Eosinophilic Mucosal Infiltrate in Infants with Congenital Gastrointestinal Obstruction

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Infiltration of the antrum and small bowel with eosinophils has been reported to be the etiologic factor for intestinal obstruction in adults with eosinophilic gastroenteritis. We report a case of a breast-fed 8-month-old infant with eosinophilic gastroenteritis (EGE), who presented with severe hematemesis and congenital obstruction of the duodenum, to emphasize that not all obstructive symptoms associated with EGE are secondary to eosinophilic infiltration. Our patient displayed many of the classic signs and symptoms of EGE, including an elevated absolute eosinophil count and marked eosinophilic infiltration in mucosal biopsies from the duodenum, stomach, and esophagus. At surgery there was marked dilation of the first portion of the duodenum and obstruction of the second portion due to malrotation of the intestine with Ladd's bands, duodenal stenosis, and annular pancreas. The dramatic clinical response to surgical correction of the duodenal obstruction leaves little doubt that this patient's symptoms were related to the anatomical lesion.

BACKGROUND

Eosinophilic gastroenteritis (EGE) is characterized by peripheral eosinophilia, eosinophilic invasion of the gastrointestinal tract, diarrhea, vomiting, gastrointestinal hemorrhages, and protein-losing enteropathy [(1–5) Appleman H, personal communication; Kahn E, personal communication]. EGE is frequently associated with peripheral eosinophilia [(5), Appleman H, and Kahn E, personal communications], and food allergy is alleged to be the etiologic factor in as many as one-half of affected children (1, 9). In many children, the symptoms of EGE respond to elimination of cow's milk and soy protein from the diet (2, 3). The presence of allergic symptoms, elevated IgE levels, and symptomatic response to corticosteroid therapy suggest that the immune system and, specifically, the immediate hypersensitivity response may be important in the pathophysiology of EGE (1). It has been proposed that, in children, EGE can be divided into two groups (3). The first group has onset of symptoms at a young age, often during the first year of life, has a short, transient clinical course, and is often associated with specific antigens such as cow's milk protein and soy protein. The second group has a later age of onset, averaging 4 yr., has a persistent clinical course, does not respond to dietary restriction, and often requires glucocorticoid therapy.

Whereas EGE with infiltration of the mucosa of the antrum and small bowel with eosinophils has been reported to be the etiologic factor for intestinal obstruction in adults (1–4), we report a breast-fed infant with eosinophilic gastroenteritis who presented with severe hematemesis and congenital obstruction of the duodenum, to emphasize that not all obstructive symptoms associated with EGE are secondary to eosinophilic infiltration.

HISTORY

K.F. is an 8-month-old girl who had been completely well until 1 month prior to admission, when her mother developed chickenpox. During this illness, the mother increased her intake of dairy products, and K.F. was breast-fed more often. Five days after onset of the varicella rash, K.F. began to have emesis that increased in severity over the next 3 wk. One week prior to admission, she had coffee ground emesis and a large tarry stool.

On admission, the subject was pale, with a pulse 122/min, respiration 35/min, blood pressure 85/55 mm Hg, and a temperature of 37.5°C. Her weight was at the 5th percentile, and her height was at the 60th percentile for age. Review of her growth pattern showed a decrease from the 50th percentile for weight at birth to the 30th percentile at 2 months prior to admission, and then a rapid decrease in weight percentile to the 5th percentile at the time of admission. Abdominal examination showed a soft nontender abdomen without masses. Stool was positive for occult blood. The remainder of the physical examination was unremarkable.

The hemoglobin was 10.4 g/dl, platelet count of 457,000/cm³, and white blood cell count of 37,700/cm³ with 23% eosinophils, 38% neutrophils, and 39%
lymphocytes. The initial abdominal x-ray suggested intestinal obstruction with a dilated loop of bowel in the right upper quadrant, but a repeat abdominal x-ray 12 h later was normal.

An esophagogastroduodenoscopy was performed to identify the source of the upper gastrointestinal bleeding, revealing severe erosive antritis and duodenitis in the bulb and second portion of the duodenum, with active bleeding and marked edema. Biopsies of the esophagus, stomach, and duodenum showed marked eosinophilic infiltration (Fig. 1) and focal ulceration. The patient was treated with Maalox, 2.5 ml p.o., seven to eight times per day.

