nophilic esophagitis, gastritis, enteritis, and colitis. Eosinophilic gastroenteritis can be classified based on the location of the eosinophilic infiltrate in the gastrointestinal wall, as well as associated symptoms. Eosinophils in eosinophilic gastroenteritis frequently infiltrate the gastrointestinal mucosa and, only rarely, the muscular or subserosal layers (4). Mucosal disease generally presents with bleeding, protein-losing enteropathy, or malabsorption. Involvement of the muscle layer may cause bowel wall thickening resulting in subsequent intestinal obstruction. The subserosal form usually presents with peritonitis and eosinophilic ascites, as in our patient. The histopathologic diagnosis of eosinophilic gastroenteritis requires the presence of greater than or equal to 25 eosinophils on microscopic examination, which was the case in the patient presented (5). Eosinophilic colitis in our patient was characterized by numerous eosinophils extending from the muscular wall of the colon to the subserosa, and even involved the serosal surface of the colon. In our case, severe eosinophilic colitis with serosal involvement was the likely cause of this patient's eosinophilic ascites.

In conclusion, clinicians should have suspicion of eosinophilic gastroenteritis when forming a differential diagnosis regarding gastrointestinal symptoms. Definite diagnosis is made by histopathological assessment. Treatment with steroids is the mainstay in the management of eosinophilic gastroenteritis. Clinical improvement is usually seen after treatment with a low dose of steroid.

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Isolated Crohn's disease of the esophagus

Özefagusta izole Crohn hastalığı

To the Editor,

Crohn's disease (CD) is a chronic inflammatory disease of unknown etiology characterized by chronic, granulomatous, segmental transmural inflammation that may occur in any part of the alimentary tract from the mouth to the anus. In the human upper digestive tract, the esophagus is the segment least commonly involved in CD (1,2). Further, almost all esophageal CD has coexisted with the disease of such sites as the ileum, rectum and colorectum, with only 12 cases in the literature having been described as isolated esophageal involvement of CD (1,3,4). Here, we report a patient with isolated esophageal CD who underwent esophagectomy for severe esophageal stricture with spontaneous perforation into the mediastinum.

A 60-year-old man was admitted to our hospital with complaints of progressive dysphagia for over two months, with sudden appearance of heartburn

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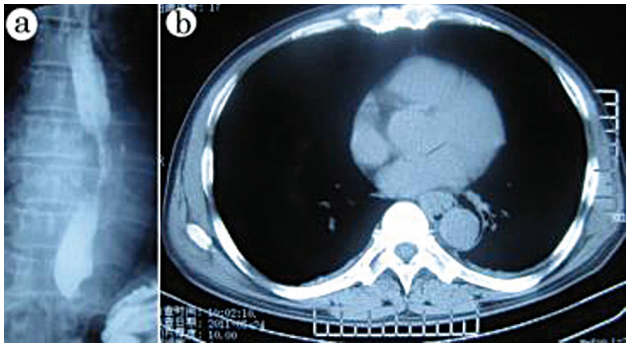

Figure 1. a. Irregular narrowing of the middle esophagus in barium swallow examination. **b.** Thickened middle esophageal wall, with pneumomediastinum and bilateral effusion in CT scan.

for seven days, without fever, cough, diarrhea, or abdominal pain. On admission, a barium swallow examination showed an irregular narrowing of the middle esophagus (Figure 1a). Esophagoscopy revealed the presence of a circular stricture of the esophagus at 25 cm from the incisor teeth, with only the pediatric gastroscope able to pass the stricture (Figure 2a). His stomach and duodenum appeared normal. A thoracic computed tomography scan revealed a thickened middle esophageal wall, with pneumomediastinum (Figure 1b). An esophagectomy was performed immediately. During the operation, the soft tissue around the esophagus was found to be edematous with pneumomediastinum. The resected specimen showed circular esophageal stricture with a deep, linear, longitudinal, multi-layered ulceration (20x15 mm) perforated into the mediastinum, and all layers of the esophagus were thickened (Figure 2b). The postoperative pathology confirmed a chronic, noncaseating granulomatous inflammation with

