

## Short reports

# Esophageal involvement in eosinophilic gastroenteritis

M. A. Matzinger and A. Daneman

Department of Radiology, Hospital for Sick Children, Toronto, Canada

**Abstract.** The radiologic appearance of esophageal involvement due to eosinophilic gastroenteritis in a 15-year-old boy is presented. The lower two thirds of the esophagus was narrowed and the peristalsis diminished. The mucosa appeared smooth. This is the fourth reported case of esophageal involvement in eosinophilic gastroenteritis.

**Key words:** Esophagus – Eosinophilic gastroenteritis – Radiology

### Introduction

Eosinophilic gastroenteritis usually involves the stomach and small bowel [1, 8, 10]. Esophageal involvement is rare [3, 8] and the radiologic appearance of esophageal involvement has been illustrated previously in only one case [10]. The purpose of this paper is to present a similar such case.

### Case report

A white male was well until the age of 6 years when he then developed episodes of diffuse abdominal pain, decreased appetite, vomiting and loose stools. These episodes lasted for approximately 2 years and occurred about once a week. At 7 years of age he was found to have an iron deficiency anemia and an eosinophilia. Investigations at that time failed to reveal an etiology for these abnormalities. The anemia responded to oral iron therapy but has recurred several times when iron has been discontinued.

At 11 years of age he was seen at the Hospital for Sick Children, Toronto, because of chronic fatigue and for further investigation of persisting hematological abnormalities. At this age he had developed intolerance to certain foods. These foods included beef, lamb, fish, nuts and chocolate. Since that time the patient has avoided these foods. The iron deficiency anemia was thought to be related to loss of blood through the gastrointestinal tract due to food allergy. At 13 years of age a small bowel enema revealed no abnormality.

At 15 years of age he was again seen at the Hospital for Sick Children, Toronto because of occasional vomiting and abdominal pain. At this time he admitted to mild dysphagia with solid foods sticking in the mid esophagus. The physical examination was unremarkable. Growth had been normal although he was somewhat thin. The hemoglobin level was 17.4 g % on oral iron therapy. The total white blood cell count was 11,800/mm<sup>3</sup> with 34% eosinophils. Serum proteins and immunoglobulins were normal. A chromium chloride study showed excessive gastrointestinal protein loss of 12.4% of the administered activity (a normal value is less than 0.8%).

There was a family history of asthma and rhinitis but the patient did not suffer from these disorders.

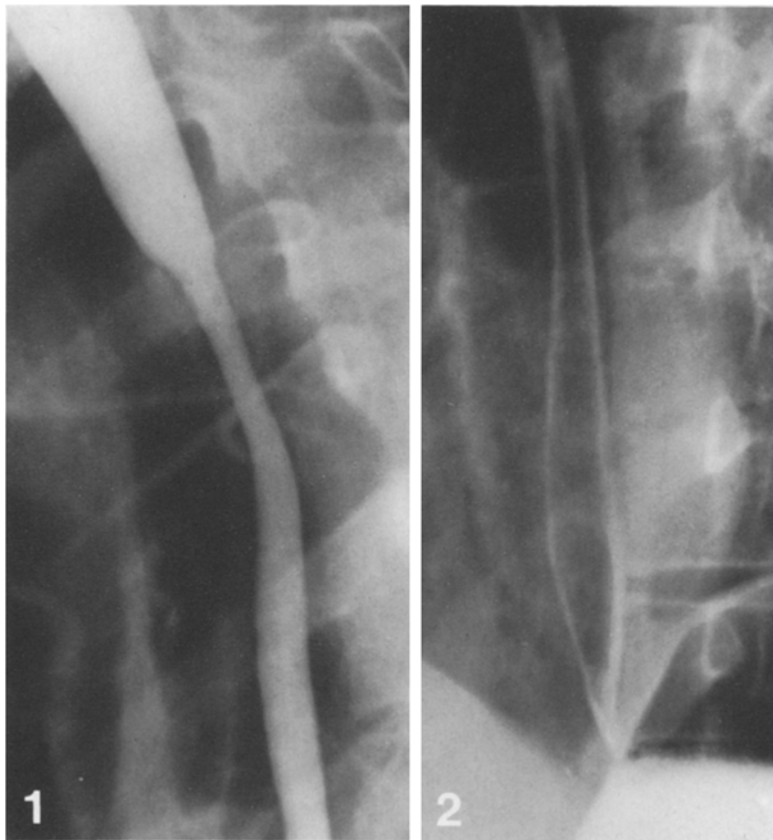
An upper gastrointestinal series and small bowel follow-through were performed at this time. There was no delay in the passage of liquid barium down the esophagus into the stomach. The lower two thirds of the esophagus remained narrowed throughout the examination and there was slight dilatation of the proximal third (Fig. 1). Peristaltic activity in the lower esophagus was diminished. The mucosa of the esophagus appeared normal (Fig. 2). At no time during the examination was there any evidence of hiatus hernia or gastroesophageal reflux. A large amount of resting fluid was present in the stomach making examination of the mucosa difficult. No gross lesions of the stomach were identified. Barium passed through the small bowel with no delay. The wall of the small bowel particularly in the jejunum was thickened and the folds throughout the small bowel were also thickened.

