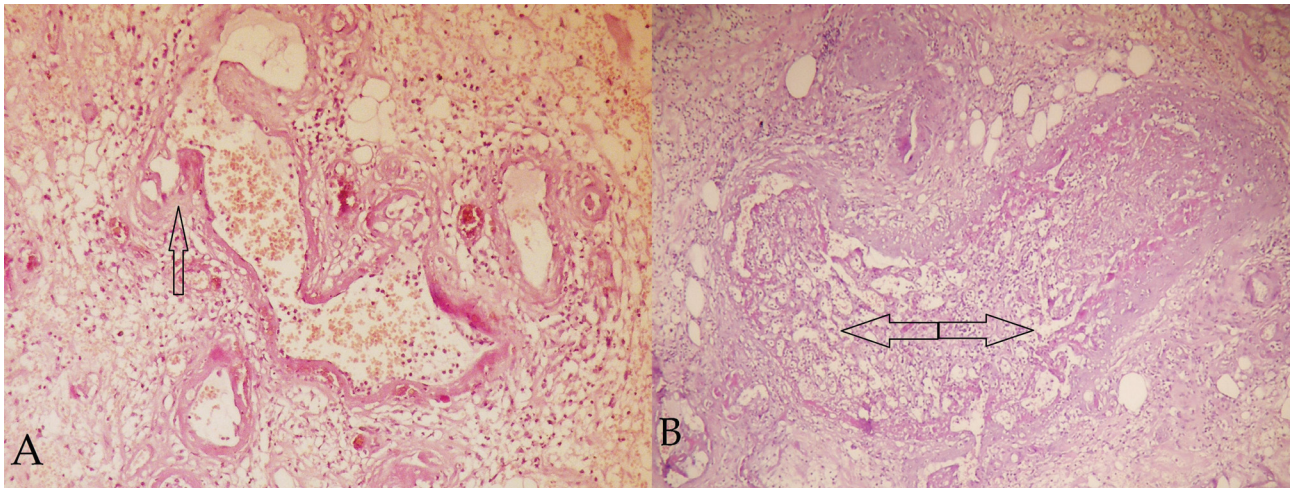

Figure 2. Histological image taken from the resected colon (hematoxylin and eosin stain, original magnification x 100): perivascular fibrin accumulation in mesenteric vein **(A)** and necrotizing venulitis characterized by polymorphonuclear leukocyte infiltration; **(B)** mesenteric vein indicating thrombotic occlusion.

se because of its rarity, nonspecific clinical findings and frequent confusion with other diseases, and requires histopathologic verification (4).

In conclusion, MIVOD is a rare mesenteric vasculitis. Quick diagnosis and surgical resection for this etiologically unknown disease are life-saving.

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An unexpected cause of occult bleeding

Okkült kanamanın tahmin edilmeyen bir nedeni

To the Editor,

A previously healthy 38-year-old female presented with melena and weight loss of 6 kg over two months. There was no associated abdominal pain, vomiting or anorexia. She had no family history of

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malignancy and denied use of non-steroidal anti-inflammatory drugs (NSAIDs). The admission hemoglobin was 4.4 g/dl. Colonoscopy revealed fresh and clotted blood throughout the colon, but the site of bleeding could not be identified. The mucosa was normal and no growth or tumor was seen. Capsule endoscopy examination revealed evidence of active bleeding at about 25 cm distal to the pylorus. A contrasted computed tomography (CT) of the abdomen and pelvis showed an enhancing mass, which was lobulated and exophytic at the D3 junction, measuring 4x5x6 cm in size.

The patient underwent a Whipple procedure and the histopathological diagnosis was duodenal gastrointestinal stromal tumor (GIST) (Figure 1A). The immunohistochemistry stained strongly positive for CD117 and negative for S100 (Figure 1B).

The patient recovered well and was followed with periodic CT scan to monitor the disease progression. The patient remained well two years after the operation, with no evidence of tumor recurrence.

GISTs are uncommon visceral sarcomas predominantly found in the GI tract. They are mesenchymal tumors of the GI tract with variable malignant potential. The reported annual incidence is about 1.2 cases per million population (1-3). Based on immunohistochemistry and electron microscopy examination, GISTs may derive from various tissues, including myogenic or neural attributes, mixed features or undifferentiated types. The exact cellular origin of GIST has been proposed to be the interstitial cell of Cajal, which is the intestinal pacemaker cell. It is increasingly recognized as a subtype of sarcoma with positive c-kit or CD117 growth factor receptor expression. The presence of the c-kit proto-oncogene is the critical factor in the pathogenesis of GIST due to its constitutive activation of the kit receptor tyrosine kinase (4). The outcome of malignant GIST is exceptionally poor. This tumor is resistant to radiotherapy and conventional chemotherapeutic agents. Curative surgical resection has been the only available treatment of choice, but surgery alone is often inadequate for advanced disease. Recently, the long-term survival has markedly improved with the use of c-kit-targeted therapy like the tyrosine kinase inhibitor, imatinib mesylate. A study involving 113 patients with primary and advanced GISTs with follow-up of up to 37 months of imatinib treatment showed clinically meaningful regression of the tumor on CT scan imaging. CT imaging has an important role in monitoring the disease progression, as there are no other established tumor markers for this tumor (5).

