CASE REPORT

Eosinophilic gastroenteritis mimicking pancreatic cancer

V Christopher, M H Thompson, S Hughes

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Eosinophilic gastroenteritis is a condition characterised by eosinophilic infiltration of the gastrointestinal tract. Biliary obstruction is an unusual presentation. A case where the initial investigations were consistent with carcinoma of the head of pancreas but the resection specimen showed no malignancy is presented. The diagnosis was suspected from a review of the patient's past history and confirmed by re-examination of the histology.

A 47 year old man presented with two weeks of fever, vomiting, anorexia, intermittent right upper quadrant and mid-scapular pain exacerbated by eating, followed by jaundice, pale stools, and dark urine. On examination, he was icteric, temperature 38.8°C, with right upper abdominal tenderness, and a palpable gallbladder. Investigations showed a raised white cell count 15.6×10^9/l (4–11.0), neutrophils 13.4×10^3/µl (2–7.5), amylase 256 IU/l (10–90), bilirubin 85 µmol/l (2–20), alanine transferase 204 IU/l (1–65), and alkaline phosphatase 897 IU/l (30–110). Abdominal ultrasound showed a grossly dilated biliary tract, 13 cm gallbladder, 3 cm common bile duct (CBD), and 8 mm pancreatic duct. No gallstones were present. Abdominal computed tomography showed an abrupt occlusion of the CBD and pancreatic duct at the head of pancreas. Endoscopic retrograde cholangiopancreatography was unsuccessful due to duodenal stenosis. Percutaneous transhepatic cholangiography found a tight stricture requiring an external biliary drain for decompression. The diagnosis was thought either to be carcinoma of the ampulla of Vater or the head of pancreas.

At laparotomy, a small hard irregular mass in the head of the pancreas was found. Pylorus preserving pancreaticoduodenectomy was performed. The resection specimen was examined by thin sections. The duodenum and ampulla had a chronic inflammatory infiltrate within normal mucosa. There was periductal chronic inflammation, fibrosis and active inflammatory exudate in the CBD, thought consistent with recent cholangitis. Lymph nodes showed follicular hyperplasia and reactive change only. No malignant cells were identified. He was discharged after an uneventful recovery.

He then had repeated hospital admissions with abdominal pain and postprandial vomiting. Initially, all investigations were normal apart from a mild neutrophilia. The symptoms resolved with conservative management. At his next admission, he also had diarrhoea. Investigations showed raised white cell count 18.0×10^9/l, neutrophils 15.5×10^3/µl, alkaline phosphatase 198 IU/l, and reduced albumin 31 g/l (35–50). Erythrocyte sedimentation rate, stool cultures, and plain radiographs were normal. Abdominal ultrasound detected ascites and a dilated CBD (1.2 cm). Further blood counts showed significant eosinophilia 2.3×10^9/l (0.04–0.4). Contrast studies demonstrated free flow through the narrowed gastrojejunal anastomosis. The ascites fluid was an exudate (protein 57 g/l) containing eosinophil cells. The diagnosis remained uncertain.

His past history was reviewed. Twelve years ago, polyarteritis nodosa had been diagnosed after prolonged fever, anorexia, weight loss, abdominal pain, and vomiting. Pulmonary oedema, pericarditis, hypertension, and renal impairment complicated this episode. Raised plasma viscosity 2.18 CP (1.45–1.72), alkaline phosphatase 270 IU/l, creatinine 243 µmol/l (50–120), and eosinophilia 1.1×10^9/l (0.04–0.4) were documented. Urine microscopy was normal. An intravenous pyelogram showed normal sized kidneys with delayed excretion. Renal biopsy showed ischaemic changes affecting the glomeruli with normal small arteries and a lymphocytic/eosinophilic infiltrate. He was treated with frusemide (furosemide), prednisolone, methyldopa, and azathioprine. After 18 months, he was well and off all treatment.

In view of this background, absent neoplasia and peripheral eosinophilia, the diagnosis of eosinophilic gastroenteritis was suggested. The resected specimen was re-examined. Marked eosinophilic infiltrate, hypertrophied muscle bundles, and fibrosis were present in the muscularis propria. Within one week from starting prednisolone, his symptoms resolved.