An attempt was made to perform esophageal manometry but the patient did not tolerate this procedure well. No measurements were obtained.

Esophagoscopy revealed spotty areas of inflammation in the lower half of the esophagus. This part of the esophagus was not distensible. Further areas of patchy mucosal inflammation and friability were noted in the fundus of the stomach. The body of the stomach and duodenum appeared normal. Several biopsies were taken from the inflamed areas in the gastric fundus and lower esophagus.

Histological examination of the biopsy specimens revealed a diffuse infiltration of inflammatory cells composed predominantly of eosinophils in both esophagus and stomach. There was no evidence of vasculitis. The squamous epithelium of the esophagus was somewhat thinned and atrophic. In several areas the eosinophils formed small aggregates: Charcot-Leyden crystals. The findings were consistent with eosinophilic gastroenteritis involving the stomach and esophagus.

The patient was started on sodium chromoglycate (Intal) but after three months he showed no improvement. Medication was then changed to oral prednisone.



**Fig. 1.** Esophagram showing a calibre change at the junction of the upper third and lower two thirds of the esophagus. At no time did the lower two thirds of the esophagus distend normally and peristalsis was diminished

**Fig. 2.** Air contrast esophagram of the lower esophagus again shows narrowing of the esophagus and the mucosa is smooth without ulceration

## Discussion

Eosinophilic gastroenteritis (EG) is an uncommon disease and its etiology is poorly understood [2, 10]. The disorder is characterized by a peripheral eosinophilia and infiltration of the gastrointestinal tract with eosinophils. The commonest sites that are affected are the stomach and small bowel – colonic and esophageal involvement are rare [8–10]. EG is a disease mainly of adults [4, 5, 7] and there have been only a few reports of the disease occurring in childhood [6, 11, 12]. Allergic disorders such as eczema, rhinitis and asthma are found in approximately one half of all patients or their families [3–5, 10, 12].

The clinical presentation depends on the site of involvement in the gastrointestinal tract as well as the layer of the bowel that is infiltrated [4, 9, 13]. Nausea, vomiting, periumbilical crampy pain and loose watery stools are typical of mucosal involvement of the stomach and small bowel. Growth retardation, weight loss, iron deficiency anemia, protein losing enteropathy, malabsorption and steatorrhea are sometimes encountered [2]. Involvement of the muscle layer may lead to gastric outlet obstruction or small bowel obstruction [2, 9, 11]. Serosal disease is rare and may

be associated with an eosinophilic ascites [6, 9]. These various types may occur in combination [9]. Concurrent eosinophilic infiltration of the bladder and pancreas has been reported in a few cases [1–3].

The clinical course may be self-limiting but short term steroids may be required to control acute episodes. Less commonly long term therapy is required to control exacerbations [2, 5, 9, 10, 13]. Elimination diets are often ineffective [2, 9]. Surgery is reserved for those patients with persistent obstructive symptoms or for patients in whom the diagnosis cannot be made in any other way [4].

The radiologic manifestations of gastric and small bowel involvement have been well documented [1, 4, 5, 9, 11]. The changes are non-specific and the lesions may be localized or diffuse and patchy [13]. Mucosal infiltration in the stomach and small bowel leads to thickening of the folds and nodularity, polypoid changes, spasm, irritability and increased secretions [9]. In children a lacy pattern has been described in the antrum [11]. Involvement of the muscle layer leads to antral rigidity with gastric outlet obstruction and a moulage or pipestem appearance in the small bowel. With separation of bowel loops the appearances may simulate Crohn's disease or lymphoma [5, 9]. Occa-

sionally minimally involved areas of stomach or small bowel may appear normal radiographically [1, 5, 9]. Colonic changes are unusual but may simulate inflammatory bowel disease [9]. After steroid therapy the radiologic changes usually revert to normal [5, 9].

Esophageal involvement in EG has previously been reported in only 3 cases [3, 8, 10]. The first case was reported by Dobbins et al. in 1977 [3]. Their patient was a 51-year-old male who presented with intermittent dysphagia and had a lifelong history of asthma. An eosinophilia of 4% was present. The barium swallow, upper gastrointestinal series and esophagoscopy were normal. Manometric studies of the esophagus revealed motility disturbances. Mucosal biopsy of the esophagus and small bowel revealed eosinophilic infiltration in both sites. The patient's symptoms were not considered serious enough to warrant steroid therapy.

Landres et al. (1978) reported the second case [8]. This was a 44-year-old male with a six-week history of epigastric pain. He had an eosinophilia of 28%. The barium swallow revealed incomplete relaxation of the lower esophageal sphincter with persistence of barium in the esophagus after five minutes (described as vigorous achalasia). Endoscopic biopsy revealed "chronic inflammation" in the mucosa of the distal esophagus. Esophageal manometry revealed changes of achalasia. At thoracotomy a full thickness biopsy of the esophageal wall was performed and this revealed a heavy eosinophilic infiltrate in the muscle layers. Following esophageal myotomy the patient became asymptomatic although the eosinophilia persisted.

