Hypoalbuminaemia and colonic polyps: a case of protein-losing enteropathy with cap polyposis?

Introduction

Cap polyposis is a rare condition affecting the distal colon. The disorder was first described in 1985 and has subsequently been reported in occasional case series. There has been one reported case where it has been associated with protein-losing enteropathy. This article describes a patient who presented with weight loss, altered bowel habit and low albumin over a 9-month period.

Discussion

Cap polyposis is a rare disorder affecting a wide age range. There is an equal male to female incidence. The condition presents with mucoid bloody diarrhoea, weight loss, and tenesmus. It may be mistaken for inflammatory bowel disease. There is often a preceding period of constipation. Macroscopically, sessile slug-like lesions are found in the rectosigmoid region, located on the crest of the mucosal fold (*Figure 1*). Microscopic appearances comprise elongated, tortorous and distended crypts. These are covered by a 'cap' of granulation tissue, leading to the condition's name.

Aetiology is incompletely understood. It is thought that mucosal prolapse coupled with abnormal rectosigmoid motility plays a role. Persistent mechanical stimulation, e.g. as a result of constipation, may lead to mucosal prolapse, hyperplasia of the mucosa and hypersecretion of proteins, reported by Konisi et al (2005). Ischaemia may be significant.

Differences have been isolated in the mucus, as reported by Busine et al (1998). There was a predominance of non-sulphated mucins, with abnormal expression of the MUC4, MUC3 and MUC5AC genes. These abnormalities have been noted in other pathological situations, suggesting they are a secondary phenomena. The mucin abnormalities reflect deregulation of expression of three apomucin genes, abnormal glycosylation, and abnormalities of secretion. They are probably significant in the condition's clinical presentation. These changes were not demonstrated in this case.

A case report by Ohira et al (2006) described a 43-year-old woman with primary pulmonary hypertension and refractory protein-losing enteropathy who, on receiving a living donor lobar lung transplantation, showed a marked improvement in lung function and protein loss.

Case Report

A 69-year-old woman was admitted in September 2005 dehydrated and unwell. She had a 6-month history of constipation, poor appetite and weight loss, and was hypoalbuminaemic (21 g/litre). She had initially been referred for colonoscopy in March to exclude colonic malignancy. Multiple pedunculated polyps up to the splenic flexure were seen, with histology felt to be consistent with hyperplastic polyps and mild inflammation. Her symptoms persisted, and by August she had lost 3 stone in weight. She started to experience loose stools, and repeat colonoscopy was planned. However, the bowel preparation made her feel unwell. She couldn't walk, and so presented to the acute medical take.

The patient looked pale and dehydrated. Her chest was clear, with a diffusely tender abdomen. Bowel sounds were present, and there were no palpable masses. Bloods showed a corrected calcium of 1.58 mmol/litre. Fluid resuscitation and calcium replacement was started. A malignant process was queried. Liver function tests showed a low albumin of 21 g/litre but were otherwise unremarkable. A computed tomography scan of the abdomen and chest were normal. A gastroscopy showed mild oesophagitis with *Candida* in the mid and lower oesophagus, gastritis in the antrum of the stomach with heavy *Helicobacter pylori* growth, and a normal duodenum. Biopsies showed no evidence of coeliac disease or giardia.

The patient's albumin fell to 8 g/litre. A 24-hour urine protein collection was 0.46 g, excluding nephrotic syndrome. Nasogastric feeding was commenced.

Gut hormones showed a modestly elevated chromogranin A (164 pmol/litre) and gastrin (307 pmol/litre) but were not diagnostic of a neuroendocrine tumour. Amyloid was queried and the original colonic biopsies re-examined. This was not found, but cap polyposis was suggested.

The patient failed to improve and died 15 days after admission. Post mortem found the distal large bowel to contain multiple dark sessile polyps, looking like slugs, on exaggerated mucosal folds (*Figure 1*). The histological findings comprised elongated, tortorous and distended crypts, with evidence of inflammation and a 'cap' of granulation tissue, confirming cap polyposis. In addition the patient had a right haemothorax. This was caused by a dissection in the pulmonary artery, with evidence of atheroma identified. The right ventricle was hypertrophic, with the lungs showing multiple haemorrhagic infarcts. These changes suggested pulmonary hypertension. Whether this was related to the cap polyposis was unclear. A reasonable explanation is that the patient had showered off pulmonary emboli from a deep vein thrombosis (DVT) for some time, secondary to immobility associated with her poor state of health. This was supported by a right calf DVT identified at post mortem. The dissection of the pulmonary artery was probably secondary to the low protein state and pulmonary artery hypertension.

