Segmental ileal resection and end-to-end anastomosis was performed. On the sixth day of admission she was discharged uneventfully.

Primary small bowel volvulus (SBV) occurs in the absence of anatomic abnormalities or predisposing factors. Although very rare in USA and Western Europe, the incidence is 5-10 times higher in Africa, Asia, and Middle East. It is thought to be related to different dietary habits such as ingestion of large volumes of fiber-rich foods after long intervals of fasting (1,2).

Secondary SBV occurs generally in the presence of predisposing factors such as anatomic disorders, malrotations, or postoperative adhesive bands. Post-operative adhesive bands are major problems for all surgeons due to their morbidity (1-3).

Post operative adhesive small bowel obstruction is a rare but documented complication of appendectomy. The frequency of this condition has been reported between 0,2 % and 10,7 % (4). Due to its rarity, long-term severe complications of appendectomy are generally neglected.

Abdominal CT has been shown to be effective for the diagnosis of strangulation of the affected bowel with a high specificity and sensitivity of 93% and 83% respectively (5). When the characteristic "whirl sign" is seen on CT scan, SBV should be suspected (5,6). SBV is an extremely rare but potentially fatal condition that should be kept in mind in all patients with severe abdominal pain. Previous abdominal operations are thought to be an etiologic factor of SBV.

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A duodenal mass and acute pancreatitis

Duodenal kitle ve akut pankreatit

To the Editor,

Eosinophilic gastroenteritis (EGE) is a rare condition, first described in 1937 by Kaijser *et al.* It is defined as a disorder primarily affecting the gastrointestinal tract with eosinophil-rich inflammation, in the absence of known causes of eosinophilia (e.g. drug reactions, parasitic infections, or malignancy) (1). Three different forms of EGE can be distinguished: mucosal disease, muscle layer disease, and subserosal disease. The symptoms of EGE are related to the layer involved. Mucosal disease is the most common form and presents with nonspecific symptoms such as abdominal pain, nausea, vomiting, diarrhea, or malabsorption. The second form, muscle layer disease, is a more serious form that presents with symptoms due to intestinal obstruction. The third form, subserosal di-

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sease, is uncommon and presents with ascites. The incidence of EGE in the USA is approximately 2,5 per 100.000 adults (2,3).

A 56-year-old man was admitted with a history of abdominal pain, nausea, and vomiting for 3 months. He had experienced postprandial fullness for the past month and lost 6 kg in weight. He had no prior history of drug use. He had no history of food allergies. His clinical examination was remarkable for abdominal distension and mild upper abdominal tenderness with palpation. Laboratory tests on admission revealed a WBC count of 21780/mm^3 with an eosinophil count of 0.8% (normal 0.5%-6%). Serum amylase was 757 U/L (normal 28-100 U/L) and serum lipase was 386 U/L (normal 22-51 U/L). Serum IgE level was 650 U/L (normal < 140 U/L). Other laboratory tests including liver function tests, routine stool studies, serum lipid profile, and serum immunoglobulins, were normal. An ultrasound and CT scan of the abdomen revealed a dilated stomach with retained food and mild thickening of the duodenal folds. The pancreas was normal, and there were no gallstones. The patient was treated conservatively with rehydration and nasogastric suction. EGD showed ulcers and thickened duodenal folds with duodenal narrowing. Multiple 2-6 mm ulcerated nodules were noted in the duodenum, with thickening of the duodenal folds extending into the second part of the duodenum (Figure 1 A, B). Biopsies of the duodenum showed chronic duodenitis with intense eosinophilic infiltration of the lamina propria and muscularis mucosa (Figure 2 A, B). The patient was treated with steroids, and his symptoms of duodenal obstruction resolved.

In EGE, eosinophilia is present in 80% of cases. Further investigations should include endoscopy for mucosal biopsy. To confirm the diagnosis of EGE, other causes leading to eosinophilic infiltration of the bowel must be ruled out, such as food allergy, drug idiosyncrasies, parasitic / helminthic infestation, connective tissue disorders, vasculitis,

