Case Report

Hepatomegaly and Periportal Oedema of the Liver in a Patient with Eosinophilic Gastroenteritis

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Abstract

Periportal halos are an uncommon finding on computerised tomography (CT) of the liver. Here, reported a case of periportal halos and hepatomegaly in a patient with eosinophilic gastroenteritis. A 49-year-old male presented with a six week history of right lower quadrant pain and diarrhoea. A CT of the abdomen showed hepatomegaly and multiple hypodense periportal halos around the patent portal veins consistent with periportal oedema. A colonoscopy showed normal looking mucosa in the colon and terminal ileum. Blind biopsies taken throughout the terminal ileum and colon showed increased numbers of eosinophils (more than 25 per high-power field) consistent with eosinophilic gastroenteritis. A liver biopsy showed minimal non-specific chronic inflammatory infiltrates and eosinophils in the portal tracts with ductular proliferation. In conclusion, eosinophilic gastroenteritis should be considered in patients presenting with periportal halos, hepatomegaly, and diarrhoea.

Keywords: *periportal, oedema, hepatomegaly, eosinophilic gastroenteritis, diarrhoea, corticosteroid*

Introduction

Periportal halos are an uncommon finding on computerised tomography (CT) of the liver. The cause of these halos is probably an accumulation of fluid around the surrounding periportal regions. These periportal halos are typical of periportal oedema and can be seen in patients with trauma, congestive heart failure, pericardial tamponade, venous occlusion after haemotopoietic stem cell transplantation, acute hepatitis, primary sclerosing cholangitis or tumours in the porta hepatis resulting in obstruction of lymphatic drainage (1,2). A case report of periportal halos in a patient with eosinophilic gastroenteritis.

Case Report

A 49-year-old male presented with a six week history of right lower quadrant pain and diarrhoea 10 times a day. He had good past health, was a non-smoker, non-alcoholic, and had not taken any drugs, Western or herbal, in the last six months. Physical examination showed tenderness over the right lower quadrant, but there was no evidence of rebound or rigidity. Liver biochemistry showed elevated serum alanine aminotransaminase 110 U/L (normal range 5–53 U/L), aspartate aminotransaminase 235 U/L (normal range 14–64 U/L), alkaline phosphatase 155 U/L (normal range 30–90 U/L), and gamma glutamyl transpeptidase 180 U/L (normal range < 84 U/L). The patient's albumin, globulin, and bilirubin were normal. A complete blood count, erythrocyte sedimentation rate, C-reactive protein, immunoglobulin E, peripheral eosinophil count, and renal biochemistry were all normal. Hepatitis A, B, C, and E markers were all negative. Repeated stool samples for parasites were all negative.

A CT of the abdomen showed hepatomegaly with the liver measuring 14.8 cm in length at the mid-clavicular line. Multiple hypodense periportal halos were noted around the patent portal veins consistent with periportal oedema (Figure 1a,1b). The hepatic and portal veins were patent. No focal mass was seen in the liver or in the rest of the abdomen.

A colonoscopy showed normal looking mucosa in the colon and terminal ileum. Blind biopsies taken throughout the terminal ileum and colon showed normal glandular architecture. However, the lamina propria and submucosal regions at the terminal ileum, caecum, ascending colon, transverse colon, and descending colon showed increased numbers of eosinophils (more

than 25 per high-power field) consistent with eosinophilic gastroenteritis.

Magnetic resonance cholangiopancreatography (MRCP) four days after the CT was normal, and there was no evidence of primary sclerosing cholangitis or ductal obstruction. A liver biopsy was performed to determine the cause of the hepatomegaly and periportal halos. It revealed minimal non-specific chronic inflammatory infiltrates in the portal tracts, with ductular proliferation. Eosinophils were identified, and the limiting plate was intact. The hepatic lobules showed mild macrovesicular fatty change and glycogenation of nuclei. Occasional foci of mild intrahepatic cholestasis and haemosiderosis were noted. There was also no evidence of liver cell apoptosis, granulomatous inflammation, dysplasia, or malignancy.