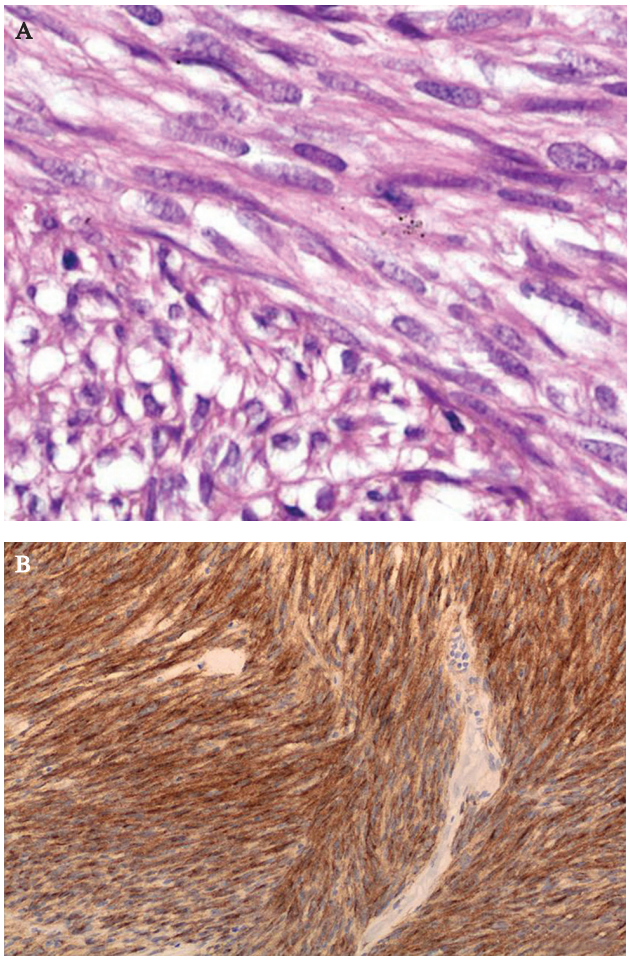

Figure 1. Histopathologic specimen of the patient stained with hemotoxylene- eosin **(1A)** and with CD117 **(1B)**.

In conclusion, GISTs should be considered when occult bleeding is suspected in the small intestine. Although the diagnosis can be made histologically, the presence of CD117 is pathognomonic. Imatinib as an adjuvant chemotherapy after resection is promising. Collection of data and case records of GIST should be integrated as part of observational evidence to enhance the understanding of the natural history of the disease. In addition, the role of capsule endoscopy as a diagnostic tool in cases of obscure GI bleeding is promising, as shown in our case. It provides adequate information for the initial diagnosis of GISTs.

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Primary giant hydatid cyst of the diaphragm

Diyafragmanın primer dev kist hidatiği

To the Editor,

The clinical course of hydatid disease (HD) may have different aspects [1]. It can be found in any part of the body, but is most often located in the liver (50-93%) [2-4]. Diaphragmatic localization is rare, with an incidence of 0.3-1.2%, and most of these are generally associated with liver disease [5]. Isolated diaphragmatic location (primary) of the cyst, not associated with the liver or lung, is very rare [4]. The aim of this work was to report a case with giant hydatid cyst located on the abdominal side of the left hemidiaphragm.

A 52-year-old female patient was admitted to our department with symptoms of mild abdominal pain, nausea, vomiting, weight loss, and intraabdominal mass. On the physical examination, a protruding mass lesion was palpated in the epigastrium and left upper quadrant. Indirect hemagglutination for HD was positive to a dilution of 1:640. Abdominal computed tomography (CT) (Figure 1) revealed a thickened-wall, cystic lesion (15 cm in diameter) with smooth borders adjacent to the stomach. The origin of the cyst was not clear. The esophagogastroduodenoscopy showed a diffuse gastritis.

A midline laparotomy was performed. During the exploration, a 15 cm in diameter cystic lesion was palpable, originating from the left hemidiaphragm. The cyst was palpable, but only a 5x5 cm part of the cyst could be seen; most of the cyst was located behind the stomach. The liver was normal. Cystotomy was performed and daughter vesicles were removed.

Because the cyst had a broad base on the left hemidiaphragm, total cystectomy was not possible. Partial cystectomy of the diaphragmatic cyst, atypical gastrectomy and splenectomy were performed due to the dense adhesions. The postoperative period was uneventful. Pathological diagnosis confirmed the HD. The patient was discharged on the 10th postoperative day, and albendazole therapy was started. The postoperative course of our patient during the follow-up period (18 months) was uneventful.

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