Two haemodialysis patients with unclear abdominal symptoms of similar origin

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Introduction

Ischaemic colitis (IC) is a dangerous condition usually caused by atherosclerosis and low blood flow states [1]. Haemodialysis (HD) patients are at increased risk of IC because they have accelerated rates of arterial vascular disease [2] and because hypotension is common during dialysis with large amounts of fluid removal. Thus, IC in the HD patient is usually attributed to atherosclerotic origin. However, there are many other conditions that can contribute to IC including arterial occlusion, thrombosis, factors that increase blood viscosity, constipation causing increased intraluminal pressure [3,4], digoxin which acts as a splanchnic vasoconstrictor, impaired baroreflex sensitivity in chronic renal failure, and, rarely, neoplasm [5,6] or amyloidosis [7–10].

We report two cases of IC in HD patients of nonatherosclerotic aetiology. The first case is a unique presentation of IC in a HD patient in association with previously undiagnosed non-Hodgkins lymphoma encroaching on mesenteric vessels. The second case is a HD patient with amyloidosis who presented with and subsequently died from IC and was found to have extensive amyloid involvement of the colon evident on post-mortem examination.

Case 1

An 80-year-old male with end-stage renal disease that developed after abdominal aortic aneurysm repair was transferred to our facility from an outside hospital with lower gastrointestinal bleeding. The patient had a 2-week history of diarrhoea without blood that preceded an episode of bright red blood per rectum the night before admission. Colonoscopy showed diffuse ischaemic appearing bowel. An atherosclerotic aetiology of IC was assumed and surgical consultation determined that the patient did not require surgical intervention.

During his hospital course, the patient complained of a dry cough and dysphagia that eventually led to a CT scan of his chest. Incidentally noted was a large mass around the pancreas that was further evaluated with CT scan of his abdomen and pelvis. The peri-pancreatic mass measured 9.0×13.5×16 cm and encased the superior mesenteric artery and vein, parts of the duodenum, the right renal artery and left renal vein and most of the inferior vena cava (Figure 1). Diffuse atherosclerotic disease was also noted. CT-guided biopsy revealed low-grade non-Hodgkin’s lymphoma.

MR angiography of the celiac axis was performed to determine if the mass was impinging on the mesenteric vessels and contributing to ischaemia of the colon. As shown in Figure 2, there was ectasia with band-like stenosis at the origin of the superior mesenteric artery with no significant flow beyond a point 2 cm distal to the stenosis. This corresponded with his area of ischaemic colitis. Upon treatment of his lymphoma with chemotherapy, the mass shrunk significantly and he had no further episodes of IC.
Case 2

A 50-year-old female with end-stage renal disease secondary to renal amyloidosis from ‘skin popping’ drug abuse was on HD for 3 years. She was admitted with a 3-day history of vomiting and severe abdominal pain. Her vital signs were initially stable; her examination revealed a firm tender abdomen and her serum lactate was 5.7 mEq/l. A naso-gastric tube was placed and 2 l of feculent material was removed. The patient was taken to the operating room for exploratory laparotomy that showed multiple adhesions but no perforation or evidence of ischaemia of the small or large bowel. Over the next 4 days, the patient developed fever and hypotension resistant to vaso-suppressors. Her lactate level rose from 5.7 mEq/l to 11.9 mEq/l and her condition continued to worsen. She expired on day 5. Post-mortem examination revealed large and small bowel with diffuse mucosal ischaemia, necrosis, and amyloid-associated protein deposition in small blood vessels of the submucosa (Figure 3).

Discussion

Ischaemic colitis in dialysis patients is usually a result of atherosclerosis and superimposed hypotensive episodes. Patients who have developed chronic renal failure usually have extensive atherosclerosis as a result of diabetes mellitus, hyperlipidaemia, and hypertension. In these patients, any condition that causes a low blood flow state can contribute to the development of IC. Even short periods of hypotension during dialysis have been shown to lead to vasoconstriction and ischaemia [11]. Poor perfusion of bowel can also be a result of shock, congestive heart failure, myocardial infarction, arrhythmia, valvular disease and vaso-constriction caused by vasopressors and digitalis. Many of these comorbid conditions exist in HD patients placing them at increased risk for IC.