No malignancy was found on CT of the abdomen and the pelvis, the MRCP, upper endoscopy, colonoscopy, or a chest X-ray. There was no evidence of parasite infestation in repeated stool samples. Serological tests for *Trichinella spiralis, Wuchereria bancrofti, Toxocara canis, Schistosoma*, and *Echinococcus* were all negative.

In view of the severity of the patient's diarrhoea and the extra-intestinal involvement of the eosinophilic gastroenteritis, an elemental diet, systemic corticosteroid (0.5 mg/kg/day), ketotifen, cetrizine, and monteleukast were prescribed for the eosinophilic gastroenteritis. Resolution of the patient's diarrhoea and abdominal pain was achieved after one week of therapy. The patient's alanine aminotransaminase, aspartate aminotransaminase, and alkaline phosphatase normalised after two weeks of the therapy. A repeat CT of the abdomen three weeks after the initiation of the therapy showed complete resolution of the periportal halos, and the liver span had also returned to normal.

The systemic corticosteroid was slowly decreased by 5 mg every two weeks after the repeat CT scan confirmed the resolution of the periportal halos. The patient received the systemic steroid for a total of 13 weeks. Cetrizine and ketotifen were discontinued after the CT scan confirmed the resolution of the periportal halos, and the patient was maintained thereafter on monteleukast only and an elemental diet. In 18 month follow-up, the patient showed no recurrence of diarrhoea or symptoms while on monteleukast and the elemental diet.

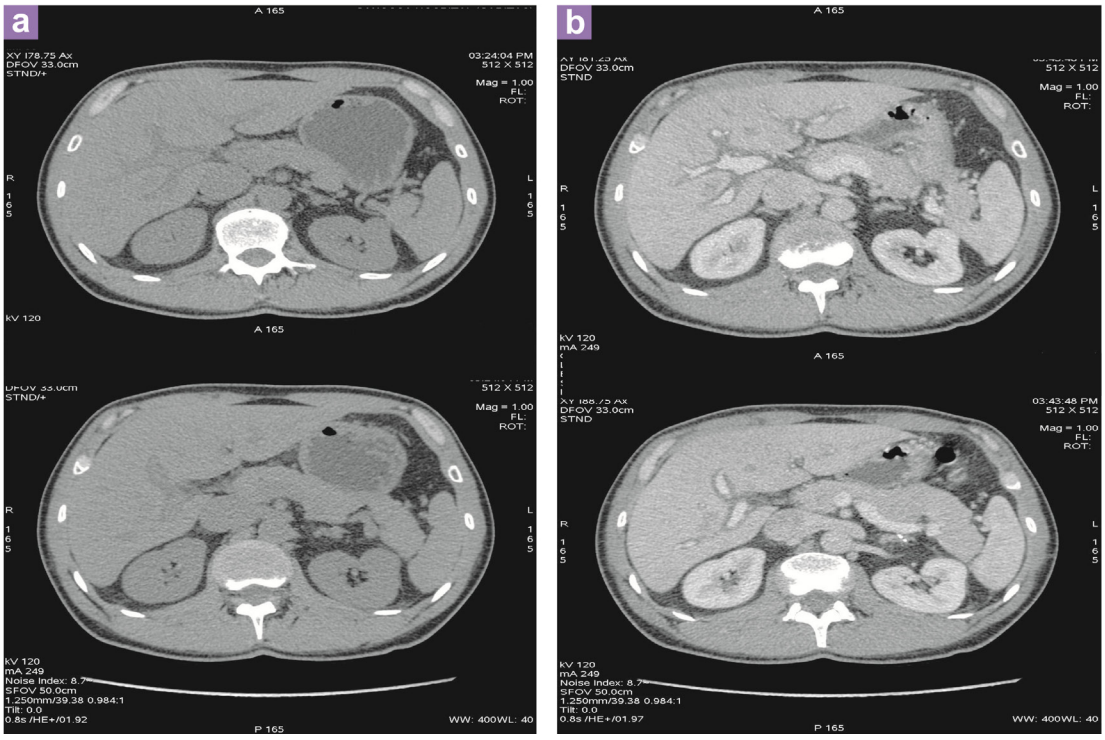

Figure 1: (a) Computerised tomography of the liver showing periportal halos typical of periportal oedema without contrast enhancement, and (b) periportal halos on computerised tomography after contrast injection.

