A 34-year-old man with recurrent melaena after renal transplantation

Abdul K. Cariem, Mike Arendse, Peter Cruse, Brian Rayner

Case presentation

The patient a 34-year-old Asian man, was an unemployed projects engineer whose medical history had begun in 1982 when he was diagnosed as having IgA nephropathy on renal biopsy after an episode of loin pain and haematuria. Initially renal function was normal, but over the ensuing years it gradually deteriorated and by April 1992 he was in end-stage renal failure requiring haemodialysis. During this period he required treatment for moderately severe hypertension, and underwent highly selective vagotomy and pyloroplasty for an intractable duodenal ulcer in 1991. After the operation he developed recurrent bouts of watery diarrhoea. These were episodic, and he passed about 30 stools per day. There was no abdominal pain, nausea, vomiting or loss of appetite. He had lost about 3 kg since surgery. There were no identifiable precipitating factors. His medication was enalapril 10 mg and atenolol 50 mg daily.

On examination the patient had pallor of the nails but no jaundice, lymphadenopathy or features of thyrotoxicosis. His abdomen was soft with no masses or visceromegaly. A midline surgical scar was present. Rectal examination was normal and the stool was negative for occult blood. The rest of the examination was unremarkable except for mild displacement of the apex beat and a blood pressure of 170/105 mmHg.

The following investigations were done: full blood count — haemoglobin 8.1 g/dl, mean corpuscular volume 88/µl, white cell count 7.82 x 10^9/l, platelets 227 x 10^9/l, erythrocyte sedimentation rate 40 mm/1st h, normal differential count; biochemistry — Na^+ 137 mmol/l, K^+ 5.5 mmol/l, urea 29.1 mmol/l, creatinine 1 026 µmol/l; B_{12} and folate — normal, stool — no red blood cells (RBCs), white blood cells or parasites, culture negative; fasting gastrin 132 pg/ml (normal 0 - 115 pg/ml); 72-hour stool — faecal fat normal, mass 208 g (normal < 200 g); upper gastro-intestinal endoscopy — normal; duodenal and rectal biopsies — normal; small-bowel enema — normal; sigmoidoscopy to 20 cm — normal; ultrasound examination of abdomen — normal; thyroid-stimulating hormone — normal; 14^C xylose — not suggestive of bacterial overgrowth; lactose tolerance —

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Clinical Discussant: Abdul K. Cariem, M.B. CH.B., F.C.P.

Pathologist: Mike Arendse, B.SC. (MED.) HONS. M.SC. (MED.), M.B. CH.B., M.MED. (PATH.)

Chairperson: Peter Cruse, M.B. CH.B., PH.D., F.R.C. PATH.

Editor: Brian Rayner, M.B. CH.B., F.C.P., M.MED.

grossly abnormal H_2 breath test; glucose tolerance test — fasting 5.1 mmol/l, 1/2 hour 4.5 mmol/l, 1 hour 6.8 mmol/l, 1 1/2 hours 4.5 mmol/l, 2 hours 4.8 mmol/l.

The patient's symptoms of diarrhoea subsided on a lactose-free diet, but he was re-admitted 1 month later with post-prandial vomiting of coffee-ground material, melaena, epigastric discomfort, fatigue, malaise and dizziness. On examination he was markedly pale, there was some epigastric tenderness and the stool was positive for blood. The haemoglobin concentration was 5.5 g/dl, and 2 units of packed cells were transfused. Upper gastro-intestinal endoscopy was normal and barium meal examination showed surgical clips at the oesophageal-gastric junction and features in keeping with an old duodenal ulcer. An RBC scan suggested duodenal or jejunal blood loss. Gastroscopy was repeated with a colonoscope, but this was also normal. A repeated RBC scan showed no further bleeding 1 week later. Maintenance dialysis was commenced after this episode.

In August 1992 the patient underwent a successful cadaver renal transplant. He received standard triple-therapy immunosuppressive treatment (steroids, azathioprine and cyclosporin), with cyclosporin being withdrawn at 6 months. At the time of surgery possible nodes were seen in the right iliac fossa; biopsy revealed extensive sinus histiocytosis. In November 1992 investigations for headache, macroscopic haematuria and fever were done. The macroscopic haematuria was attributed to recurrent IgA nephropathy in the transplanted kidney, and the headache and fever settled without specific treatment.