DISCUSSION

Eosinophilic gastroenteritis is characterised by eosinophilic infiltration of the gastrointestinal tract and peripheral eosinophilia. Symptoms are non-specific with nausea, vomiting, dyspepsia, and weight loss. Approximately 80% have symptoms for several years before diagnosis. Eosinophil levels fluctuate, predating presentation by years and may be absent at presentation. The gastric antrum and proximal small bowel are the most affected sites, commonly presenting with obstruction. Frank ulceration and haemorrhage are unusual. Computed tomography and contrast studies show non-specific features of thickened mucosa and bowel wall. The histology is characteristic with mucosal oedema, a dense eosinophilic infiltrate, muscle bundle hypertrophy, and fibrosis. The submucosa is most commonly affected and full thickness biopsies may be needed for diagnosis. Subserosal disease occurs as inflammatory nodules of eosinophil-laden tissue. Serosal inflammation causes eosinophilic ascites as in this patient.

Biliary obstruction is a rare presentation of eosinophilic gastroenteritis. There are four case reports of biliary involvement, with three cases of duodenal and biliary obstruction and one case of cholangitis and colitis.[1–4] Only one case describes a hypoechoic mass in the head of pancreas occurring with extensive gastrointestinal tract involvement. All three cases of duodenal and biliary obstruction required laparotomy for histological diagnosis. The diagnosis in this case was elusive because of the unusual site and absent peripheral eosinophilia. Overlap with eosinophilic gastroenteritis and hypereosinophilic syndrome exists; the latter is now strictly defined as the presence of persistent blood eosinophilia exceeding 1.5×10^9/l for six months or more with no other cause for reactive eosinophilia. Presentation may vary from a single organ affected by eosinophilic infiltrate to that of multisystem involvement. Men are most affected (sex ratio 9:1); the average age of onset is 37 years.[5] Our patient originally had eosinophilic gastroenteritis with systemic involvement, not polyarteritis nodosa as the histological features of thrombosis

Learning points for eosinophilic gastroenteritis

- Symptoms can predate diagnosis by several years.
- Presentation can mimic malignancy.
- It is characterised by eosinophilic infiltration of the gastrointestinal tract and circulating blood eosinophilia.
- Presence of peripheral eosinophilia fluctuates, and may be absent at diagnosis.
- Histology is characteristic with mucosal oedema, dense eosinophilic infiltrate, muscle bundle hypertrophy, and fibrosis.

and aneurysms affecting small/medium sized arteries are absent from his renal biopsy. The long delay between presentations is consistent with eosinophilic gastroenteritis. Eosinophilic gastroenteritis can present with protein-losing enteropathy, a possible cause of the low albumin and diarrhoea in our patient. Serosal inflammation is the most likely cause of the exudative ascites.

There are no published controlled trials of treatment efficacy, which remains empirical with prednisolone 20–40 mg per day. Resolution is dramatic, occurring within days. Twenty five per cent have a history of atopy. Elimination diets and sodium cromoglycate are successful in rare cases where the causative antigen is isolated.[6 7] Spontaneous resolution may occur. Polypoid gastric disease has prompted gastrectomy for suspected neoplasia. Had the correct diagnosis been suspected before surgery, biliary decompression with external drainage while awaiting resolution of active disease may have been effective. The diagnosis of eosinophilic gastroenteritis can be difficult. It is important to delve into the past medical history for illnesses involving abdominal pain and eosinophilia and review any histology for eosinophilic infiltration if the diagnosis is suspected. Eosinophilic gastroenteritis can mimic pancreatic cancer.

Authors' affiliations
V Christopher, M H Thompson, S Hughes, Department of Gastrointestinal Medicine and Surgery, Southmead Hospital, Bristol, UK

Correspondence to: Dr S Hughes, Southmead Hospital, Westbury on Trym, Bristol BS10 5NB, UK; themadhaggis@hotmail.com

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