Discussion

Eosinophilic gastroenteritis was first described in the 1930s (3). Eosinophilic gastroenteritis is an uncommon disease, with an estimated incidence around 28 per 105/year (4). It can affect any part of the gastrointestinal tract.

Eosinophilic gastroenteritis can be divided into three types: mucosal, subserosal, and muscular. Mucosal disease usually results in protein-losing enteropathy or malabsorption; the subserosal form can give rise to peritonitis or ascites. The muscular form, which is associated with involvement of the muscle layer, can result in thickening of the bowel wall and intestinal obstruction (5).

The colon (88%) and the ileum (72%) are the commonest sites of involvement in eosinophilic gastroenteritis (5). Seventy percent of those with eosinophilic gastroenteritis will have at least two segments of the intestine affected (5). Eosinophilic gastroenteritis has also been reported to be associated with extra-intestinal involvement such as transmural eosinophilic cholecystocholangitis and acute pancreatitis (6,7). However, to the best of our knowledge, this is the first report of eosinophilic gastroenteritis with hepatic involvement.

The eosinophilic gastroenteritis in this patient was complicated by hepatitis, periportal halos and hepatomegaly. The hepatitis was most likely due to an inflammatory response in the liver secondary to the eosinophilic gastroenteritis. The eosinophilic gastroenteritis may have caused inflammatory cell infiltration into the portal areas and ductular proliferation, with resultant periportal halos.

Dilated lymphatics and lymphoedema on CT scans have been described in both intra-hepatic and extra-hepatic diseases (1,2). These dilated lymphatics and lymphoedema are due to altered hepatic lymphatics and appear as periportal halos on CT. These periportal halos may represent the accumulation of dilated lymphatics or fluid in the interstitial space around the portal vein or around the portal triads (1,2).

Periportal halos on CT scans have also been reported in patients with acute hepatitis (8,9). In a study by Cakir et al. (9), patients with acute hepatitis with periportal halos on CT had inflammatory infiltrates in the portal tracts with ductular proliferation and periportal halos in liver biopsy. These findings are similar to the histological findings in this case report. Similar to the cases of acute hepatitis (9), the periportal halos resolved completely in this patient following the successful treatment of the eosinophilic gastroenteritis.

The incidence of eosinophilic gastroenteritis has been increasing in the last 16 years (4). However, due to its low incidence, there is a dearth of large prospective randomised trials on the treatment of this condition (4,5,10). Therefore, at present, there is a lack of consensus on the optimal treatment of eosinophilic gastroenteritis. Currently, treatment for this condition includes allergy avoidance, an elemental diet, topical corticosteroids and/or systemic glucocorticoids (10).

Some experts have suggested that clinicians should initially consider allergy avoidance and that topical glucocorticoid should be initiated if allergy avoidance fails to improve symptoms (10). If there is no response to the topical glucocorticoid, then systemic glucocorticoid should be considered (10). They have also recommended an elemental diet with the aim of avoiding protein antigen exposure (10).

In view of the extra-intestinal involvement of the eosinophilic gastroenteritis and the severity of the patient's diarrhoea, systemic steroid, monteleukast, ketotifen and cetrizine were prescribed with the objective of inducing remission as soon as possible. Systemic corticosteroid was commenced upon the diagnosis because its' use has been found to rapidly improve eosinophilic gastroenteritis with extra-intestinal involvement (6,7).

There was no recurrence of the eosinophilic gastroenteritis in the 18 month follow-up. It is uncertain whether this was due to the elemental diet, monteleukast or the natural history of eosinophilic gastroenteritis. As reported earlier, 42% of those with eosinophilic gastroenteritis will experience only one single episode without any recurrence (5). More randomised controlled studies on the use of an elemental diet in managing eosinophilic gastroenteritis should be conducted to determine its efficacy.

Conclusion

In conclusion, eosinophilic gastroenteritis should be considered in patients presenting with periportal halos, hepatomegaly and diarrhoea. The periportal oedema and hepatomegaly may resolve following successful treatment of the eosinophilic gastroenteritis.

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Conflict of Interest

None.

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