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CASE REPORT

Eosinophilic Gastroenteritis in Children-Report of One Case

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SUMMARY **Eosinophilic gastroenteritis is rare in pediatric patients. The three main manifestations, defined by Klein *et al.* in 1970, were (a) predominant mucosal, (b) predominant muscular-layer, and (c) predominant subserosal disease. The predominant subserosal type is the rarest of the three. We report on a 43-month-old boy who, on admission, suffered from recurrent abdominal pain, vomiting and diarrhea for one week, with ascites and pleural effusion noted. The white blood cell (WBC) count of ascites fluid was 8,000/mm^3, with a differential count of 99% eosinophils. The peripheral WBC count was 44,000/mm^3, with 78% eosinophils. Three days after diagnosis, ascites, pleural effusion and other gastrointestinal symptoms were gradually relieved using steroid therapy, with the peripheral eosinophil count returning to normal. The steroid therapy was discontinued after two months with tapering dose. The boy was in good health with no recurrence of symptoms in a follow-up conducted after one year.**

Eosinophilic gastroenteritis is a disease where the gastrointestinal tract is infiltrated with eosinophilic leukocytes.[1] Clinical symptoms are presumably related to the particular site in the gastrointestinal tract that is involved. Several extra-gastrointestinal clinical manifestations have been reported but none are consistent.[2] We report a rare type of pediatric eosinophilic gastroenteritis, with the major clinical symptoms of ascites and pleural effusion.

CASE REPORT

A 43-month-old boy was admitted to our hospital with a one-week history of intermittent vomiting and watery diarrhea. Initially, oral medication was prescribed for a diagnosis of viral gastroenteritis. Two days prior to admission, the boy deteriorated with the development of recurrent abdominal pain, watery diarrhea, poor appetite, and abdominal distension. He was then referred to Chung Shan Medical and Dental College Hospital for further evaluation and treatment.

On admission, physical examination revealed abdominal distension with tenderness over the periumbilical area, hypoactive bowel sounds with shifting percussion dullness; there were no specific findings otherwise. He was born at term with a birth weight of 2,950 gm. Body weight at the time of admission was 22 kg. An assessment of personal and familial medical history revealed no evidence of food allergy, neoplasm, urticaria or hyperreactive airway disease. Laboratory findings indicated a WBC count of 44,000/mm^3 with a differential count of 10% segmented neutrophils, 78% eosinophils, 12% lymphocytes and no immature cells. Hemoglobin was 14.0 g/dl, red blood cell count was 511 x 10^4/mm^3 and platelet count was 285,000/mm^3. The C-reactive protein (CRP) level was 2.64 mg/dl (normal < 0.3 mg/dl). Erythrocyte sedimentation rate (Westergren)

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was 14 mm/hr and 33 mm/2hrs. Total albumin level was 3.5 g/dl. Serum amylase was 39 U/l and the α-fetal protein level was 5.9 ng/ml. Urinalysis was normal. Examination of four stool samples for ova and parasites were negative. Stool cultures revealed normal flora. The serum indirect hemagglutination test for ameba, Rota virus and *Toxoplasma gondii* were negative. Serum aspartate aminotransferase, alanine aminotransferase, blood urea nitrogen and creatinine were within normal limits. Abdominal sonography revealed moderate quantities of ascites and an MRI of the abdomen showed ascites, mild bilateral pleural effusion and no evidence of abnormal mass or lymph node enlargement.

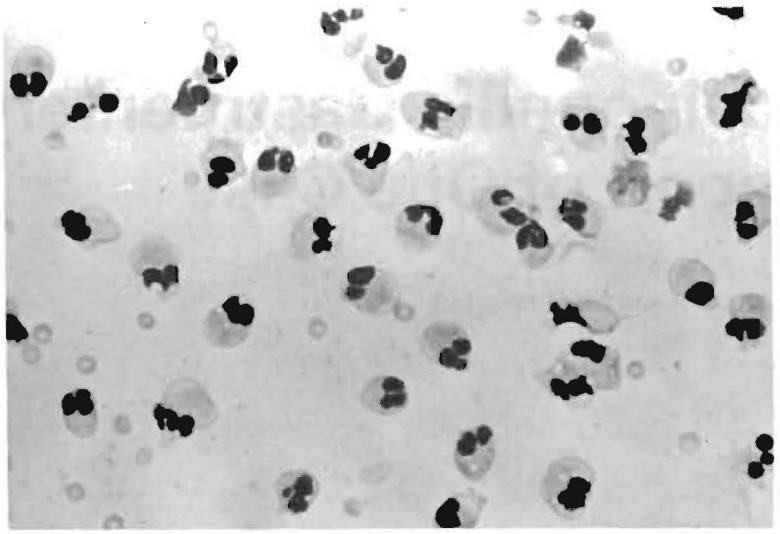

Fig. 1 Numerous eosinophilic cells in the ascites fluid of the patient.