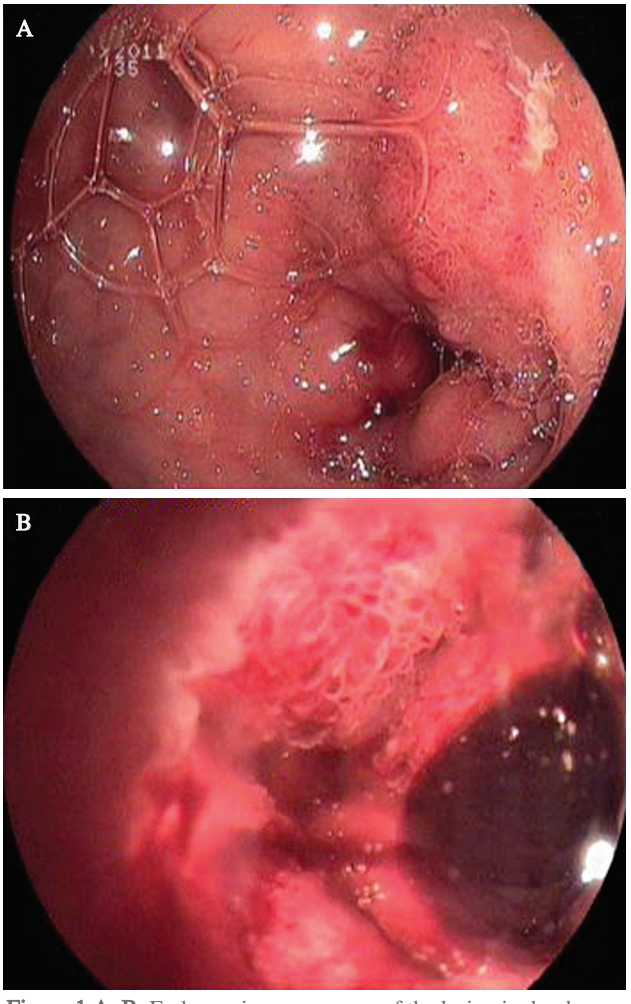

Figure 1 A, B. Endoscopic appearances of the lesion in duodenum

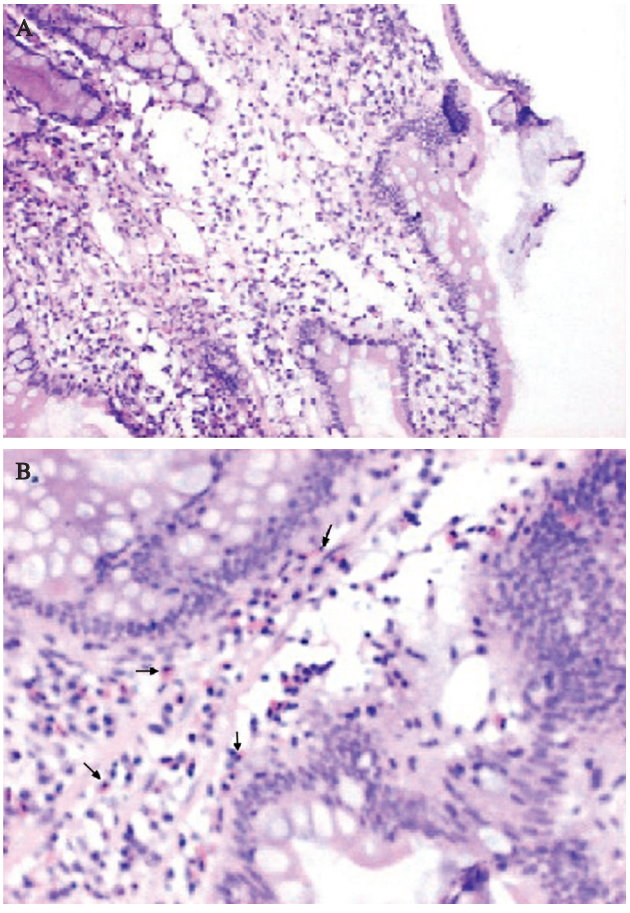

Figure 2. (A) Microscopic appearance of eosinophil infiltration in lamina propria and muscularis mucosa (Hematoxylin eosin, x40) **(B)** arrows, eosinophils (Hematoxylin eosin, x100)

malignancy, Crohn's disease, and non-tropical sprue. Another important differential diagnosis is the hypereosinophilic syndrome. The syndrome presents with longer than 6 months of persistent eosinophilia and may also be present in extraintestinal organs (skin, lymph nodes, heart, lungs, liver, spleen, central nervous system, etc..) (4,5).

We underline the importance of recognising EGE, since proper treatment can prevent further mucosal damage and progression to severe malabsorption and malnutrition.

In our case, eosinophilic infiltration in the duodenum, resulting in luminal obstruction of the second part of duodenum, was encountered as a complication of acute pancreatitis. This is a rare condition. In conclusion, the diagnosis of eosinophilic gastroenteritis should be considered in the patients with a duodenal mass.

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