Between August 1994 and April 1995 the patient had six episodes of melaena requiring admission to hospital and transfusion. There was no obvious precipitating event and he only experienced symptoms related to blood loss. The melaena usually subsided after a few days. He had some dyspeptic symptoms which usually settled with symptomatic treatment with antacids. There was no loss of weight, loss of appetite, abdominal pain, or change of bowel habit except during periods of melaena. A chest radiograph done in December 1994 showed a nodular infiltrate in the right upper lobe, but the patient had no cough, sputum, fever or night sweats and this was not investigated further. His medication was prednisone 7.5 mg daily, azathioprine 100 mg daily and several antihypertensive drugs. Renal function had been declining progressively owing to chronic rejection, and by March 1995 he once more required haemodialysis. Haematological investigation showed no bleeding diathesis, liver function tests were normal, and cytomegalovirus titres were always IgG-positive and IgM-negative. The following gastro-intestinal investigations were all normal: gastroscopy (4 times), colonoscopy (twice), sigmoidoscopy, and angiography (twice). An RBC-labelled scan suggested blood loss from the terminal ileum during March 1995.

Differential diagnosis

Dr Abdul Cariem: The case under discussion is that of a young man who, after renal transplantation, was admitted on six occasions over an 8-month period with episodes of life-threatening gastro-intestinal haemorrhage, presenting as episodes of melaena. None of these episodes were accompanied by haematemesis, and in addition 4 gastroscopies were reported as normal. We are not informed whether or not altered blood was present in the stomach, which, in the absence of a mucosal lesion, would have raised the possibility of haematobilia or bleeding from the pancreas. However, the patient does not have the medical background to place him at risk of these complications, i.e. a history of blunt or penetrating injury to the liver or pancreatitis. With repeatedly normal upper endoscopy (including one performed using a colonoscope, though we are not informed how successful this was and to what extent the proximal small bowel was examined), 2 negative colonoscopies and 2 unhelpful abdominal angiographies, it would therefore appear that the source of bleeding is localised in the mid- or distal small bowel. This conclusion is supported by a labelled RBC scan which suggested blood loss from the terminal ileum.

Routine investigations having been negative, the patient can be labelled as having gastro-intestinal bleeding of obscure origin. Small-bowel lesions described[1] as presenting in this manner are outlined in Table I.

Table I. Small-bowel lesions causing gastro-intestinal bleeding of obscure origin

Meckel's diverticulum
Enteric duplication cyst
Vascular anomalies — arteriovenous malformations, haemangiomas
Vasculitis
Neoplasms
Zollinger-Ellison syndrome
Crohn's disease
Tuberculosis
Syphilis
Typhoid fever
Histoplasmosis
Mesenteric ischaemia
Coeliac disease
Chronic ulcerative jejunitis
Drugs (Slow-K, 6-mercaptopurine)

In a series of 131 patients reported by Thompson *et al.*,[2] with obscure gastro-intestinal blood loss, 106 of whom were assessed, the modes of presentation were melaena in 55 patients, anaemia in 35, rectal bleeding in 34, haematemesis in 6, and bleeding from an ileostomy in 1. In patients who had a small-bowel source for the blood loss vascular anomalies were the commonest lesions (16 patients), followed by Meckel's diverticula (9) and leiomyomas (7). The remaining 9 patients with small-bowel lesions had, in addition to angiodysplasia, Meckel's diverticulum (4 patients) and with vascular anomaly, ulcerated duodenal diverticulum, solitary jejunal ulcer, chronic pancreatitis, and Crohn's disease (1 each). Unless active bleeding is observed from a site where more than one lesion is present, it may be difficult to decide which is the source of blood loss. Let us now look at this patient's medical background for clues as to the nature of the distal small-bowel lesion.