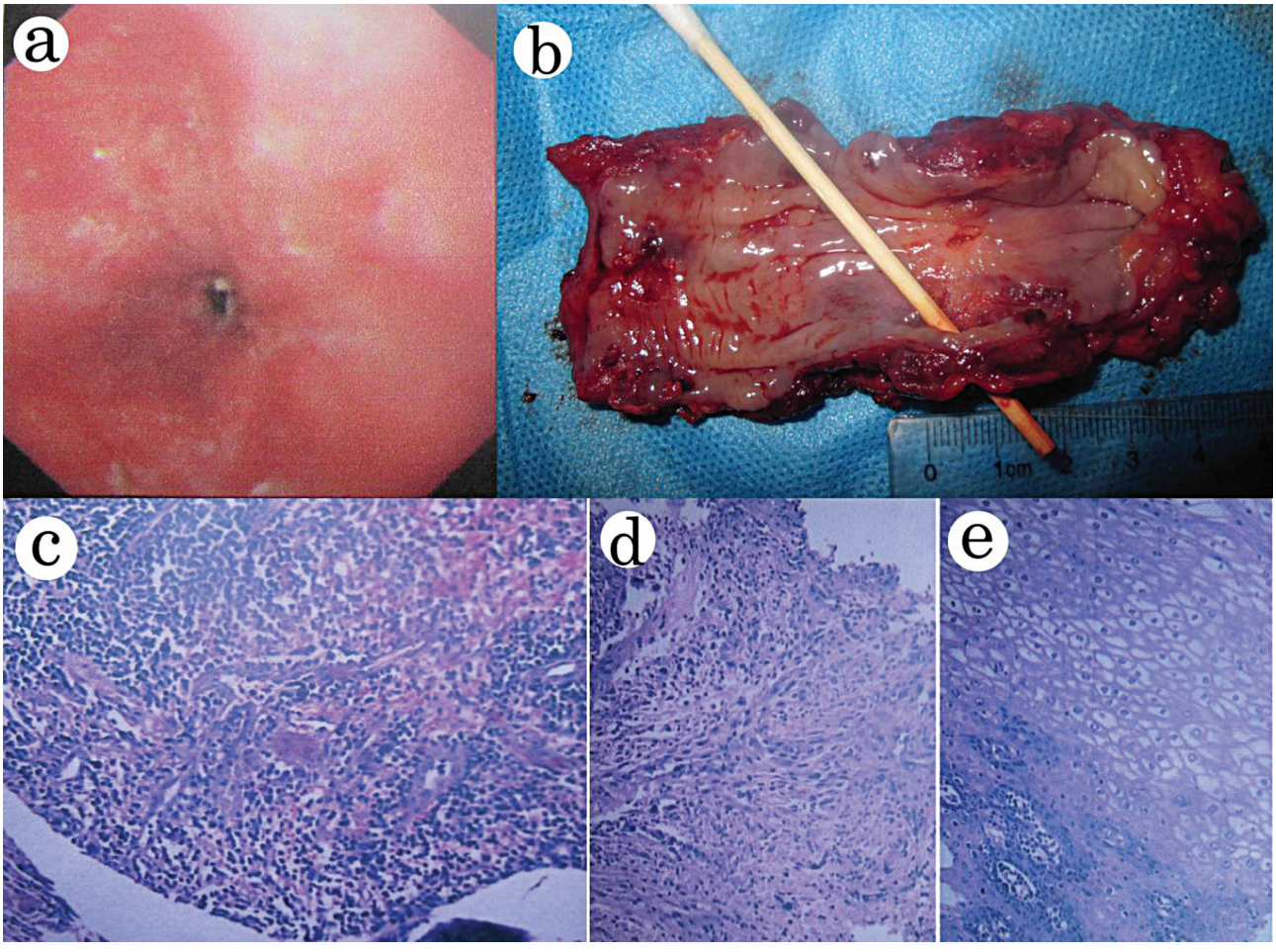

Figure 2. a. Endoscopy revealed circular stricture of the esophagus 25 cm from the incisor teeth. **b.** Gross specimen of the resected esophagus showed a deep, linear, longitudinal, multi-layered ulceration (20x15 mm) perforated into the mediastinum, and all layers of the esophagus were thickened, but the mucosa was smooth. **c.** Pathology examination showed chronic inflammation of the ulcerative esophagus (hematoxylin and eosin [H&E], original magnification 10x10). **d.** Pathology showed noncaseating granulomatous inflammation of the submucosa tissue (H&E, 10x10). **e.** Pathology showed squamous cell proliferation of the esophageal mucosa (H&E, 10x30).

lymphocyte cell infiltration and squamous cell proliferation (Figures 2c, d, e). No abnormalities of other digestive tract sites including the terminal ileum were found through further endoscopy. The patient was diagnosed as isolated esophageal CD. The postoperative course was uneventful.

Esophageal CD is rare, with an adult prevalence of 0.2–3% in patients with coexisting ileocolonic disease, and very few cases of isolated esophageal involvement have been reported (1,2). Due to lack of presentations of other digestive tract sites and that little consideration of isolated esophageal CD is given, the accurate diagnosis and treatment are often made rather late in its course when severe dysphagia secondary to stricture or other complications has occurred, necessitating surgical intervention3,5,6. Previous studies have reported fistula drainage to the pleural cavity, bronchus, esophageal wall, and even the stomach (3,5,7,8). Our case had an esophageal fistula to the mediastinum due to end-stage isolated esophageal CD. Esophagectomy was performed in a timely manner, when the mediastinal infection was limited. This case highlights features of the end stage of the disease, revealing that early discovery along with surgical intervention may bring promising results.

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Albendazole-induced toxic hepatitis: A case report

Albendazole bağlı bir toxic hepatitis

To the editor,

Echinococcus or hydatid cyst (HC) is a parasitic disease, that is among the important health problems of developing countries (1,2). According to data from the Turkish Ministry of Health, there are 2000 patients per year and HC case incidence is 6.3 / 100,000 in Turkey (3).

Albendazole is an anti-helmintic agent with a wide anti-parasitic spectrum and is indicated for usage in HC. Its side effects include nausea and vomiting, constipation, vertigo, headache, hair loss, and itching. It is metabolized in the liver. The most common reason necessitating withdrawal or cessation of the drug is major hepatic toxicity (4).

We report herein a case with rarely published toxic hepatitis induced by administration of albendazole treatment for HC.

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