Not only are HD patients predisposed to develop intestinal ischaemia, there are numerous case reports suggesting that these patients may suffer from more severe cases that are often associated with complications such as colonic necrosis and rupture [4,12,13]. A recent study by Flobert et al. [14] attempted to identify factors predictive of a poor outcome in patients with IC. Involvement of the right colon was an independent predictor of poor outcome; chronic renal failure, HD, and short time period from development of symptoms and diagnosis were ‘significantly more common in the patients with severe colitis’. Ischaemia of the right

Fig. 1. Computed tomography scan of the patient’s abdomen revealing a large peripancreatic mass (arrow) encasing the superior mesenteric artery and vein, parts of the duodenum, the right renal artery and left renal vein and most of the inferior vena cava.

Fig. 2. Magnetic resonance angiography of the aorta and superior mesenteric artery (SMA) demonstrating no flow beyond 2 cm distal of the stenosis at the origin of the SMA (arrow).

Fig. 3. Immunoperoxidase staining using a specific monoclonal antibody to amyloid associated (AA) protein demonstrates amyloid in the blood vessels of the submucosa (arrows).
colon has been reported in other severe cases of IC in HD patients [4,12].

Although it is not understood why the right colon seems to be preferentially involved in HD patients, it is thought that there is less collateral flow and the vasa recta originate farther away from the bowel on the right side predisposing the right side to non-occlusive ischaemia [14]. Other reports have cited involvement of the sigmoid colon when colonic rupture occurred [3,15]. One might expect the transverse and descending colon around the area of the splenic flexure to be involved more frequently since it is a watershed area. Perhaps the nature of the vascular supply to the right colon plays a more important role in sensitivity to haemodynamic changes than the watershed phenomenon at the splenic flexure. Herein may lie the reason that dialysis patients seem to have increased involvement of the right colon given their propensity for hypotension during HD.

Although atherosclerosis and hypotension are the usual causes of low blood flow states, there are a few case reports of rare causes of vascular compromise secondary to neoplasia that contributed to ischaemic bowel disease. Hsiao et al. [5] published a case series of IC cases related to angiocentric T cell lymphoma. Histopathological examination showed ‘angiocentric and angiodestructive infiltration by atypical lymphocytes’ that presumably caused bowel ischaemia. Similarly, Singh et al. [6] reported a case of lymphomatoid granulomatosis that caused thickened mesenteric arteries with intimal fibrosis and lumen narrowing and subsequent IC. While rare, in atypical presentations of IC, it is important to keep unusual causes in mind, especially in very young patients with IC.

The two patients described in this report also had rare causes of ischaemic bowel disease. Case number one is a unique report of non-Hodgkin’s lymphoma encroaching on mesenteric vessels causing vascular compromise and ischaemic bowel. To our knowledge, this has not been reported before. This patient had a fairly typical presentation of IC after hypotension on a dialysis run and he had numerous risk factors for atherosclerosis. There was no reason to suspect any other cause for ischaemia in his case. In fact, Figure 1 shows probable atherosclerosis of the very proximal portion of the superior mesenteric artery. However, there clearly is flow after that lesion which is then attenuated 2 cm distal to the take-off from the aorta representing the mass effect. In this case, identification of the lymphoma and subsequent treatment shrank the mass and decreased the likelihood of recurrence of IC.

The second case is a patient with end-stage renal disease secondary to amyloidosis who developed IC. Amyloidosis affects many organ systems; heart disease and renal failure are two major causes of death in these patients. There have been previous case reports of HD patients with IC colitis from amyloid involvement [7–10]. Typically, the mucosa and submucosa are involved and the vessel walls have high amyloid-associated protein levels [8,10]. The histopathological examination of our patient is consistent with these prior reports. Presumably, involvement of small blood vessels leads to physical obstruction of blood flow or functional impairment of the vessels with resultant hypoperfusion of bowel. In patients known to have amyloidosis, it is important to understand the potential complications from multi-organ system involvement. If there is known bowel involvement in patients with amyloidosis, particular attention should be paid to avoiding hypotension on dialysis runs.

These two cases demonstrate the importance of considering non-atherosclerotic aetiologies of IC in the HD patient. Identification of the aetiology, as evidenced in our first patient, changed outcome.

Teaching points

(i) In dialysis patients with unclear abdominal symptoms, consider ischaemic colitis.

(ii) Ischaemic colitis can be caused not only by atherosclerosis, but also by amyloidosis of the vessels or lymphomas.

References