Abdominal paracentesis yielded 30 ml of exudates with a WBC count of 8,000/mm^3, a differential count of 99% eosinophils and 1% histiocytes. Gram stain revealed no microorganisms. Culture for aerobes, anaerobes, fungi and acid-fast stain of the ascites fluid were all negative. The cytology of the ascites fluid revealed numerous eosinophilic cells (Fig. 1) and with no malignant cells. Endoscopy of the upper gastrointestinal tract revealed mild hyperemia at the gastric antrum, body of the stomach and bulb of duodenum, no specific abnormal finding down to the proximal jejunum. A biopsy of the antrum and body of stomach revealed chronic inflammatory lymphocyte infiltration in the lamina propria. No *Helicobacter pylori* or evidence of malignancy was present. The patient was on oral prednisolone therapy at the dosage of 1 mg/kg/day with an impression of eosinophilic gastroenteritis. Three days later, the abdominal distension, vomiting and watery diarrhea gradually improved. Before discharge, the WBC count was 10,800/mm^3 with only 0.3% eosinophils. The steroid therapy was tapered off and finally discontinued altogether after two months. The boy did well subsequently without recurrence of symptoms in a follow-up at one year.

DISCUSSION

Eosinophilic gastroenteritis is an uncommon disease. Although rare, it has been reported in pediatric patients of all ages. The disease occurs in both sexes, however, males are affected at a slightly higher frequency.[2] The pathogenesis of this condition is poorly understood.[3,4] In a 1970 review by Klein *et al.*,[4] they related the clinical manifestations to the area of maximal gastrointestinal involvement and the depth of the disease process and proposed a classification closely linked to the histologic level of eosinophilic infiltration. This classification is more practical to the clinician and gained wide acceptance.

Clinical manifestations depend on the area of maximal gastrointestinal involvement, and particularly on the depth of maximal disease process. Three main patterns can be delineated:

1. Predominant mucosal disease- the most common form, presentation includes iron-deficiency anemia, fecal blood loss, hypoproteinemia and failure to thrive.

2. Predominant muscle-layer disease- characterized by marked thickening and rigidity of the gut and development of obstructive symptoms, such as pyloric obstruction.

3. Predominant subserosal disease- the rarest form, characterized by eosinophilic ascites, pleural effusion and eosinophilic granuloma.

In our patient, eosinophilic ascites and pleural effusion were the major findings, and compatible

with a diagnosis of predominant subserosal disease, responding dramatically to steroid therapy. The predominant mucosal and muscle-layer variants of the disease could not be ruled out because the biopsy revealed only chronic inflammatory lymphocyte infiltration. Eosinophilic infiltration of the mucosal and muscular layers could be patchy and thus could be missed during biopsy.[3]

Eosinophilic gastroenteritis must be differentiated from cow-milk-protein allergy, although there are clinical/pathologic similarities for both disorders, especially for infants. Patients with eosinophilic gastroenteritis tend to be older, however, and may exhibit more severe symptoms over a spectrum of dysfunction, both intestinal and systemic, which do not respond to dietary manipulations alone. Further, cow-milk-protein allergy is a self-limited condition in the majority of cases, whereas eosinophilic gastroenteritis requires steroid therapy.

The pathogenesis and etiology of eosinophilic gastroenteritis are unknown. Some authors have reported a strong association between the ingestion of certain foods (allergy to which can be identified by skin-prick test or radioallergosorbent testing; RAST), and the development of gastrointestinal tract symptoms.[2,5] Immunological studies in our patient revealed a total IgE level of 20 kU/l and negative CAP (Pharmacia & Upjohn, Sweden) food allergen tests for egg white, milk, cod fish, crab, wheat, peanut, soy bean, yeast and melons. An elimination diet was not implemented either during or after the treatment period. However, some authors have reported a relationship for eosinophilic gastroenteritis and dietary factors. Cello[6] has also supported the use of elimination diets which should have rigid restriction of substances that were known to exacerbate symptoms or were suspected of exacerbating symptoms, especially for those patients with an atopic history.

The differential count for eosinophils for our patient was as high as 75-85%. Talley *et al.*[3] reviewed a series of eosinophilic gastroenteritis cases and reported that a higher eosinophil count ($p < 0.05$) was noted for patients diagnosed with predominant subserosal disease. By contrast, the eosinophil count was normal for patients with the mucosal and muscular variants, suggesting that peripheral eosinophilia is not a definite diagnostic criterion.

In conclusion, eosinophilic gastroenteritis is a rare disease in children. Patients presenting with gastrointestinal symptoms and eosinophilic infiltration of some portion of gastrointestinal tract, and/or peripheral eosinophilia, should be diagnosed having eosinophilic diseases. Steroid therapy provides effective treatment for these cases.

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