CASE REPORT

EOSINOPHILIC COLITIS MIMICKING CAECAL MALIGNANCY

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Primary eosinophilic diseases of the gastrointestinal tract are increasingly being recognised in adults. Eosinophilic colitis is even less understood and presents with highly variable symptoms depending on the depth of mucosal involvement. We are presenting a case of primary eosinophilic colitis presenting with diarrhoea and localized caecal perforation. Pre-operative computed tomography suggested caecal malignancy and possible liver metastasis. Patient underwent an emergency laparoscopic right hemicoloectomy and histology revealed eosinophilic colitis. Post-operative period was complicated by pulmonary embolism and deep vein thrombosis. Secondary causes of eosinophilia were appropriately investigated and excluded. She made a good recovery and a post-operative colonoscopy looking for other areas of eosinophilia was normal.

Keywords: Colitis, Eosinnophilic colitis, Budesonide, Caecal perforation

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INTRODUCTION

Primary eosinophilic colitis is a rare form of colitis which was first described by Kaijser in 1937.[1] Secondary causes are much more prevalent and further diagnostic dilemma is caused by wide spectrum of clinical presentations. Although, previously seen as condition of paediatric population it is increasingly been reported in adults. The case described is one of the very few reported cases mimicking caecal malignancy.

CASE REPORT

A 44 year old female presented with 2 week history of watery diarrhoea 5-6 times daily, after returning from holiday in Tunisia. She also developed severe nausea and vomiting just before admission to hospital. There was no significant past medical history or family history. She was prescribed diarolyte and ciprofloxacin by the general practitioner. Her physical examination revealed nothing except a generalized lower abdominal tenderness. Blood investigations revealed a haemoglobin of 13.6, white cell count of 14.3 with neutrophilia of 11.2 and Eosinophilia of 1.4. Her C-reactive protein was elevated at 102 with normal renal and liver biochemistry. X-ray examination of Abdomen and erect chest were also normal.

She was initially treated as gastroenteritis with intravenous fluids and antiemetic's while stool cultures were awaited. As the initial response was inadequate she had an Ultrasound of abdomen and pelvis which was largely unremarkable apart from some fluid in the Douglas's pouch.

Her condition further deteriorated with worsening abdominal pain and tenderness. She developed high grade temperature, tachycardia and the C-reactive protein increased to 362 with white cell count of 17.4. A computerized tomographic scan of the abdomen was organized which revealed thickened ascending colon, possible caecal perforation, intra-abdominal lymphadenopathy and multiple low density liver lesions which were questioned as metastasis.

She underwent an emergency laparotomy which revealed caecal infarction with perforation, but ascending colon and small bowel were normal. A right hemi colectomy and end ileostomy was performed. Post operatively she had a slow recovery complicated by pulmonary embolism and then a Left popliteal deep vein thrombosis.

Histology sample from the surgery showed patchy infarction of caecum with marked eosinophilic infiltration and no parasites were identified. She was extensively investigated for parasitic infections by the infectious diseases team and for systemic vasculitic conditions by the rheumatology team and secondary causes were excluded. The final diagnosis was primary eosinophilic colitis.

The patient is currently well and her follow up CT showed that liver lesions were resolving. Furthermore, colonoscopy post operatively was normal and patient is currently waiting reversal of her Ileostomy.

DISCUSSION

Eosinophils are predominantly tissue dwelling cells with relatively few present in the peripheral circulation. They respond to external stimuli, such as trauma, infection and allergens, by degranulation and release of inflammatory mediators including leukotrienes, vasoactive intestinal polypeptide, tumour necrosis factor and interleukins.[2]

Primary Eosinophilic colitis is a rare form of colitis which was first described by Kaijser in 1937.[1] This condition has highly variable symptoms, depending on depth of mucosal involvement, and

diagnosis is more thought provoking due to secondary causes of eosinophilia.[3] Aetiology of this condition is unknown, although food allergy has been implicated, supported by presence of allergy or atopy in vast majority of patients.[4] IgE might have a role as demonstrated by mast cell accumulation in colon.[5]

Three hall mark features of eosinophilic gastrointestinal diseases (EGID) include peripheral eosinophilia, segmental infiltration of gastrointestinal tract and functional abnormalities.[1,6] Interestingly, up to 23% of patients with EGID have no peripheral eosinophilia.[6] Klein *et al* divided the disease according to the layer of the intestinal wall involved into mucosa predominant, muscularis propria predominant and serosa predominant.[7] This correlates well with clinical manifestation of the disease. Mucosa-predominant disease shows evidence of mucosal dysfunction, such as protein-losing enteropathy, malabsorption, and diarrhoea. Trans mural disease is recognized by symptoms of intestinal obstruction, bowel wall thickening on imaging studies and even bowel perforation.[7,8] Finally, serosal involvement is distinguished by the presence of eosinophilic ascites, with up to 88% eosinophils seen on fluid analysis.[7,9]

Unlike eosinophilic esophagitis there are no consensus diagnostic criteria for eosinophilic esophagitis. Secondary causes of eosinophilia are much more common and need to be ruled out. There are no randomized control trials and as with any other allergic condition, corticosteroids have been used effectively.[4]

Budesonide has a role in right sided disease and ileal involvement.[4,8] Steroid sparing drugs, antihistamines, montelukast and biologics have also been used.[8] It has not been demonstrated that treatments such as steroids reduce the mucosal eosinophilic infiltration. The symptomatic relief should be the aim of treatment rather than histological improvement.[10]

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