He first came to medical attention in 1982 at the age of 21 years when he presented with an episode of loin pain and haematuria. The diagnosis of IgA nephropathy was confirmed on renal biopsy. Renal function deteriorated gradually over the next 10 years, and by 1992 he required

haemodialysis. His case is typical of IgA nephropathy in terms of age of presentation and sex. IgA nephropathy may present as asymptomatic microscopic haematuria or as macroscopic haematuria. It is characterised by mesangial deposition of IgA immunoglobulins, and in this way bears similarities to Henoch-Schönlein purpura. This latter condition is characterised by multisystem involvement, and abdominal pain and gastro-intestinal blood loss is well described as a manifestation.[3] Although deposits of IgA have been demonstrated in skin capillaries in patients with IgA nephropathy, it does not involve any other organ system, and the distal ileal lesion cannot be related to this disease.

In 1991 the patient underwent highly selective vagotomy and pyloroplasty for intractable duodenal ulcer disease. No details are given regarding the medical management of the peptic ulcer disease and the reasons for resorting to surgery; also, a drainage procedure is not usually required with a highly selective vagotomy. A short while later he presented with diarrhoea and after a series of investigations, mostly negative except for a grossly abnormal lactose tolerance test, was found to have lactose intolerance. The prevalence of lactase deficiency is high in adults, particularly those of Asian and African descent.[4] The rapid gastric emptying following the surgery resulted in an overload of the available lactase and secondary lactose intolerance. The diarrhoea responded to a lactose-free diet.

One month later the patient presented with coffee-ground vomitus, melaena, and symptomatic anaemia; the haemoglobin concentration had fallen to 5.5 g/dl. Upper endoscopy was normal and barium meal examination merely showed evidence of scarring of old duodenal ulcer disease. A labelled RBC scan suggested duodenal or jejunal blood loss, but a lesion could not be demonstrated. The incidence of recurrence of peptic ulcer disease is higher after surgery for duodenal ulcers than after surgery for gastric ulcers, and with a highly selective vagotomy there is a 7 - 10% relapse rate.[5] There is also a lower incidence of pain and a higher incidence of haemorrhage as the presenting symptom. It is also possible to miss a small lesion, both on endoscopy and on barium study, in a patient with a scarred and distorted duodenum. None of the patient's subsequent presentations with melaena were associated with coffee-ground vomitus, and with 4 normal upper endoscopies and presumably no evidence of blood in the stomach it is very unlikely that the episodes of melaena were due to recurrent duodenal ulcer disease.

The next possibly relevant factor we need to consider is that of renal failure and renal transplantation. The patient had a renal transplant in August 1992 with evidence suggesting recurrence of the IgA nephropathy in the transplanted kidney 3 months later. An increased incidence of occult gastro-intestinal blood loss has been demonstrated in patients with impaired renal function and in those on haemodialysis. This has been documented at 0.83 ml/day in normal controls, 3.15 ml/day in uraemic patients (thought to be due to impaired platelet function), and 6.25 ml/day in patients on haemodialysis (with the use of anticoagulants an added factor).[6] Although we have excluded peptic ulcer disease as the cause of our patient's episodes of melaena, it may be useful to note that, contrary to popular perception, recent studies where endoscopy was used as the mode of investigation, showed the prevalence of peptic ulcer disease in patients with renal failure to be the same as that in the general population.[7] An increased prevalence has been noted in transplant patients, particularly during the first 5 months after transplantation, with a higher incidence of duodenal ulcers and a lower incidence of gastric ulcers in transplant patients compared with patients on dialysis. A poor correlation with symptoms has also been noted.[8]

Of greater relevance in this patient is the association between uraemia and gastro-intestinal telangiectasia. Results are conflicting, some studies reporting a low prevalence of gastro-intestinal telangiectasia in uraemic patients with gastro-intestinal haemorrhage, while others report a 24 - 32% incidence of vascular malformations in uraemic patients with gastro-intestinal haemorrhage.[9,10] Conversely, in a group of 65 patients with upper or lower gut angiodysplasia, 35% had renal insufficiency.[11] In the latter group 65.2% of the lesions were found proximal to the ligament of Treitz. In the patient under discussion, failure to demonstrate angiodysplastic lesions on upper and lower endoscopy makes this an unlikely cause of his bouts of melaena, though theoretically it is possible for the lesions to be localised to the area of the small bowel not visualised. Another confounding problem is whether the finding of angiodysplasia on endoscopy means that it is the cause of bleeding. In one study 22.5% of patients with right-sided colonic angiodysplasia were subsequently found to be bleeding from a small-bowel lesion.[12]