In 1981 Picus and Frank [10] reported the third case. The patient was a 16-year-old male who presented with progressively increasing dysphagia over the previous one and a half years. An eosinophilia of 16% was present. An esophagram revealed a 6-cm stricture at the junction of the proximal and middle thirds of the esophagus with mucosal nodularity in this region. The stomach and small bowel were normal. Mucosal ulceration and polyp formation were observed endoscopically in the esophagus in the narrowed area. A biopsy revealed eosinophilic infiltration in the esophageal mucosa and submucosa. The lesions resolved on prednisone therapy. This report was the first to illustrate the radiographic appearance of esophageal involvement in EG.

The case presented in this paper had radiologic evidence of lesions present in the esophagus (Figs. 1 and 2) and small bowel. The increased resting fluid in the stomach may have been due to the gastric involvement. Histologic examination of endoscopic biopsies revealed a heavy mucosal infiltrate of eosinophils in the lower esophagus and gastric fundus. Despite the

long length of esophagus involved, the patient complained of only mild dysphagia.

Previous authors have stressed that the extent of eosinophilic infiltration in the gastrointestinal tract can only be adequately assessed by full thickness biopsy [5, 10]. We agree with Picus and Frank [10] that the prominent motility changes in the esophagus in the first two cases [3, 8] and the esophageal narrowing in their case [10] suggests muscular involvement. Only in the second case [8] was muscle layer infiltration documented by a full thickness biopsy. We feel that the esophageal narrowing and mildly diminished peristalsis noted in the present case probably also reflect muscle layer involvement.

The treatment of esophageal involvement, like involvement elsewhere in the gastrointestinal tract, varies depending on the severity of the process. Minimal involvement may require no therapy [3]. Steroids may be required for more severe cases [10]. Surgery is reserved for those patients who have persistent obstructive symptoms or if the diagnosis cannot be made in any other way [8]. In the present case an initial trial of Intal therapy failed and steroids were introduced.

## Summary

1. Radiologists should thus be aware that eosinophilic gastroenteritis can rarely affect the esophagus.
2. The radiological findings are non-specific and appear to be governed by the severity of the disease and the layer of the esophagus that is involved: mucosal irregularity with mucosal infiltration [10] and esophageal narrowing and motility disturbances with muscular infiltration [7, 10]. EG should thus be included in the differential diagnosis of these types of abnormalities. Minimal involvement may cause no radiographic abnormality [3].
3. A history of food intolerance, allergies, peripheral eosinophilia and accompanying radiologic changes in the stomach and small bowel should aid the radiologist in making the correct diagnosis.

*Acknowledgements.* The authors would like to thank Dr. Dan Wilmot for reviewing the manuscript and Linda Gibbs for typing the manuscript.

## References

1. Burhenne HJ, Carbone JV (1966) Eosinophilic (allergic) gastroenteritis. *Am J Roentgenol* 96: 332
2. Cello JP (1979) Eosinophilic gastroenteritis – a complex disease entity. *Am J Med* 67: 1097
3. Dobbins JW, Sheahan DG, Behar J (1977) Eosinophilic gastroenteritis with esophageal involvement. *Gastroenterology* 72: 1312

4. Edelman MJ, March TL (1964) Eosinophilic gastroenteritis. *Am J Roentgenol* 91: 773
5. Goldberg HI, O'Kieffe D, Jenis EH, Boyce HW (1973) Diffuse eosinophilic gastroenteritis. *Am J Roentgenol* 119: 342
6. Jona JZ, Belin RP, Burke JA (1976) Eosinophilic infiltration of the gastrointestinal tract in children. *Am J Dis Child* 130: 1136
7. Klein NC, Hargrove RL, Sleisenger MH, Jeffries GH (1970) Eosinophilic gastroenteritis. *Medicine* 49: 299
8. Landres RT, Kuster GGR, Strum WB (1978) Eosinophilic esophagitis in a patient with vigorous achalasia. *Gastroenterology* 74: 1298
9. Marshak RH, Lindner A, Maklansky D, Gelb A (1981) Eosinophilic gastroenteritis. *J Am Med Assoc* 245: 1677
10. Picus D, Frank PH (1981) Eosinophilic esophagitis. *Am J Roentgenol* 136: 1001
11. Teele RL, Katz AJ, Goldman H, Kettell RM (1979) Radiographic features of eosinophilic gastroenteritis (allergic gastroenteropathy) of childhood. *Am J Roentgenol* 132: 575
12. Waldmann TA, Wochner RD, Laster L, Gordon RS (1967) Allergic gastroenteropathy: a cause of excessive gastrointestinal protein loss. *N Engl J Med* 276: 761
13. Wehunt WD, Olmsted WW, Neiman HL, Phillips JF (1976) Eosinophilic gastritis. *Radiology* 120: 85

Date of final acceptance: November 1981

Dr. Alan Daneman  
Department of Radiology  
Hospital for Sick Children  
555 University Avenue  
Toronto, Ontario, M5G 1X8  
Canada