mucosal distortion (Figure 3). A combination of oral mesalazine, ursodeoxycholic acid, and prednisone treatment was started. There was no diarrhea during follow-up, proteinuria decreased, and renal function improved at third month. There were no significant changes in the liver function tests. The patient presented to emergency room with complaints of fever (39ºC) and shortness of breath. His chest X-ray showed diffuse interstitial pulmonary infiltrates. The patient died from an opportunistic infection (pneumonia) ten days after hospitalization.

In conclusion, here, we reported a rare case of PSC and FSGS, which concomitantly occurred prior to IBD.

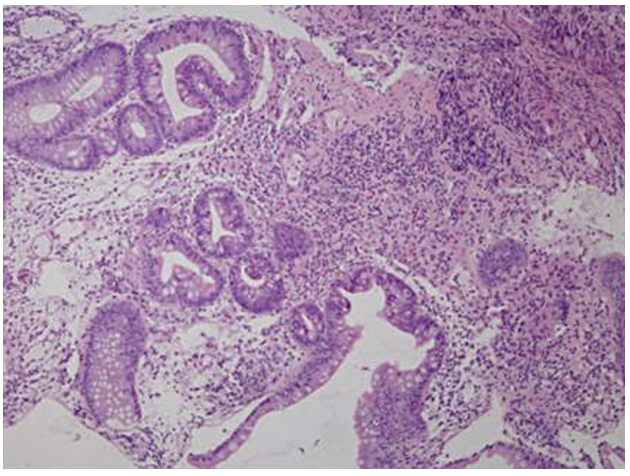

Figure 3. Intestine (x10 HE).

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Pancreatic lymphoma presenting with acute pancreatitis

Akut pankreatitle prezente olan pankreas lenfoması

To the Editor,

Primary pancreatic lymphoma (PPL) is a very rare disease, accounting for less than 0.5% of pancreatic tumors (1). The clinical presentation of PPL is usually nonspecific. Most common presenting symptom reported in the literature is abdominal pain, found in about 83% of cases, followed by (in descending order of frequency) abdominal mass (58%), weight loss (50%), jaundice (37%), acute pancreatitis (12%), small-bowel obstruction (12%), and diarrhea (12%) (2). There are few cases of PPL presenting with severe pain and acute pancreatitis in the literature (3).

A 45-year-old female presented to emergency department with worsening abdominal pain. Abdominal examination showed epigastric tenderness. The patient's laboratory findings on admission we-

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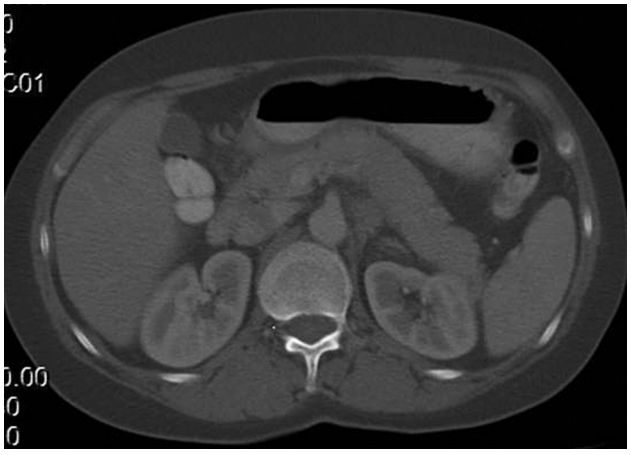

Figure 1. Computed tomography (CT) showed a 4.0×6.5 cm homogeneously enhancing mass localized superior of the pancreatic corpus.

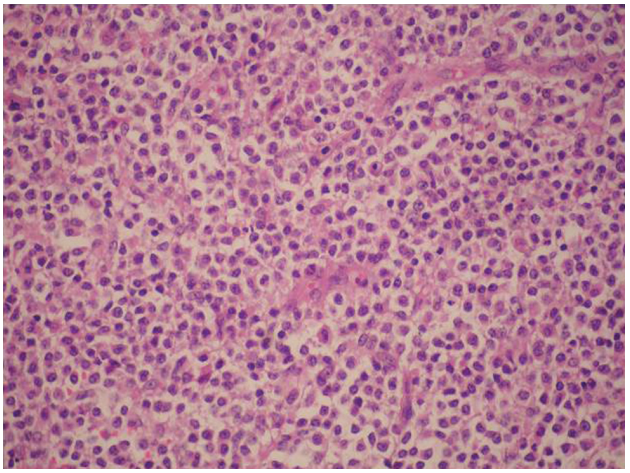

Figure 2. Diffuse large B-cell lymphoma (HEx400).