This patient had his first episode of melaena almost 2 years after renal transplantation. He was on immunosuppressive therapy in the form of steroids and azathioprine, having received cyclosporin during the first 6 months only. He was admitted with episodes of melaena on 6 occasions over an 8-month period, and towards the latter part of that period haemodialysis was recommenced owing to recurrence of IgA nephropathy in the transplanted kidney. We are not told at what stage immunosuppressive therapy was discontinued. Nevertheless, there is a 3 - 10 times increased risk of *de novo* malignant tumours after renal transplantation. This is seen both in patients who received only steroids and azathioprine and in those receiving the more recent cyclosporin-based immunosuppressive regimens. These tumours appear to present earlier in patients receiving cyclosporin, means of 36 and 36.2 months after transplantation versus 95 and 75 months, respectively, as has been reported by two groups[13,14] recently. In reports from Germany and Spain[13,14] the skin appears to be the most common site (16 of 31 cases and 11 of 26 cases, respectively), whereas a report from Japan[15] found the gut and liver most commonly involved (5 and 6 cases, respectively, of a total of 22). The incidence of malignant lymphoma was found to be low in all three series — 2 out of a total of 79 cases. Again, although theoretically it is possible for our patient's melaena to be caused by a malignant lesion in the distal small bowel, in view of the short duration of his immunosuppressive therapy we should consider other possibilities.

The last piece of information we are given is a possible abnormality on the chest radiograph midway through the patient's 6 admissions with melaena. On review of the relevant radiographs a nodular infiltrate in the right upper zone

adjacent to the mediastinum is clearly evident. There is no evidence of any breakdown, and no hilar lymphadenopathy.

Opportunistic pneumonias after renal transplantation tend to occur in the first 1 - 6 months after transplantation. The organisms involved include cytomegalovirus, *Pneumocystis pneumoniae,* fungi such as *Aspergillus, Cryptococcus* and *Candida,* and Gram-negative bacilli such as *Listeria* and *Nocardia*.[16] The characteristics of the chest radiograph are a vital clue to the aetiological agent. In this patient — 2 years after renal transplantation, immunocompromised on the basis of immunosuppressive therapy as well as impaired renal function, with a focal nodular infiltrate not characteristic of the above infective agents, and no associated respiratory symptoms — tuberculosis must be considered the most likely cause of the infiltrate seen on the chest radiograph, particularly bearing in mind the high local prevalence of this disease.

An increased incidence of tuberculosis in patients with renal transplants due to uraemia and the use of immunosuppressants has been reported worldwide. The incidence varies with the local prevalence of the disease, ranging from 0.5 - 1% in North America and 1.0 - 4% in Europe to as high as 9.5% in India.[17] Locally, in an 11-year (1980 - 1990) review, 20 cases of tuberculosis occurred in 407 transplant recipients available for review, an incidence of about 5%.[18] The mean time from transplant to diagnosis of tuberculosis was 15 months (range 2 - 74 months). Consolidation on the chest radiograph was present in 13 patients, a miliary pattern in 4, pleural effusion in 3, cavitation in 1, hilar lymphadenopathy in 1, and a tuberculoma in 1 patient. Extrapulmonary tuberculosis occurred in 6 patients; 1 had meningitis, 1 renal involvement, and 4 disseminated tuberculosis.

