. The laboratory investigations in IBD are non-specific. The presence of clear dialysate in a CAPD patient with abdominal pain is not a reliable sign of a benign process. When mesenteric ischaemia has not yet led to bowel infarction, the mucosal and serosal layers may still be intact so that the dialysate may remain clear even when bloody diarrhoea is present (as in Case 1) [16]. Although hyperamylasaemia (> 300 U/l) is common in asymptomatic CAPD patients, including those patients with peritonitis, a major increase of amylase in serum (> 600 U/l) and peritoneal effluent (> 100 U/l) is a useful help in the evaluation of a CAPD patient with abdominal symptoms [17]. We cannot explain the low level of amylase in the serum and peritoneal effluent in our patients other than to speculate that it was the result of an intact serosa of the small bowel. Abdominal plain film may reveal air–fluid level and bowel loop dilatation. Barium study of the small intestine may show non-specific dilation, poor motility, and thickened mucosal folds (thumb-printing) [18].

Should angiography be undertaken in a patient with acute abdominal pain of suspected ischaemic origin? Not everyone agrees with this approach except in patients with atrial fibrillation or other conditions predisposing to embolization [19]. The abdominal CT scan typically shows thickening of the bowel wall with oedema and fluid accumulation. When the bowel wall is necrotic, air gains entrance to the suberosal space giving the appearance of a string or ring of gas outside the bowel lumen, i.e., intramural pneumatisos (as in our Case 1), or even lead to air in portal vessels [18]. Bowel ischaemia is associated with a disequilibrium of the enteric flora and predisposes to proliferation of anaerobic bacteriae. When the integrity of the intestinal mucosa has been disrupted, bacterial invasion of the blood stream and leakage into peritoneum may occur. Successful management depends on early recognition of this syndrome. In one study, all of the survivors were operated within 24 h [6].

The prognosis of IBD is grave. Clinical findings of advanced bowel infarction usually develop late [20]; these are diffuse peritoneal signs, elevated serum level of lactic dehydrogenase, and lactate, persistent metabolic acidosis, disseminated intravascular coagulation and refractory shock (as in our cases). Although resection is technically feasible, the course of advanced infarction is usually not altered by surgery [21]. In conclusion, although IBD is relatively rare in patients on CAPD, it should be considered in the differential diagnosis of CAPD patients with abdominal symptoms, because in IBD the success of intervention depends on early diagnosis.

References