Figure 1. Protein-losing enteropathy in cap polyposis showing sessile slug-like lesions.

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There may be an association between cap polyposis and *Helicobacter pylori* carriage. A series of three women with proven cap polyposis demonstrated remission of polyps after eradication of *H. pylori*, sustained at 26 months, demonstrated by Akamatsu et al (2004). No organisms could be seen in the polyps themselves, nor a plausible method for causing the disease. The authors' patient had a heavy load of *Helicobacter*.

The authors identified one other case of cap polyposis with protein-losing enteropathy, reported by Oshitani et al (1995). A 54-year-old woman with a history of constipation developed diarrhoea and hypoalbuminaemia. She was investigated with technetium-labelled human albumin which indicated protein loss from the distal colon. This is the gold standard investigation. Faecal alpha fetoprotein can be screened for in patients unsuitable for this. Although the authors were unable to demonstrate protein loss from the polyps in this case, it seems the most plausible explanation. There was no evidence of malignancy or liver disease on post mortem, and a nephrotic syndrome had been excluded. Histology confirmed cap polyposis and in the absence of any other cause would appear to be the answer to the clinical presentation.

A variety of treatments have been tried. Medical management has involved infliximab, prednisolone, metronidazole and *Helicobacter* eradication. For intractable cases polypectomy or proctocolectomy can be effective. **BJHM**

Akamatsu T, Nakamur N, Kawamura Y et al (2004) Possible relationship between *Helicobacter pylori* infection and Cap polyposis of the colon. *Helicobacter* **9**(6): 651–6

Busine MP, Colombel JF, Lecomte-Houcke M et al (1998) Abnormal mucus in cap polyposis. *Gut* **42**(1): 135–8

Konishi T, Watanabe T, Takei Y, Kojima T, Nagawa H (2005) Cap polyposis: an inflammatory disorder or a spectrum of mucosal prolapse syndrome? *Gut* **54:** 1342–3

Ohira H, Tsujino I, Sakaue S et al (2006) Recovery of protein-losing enteropathy after living-donor lobar lung transplantation in primary pulmonary hypertension. *J Heart Lung Transplant* **25**(4): 486–8

Oshitani N, Moriyama Y, Matsumoto T, Kobayashi K, Kitano Nobuhide A (1995) Protein-losing enteropathy from cap polyposis. *Lancet* **346:** 1567

Crohn's disease discovered by an obstructing chick pea

A 60-year-old man presented to accident and emergency with a 2-day history of increasing central abdominal pain, nausea and vomiting. He had previously been well with no significant past medical history. On presentation he was pale and mottled, with marked hypotension. His abdomen was distended and diffusely tender with guarding. There was no palpable aortic aneurysm.

Inflammatory markers were raised and renal function was deranged. Erect chest radiograph showed no evidence of pneumoperitoneum. Intravenous contrast enhanced computed tomography of the abdomen and pelvis was performed which demonstrated dilated stomach and small bowel to the terminal ileum in which a 10 cm ileal segment exhibited mural thickening. There was the suggestion of an obstructing foreign body within the lumen (*Figure 1*).

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The patient proceeded to laparotomy, which revealed a terminal ileal stricture with a co-existing foreign body obstructing the lumen. No other abnormality was seen and a side-to-side ileocaecal resection was performed.

Histology of the specimen confirmed stricture formation as a result of Crohn's disease. Within the lumen a 10 mm foreign body was confirmed to be a chick pea. Following recovery and in hindsight he said that his sister had been diagnosed with Crohn's disease although with more conventional symptoms. **BJHM**

Figure 1. Computed tomography of the abdomen showing (1) dilated proximal small bowel lumen, (2) thickened, oedematous bowel wall and (3) a hypoattenuating filling defect in bowel lumen representing the chick pea.