Two 10-year reviews of gastro-intestinal tuberculosis have been reported from this hospital (1962 - 1971 and 1972 - 1981), a total of 242 patients (117 and 125 respectively).[19,20] A total of 95 patients (49 and 46 respectively) had intestinal tuberculosis, 129 tuberculous peritonitis, and 18 mesenteric node involvement. The commonest symptoms experienced by the patients with intestinal tuberculosis were weight loss (49/49 and 38/46), abdominal pain (43/49 and 37/46), diarrhoea (26/49 and 22/46) and vomiting (10/49 and 23/46). The chest radiograph was abnormal in 20/49 and 23/46 patients. In the second review,[20] of the 46 patients with intestinal involvement, 13 had small-bowel involvement. Five patients presented with malabsorption, of whom 2 had active pulmonary tuberculosis. Of the remaining 8 patients, 4 presented with obstruction, 2 with perforation, 1 with melaena, and 1 with pyrexia of unknown origin. The lesion was localised to the distal small bowel in 7 of these 8 patients, and 2 had active pulmonary tuberculosis.

Occult bleeding due to gastro-intestinal tuberculosis has been described in up to 39% of patients.[21] In the above-mentioned series of 46 patients with intestinal tuberculosis,[20] 14 had evidence of gastro-intestinal blood loss; this was occult in 13 cases, while 1 patient had melaena. However, in another 2 series describing 109 and 196 patients with gastro-intestinal tuberculosis,[22,23] there was no evidence of gastro-intestinal blood loss, though anaemia was common and occurred in up to 60% of patients.

Although overt gastro-intestinal bleeding due to intestinal tuberculosis may be rare, a number of isolated case reports have appeared describing massive life-threatening gastro-intestinal bleeding. This has complicated lesions in the stomach, small bowel and colon.[24-28]

In summary, we have a young man post-renal transplantation on immunosuppressive therapy and with renal impairment, with a pulmonary infiltrate consistent with pulmonary tuberculosis, presenting with repeated episodes of massive intestinal bleeding, localised to the distal small bowel on a labelled RBC scan. Although it is possible that he may have a totally unrelated intestinal lesion such as a Meckel's diverticulum, or an isolated small-bowel angiodysplastic lesion related to his renal impairment, or a malignant lesion related to his immunosuppressive therapy, I consider that the pulmonary infiltrate and the site of the intestinal lesion would favour intestinal tuberculosis as the most likely cause of the lesion and the blood loss. It is difficult to explain why the infection failed to become rampant over the 8-month period during which the patient experienced the episodes of melaena, in view of his immunocompromised state. I suspect that the immunosuppressive therapy was discontinued when it became evident that failure of the transplant kidney was inevitable, and not continued to the stage at which haemodialysis was recommenced.

The diagnostic procedure indicated, in view of the life-threatening nature of the bleeding episodes and positive localisation of the lesion with the labelled RBC scan, would be a laparotomy with a view to resection of the involved segment of bowel or biopsy to confirm the diagnosis, followed by medical therapy.

Dr Abdul Cariem's diagnosis. Ileal tuberculosis.

Operative findings

A diagnostic laparotomy was performed. At surgery there was found to be blood in the small bowel with multiple nodular lesions from ileum to jejenum. Mesenteric nodes were palpable. A small segmental resection of the ileum was performed.

Pathological findings

Two specimens were submitted. One consisted of a lymph node, 14 x 8 x 4 mm in its greatest dimensions. Cross-section of the lymph node showed caseous necrosis. The other was a full-thickness bowel biopsy specimen, 25 mm in length and 50 mm in circumference. A transverse ulcer was present, measuring 20 mm in diameter and 3 mm in length.

Microscopy

Sections taken from the ulcer show caseous granulomatous inflammation replete with Langerhans-type giant cells in the base of the ulcer. The inflammation extends transmurally with interruption of the external muscular layer. Sections taken adjacent to the ulcer show caseous granulomatous inflammation within the submucosa of the specimen (Fig. 1).

Sections of the mesenteric node show caseous granulomatous inflammation replacing the normal nodal architecture. Stains for acid- and alcohol-fast bacilli revealed stainable bacilli within Langerhans-type giant cells.

Fig. 1. A full-thickness biopsy of the terminal ileum shows features of granulomatous inflammation at the base of the ulcer.

Anatomical diagnosis. *M. tuberculosis* infection of the small bowel.

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