Prior to reintroduction of breast feedings, while on Pedialyte for 4 days, the infant's absolute eosinophil count decreased from 8320 to 530 (normal < 500). Dairy products were excluded from the mother's diet for 2 days, but when breast feedings were reintroduced, the absolute eosinophil count rose to 6800, and K.F. again became irritable. The diet was changed to Pregestimil, and breast feeding was discontinued. Vomiting resolved, and occult blood was no longer present in the stool. Two weeks after discharge, she was still vomiting one time each day. Because of persistent emesis, an upper gastrointestinal series was performed which showed an obstruction at the junction of the second and third portion of the duodenum (Fig. 2). Surgical exploration revealed partial malrotation of the small intestine, Ladd's bands, duodenal stenosis, and a non-obstructing annular pancreas. A Ladd's procedure and duodenostomy were performed. Postoperatively, vomiting resolved. The infant was maintained on Pregestimil for just 2 wk, and then Pregestimil was replaced with Enfamil. Following surgery, she had excellent weight gain, and 3 wk after the operation had an absolute eosinophil count of approximately 1550 (normal < 500).

**DISCUSSION**

This infant with congenital partial obstruction of the duodenum developed eosinophilic infiltration of the intestinal tract. Although she presented with many of the classical signs and symptoms of EGE, all such signs and symptoms resolved, following surgical repair of the obstruction.

Eosinophilic infiltration of the gastrointestinal tract has been reported in patients with parasitic infection, peripheral hypereosinophilia syndromes, allergic gastroenteropathy due to milk and soy protein, inflammatory bowel disease, and obstruction of the gastrointestinal tract (5). It has been noted and proposed that adults with obstruction of the gastrointestinal tract have eosinophilic infiltration of the gastrointestinal mucosa (Appleman H. personal communication), and marked eosinophilic infiltrate has also been noted in an infant with obstruction of the gastrointestinal tract due to Hirschsprung's disease (Kahn E, personal communication). The number of eosinophiles present in the gastrointestinal tract of infants is increased, compared with the number found in the adult gastrointestinal tract, a difference that parallels an increased average eosinophil count in the blood stream.

The symptoms present in our patient are consistent with the diagnosis of eosinophilic gastroenteritis. Gastrointestinal bleeding is a frequent presentation in patients with eosinophilic gastroenteritis, and anemia sec-

![Fig. 1. Eosinophilic gastroenteritis is illustrated in a biopsy taken from the small intestine. Note the marked infiltration with eosinophils, which was also present in the esophagus, stomach, and duodenum. Eosinophils are indicated by arrows.](image-url)
The barium swallow illustrates the duodenal obstruction present at the junction of the second and third portions of the duodenum. Note the marked dilation of the duodenal bulb. Secondary to gastrointestinal blood loss is a common finding (6–8). Peripheral eosinophilia as high as 55% of the total white blood cell count is also a common finding in patients with EGE (2, 8, 11). Our patient presented with an elevated absolute eosinophil count that decreased when breast feeding was discontinued and increased upon reintroduction of breast feeding. Cow’s milk proteins have been shown to be excreted in breast milk as antigenically intact particles (3, 4, 12–14). Removal of the antigen, either by removal of cow’s milk from the maternal diet or by discontinuation of breast feeding, has been shown to improve or eliminate allergic symptoms (3, 4). The increased maternal intake of cow’s milk combined with the infant’s increased intake of breast milk may have contributed to an overall increase in the daily antigen load and the resultant peripheral eosinophilia and eosinophilic infiltrate of the intestinal mucosa. It is possible that mechanical obstruction of the gastrointestinal tract predisposed to stasis and antigen penetration, precipitating allergic gastroenteritis. This hypothesis is supported by the dramatic increase in peripheral eosinophilia upon reintroduction of breast milk feedings (14).

Our case illustrates that endoscopy has clear limitations in identifying and diagnosing congenital partial obstruction. All patients with a double bubble should have a diagnostic upper gastrointestinal series. The relative drop in weight percentile during the first 2 months of life suggests that, although asymptomatic, our patient may have had a decrease in oral intake related to the partial obstruction of the upper gastrointestinal tract.

Caution must be used in evaluating obstructive signs and symptoms in association with allergic eosinophilic gastroenteritis. Although many patients may have obstructive symptoms due to eosinophilic infiltration, surgery is rarely required. This case emphasizes that peripheral eosinophilia and eosinophilic infiltration of the mucosa may occur as a result of intestinal obstruction, and that not all obstructive symptoms associated with eosinophilic gastroenteritis are secondary to eosinophilic infiltration.

Fig. 2. The barium swallow illustrates the duodenal obstruction present at the junction of the second and third portions of the duodenum. Note the marked dilation of the duodenal bulb.

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REFERENCES