re as follows: white blood cell count $12.6\times10^3/\mu L$, hemoglobin 13.6 g/dL, platelet count $250\times10^3/\mu L$, creatinine 1.1 mg/dL, total protein 6.9 g/dL, total bilirubin 1.3 mg/dL, liver enzymes aspartate aminotransaminase 94 IU/L and alanine aminotransferase 82 IU/L, pancreatic enzymes amylase 1215 IU/L and lipase 987 IU/L; C-reactive protein was 0.8 mg/dL. Abdominal ultrasound was normal. The patient was admitted to the hospital with the diagnosis of acute pancreatitis. After hospitalization, computed tomography (CT) was done which showed a 4.0×6.5 cm homogeneously enhancing mass localized superior of the pancreatic corpus. The common pancreatic duct and the common bile duct were normal (Figure 1). On physical examination, tumor and whole body lymph nodes were not palpable. After CT, the patient was diagnosed with pancreatic corpus mass and distal pancreatectomy was performed. Histopathology showed large tumor cells without cell adhesion or tissue structure proliferation (Figure 2), and immunochemical staining was positive for B cell markers CD20 and CD79 and negative for T cell marker CD10 (Figure 3). Pathological examination was compatible with non-Hodgkin's diffuse large B-cell lymphoma. The patient's postoperative course was uneventful, and she was discharged from the hospital on the 19th postoperative day. A month later, she was referred to medical oncology department for chemotherapy.

The majority of PPLs are of the B-cell type (4). The 1-year survival for B-cell PPL (51.9%) has been reported to be higher than that for T-cell PPL (0%) (5). PPL can mimic pancreatic carcinoma (2). The

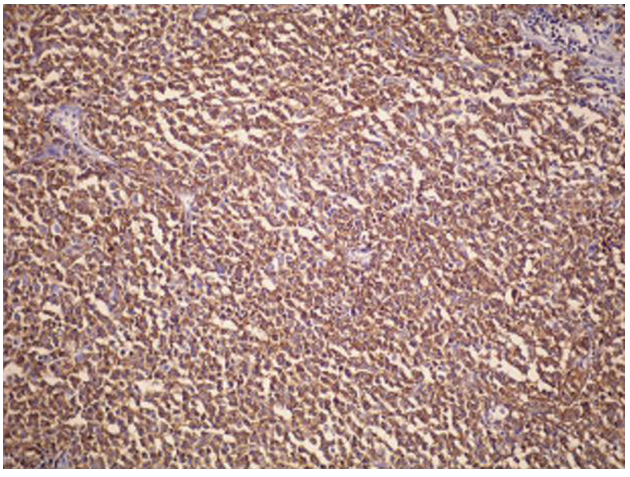

Figure 3. Diffuse CD20 positivity in the tumor (immunoperoxidase X20).

treatment and prognosis of pancreatic cancer and pancreatic lymphoma are quite different, and the differential diagnosis of these two diseases is therefore very important (6). Significant, however, is the fact that PPLs are potentially treatable (7). Regarding the treatment of PPL, chemotherapy is considered as standard.

It is advocated that surgery should be reserved for cases where percutaneous or endoscopic biopsies are not diagnostic, or treatment with chemotherapy and/or radiation therapy is not successful (8). On the other hand, for patients with symptoms caused by obstruction of biliary tract, surgical treatment may be effective (9).

PPL can present with severe abdominal pain and acute pancreatitis. This clinical presentation can masquerade the real diagnosis.

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A case of acute acalculous cholecystitis during the course of reactive Epstein-Barr virus infection

Reaktif Epstein-Barr virus enfeksiyonunda gelişen akut taşsız kolesistit olgusu

To the Editor,

A previously healthy 8-year-old girl was admitted to our hospital with a seven-day history of fever, abdominal pain, and vomiting. On physical examination, exudative tonsillopharyngitis and cervical adenopathy were noticed. Abdominal examination revealed tenderness in the right upper quadrant. The liver and spleen were both enlarged. Laboratory investigations revealed a white blood cell count of 10.240/mm^3 (74% lymphocytes); alanine aminotransferase (ALT) 496 IU/L (N: 0-39 IU/L), aspartate aminotransferase (AST) 569 IU/L (N: 0-36 IU/L), alkaline phosphatase (ALP) 434 IU/L (N: 118-360 IU/L), gamma-glutamyltransferase (GGT) 103 IU/L (N: 0-23 IU/L), total serum bilirubin 4,6 mg/dL with a direct fraction of 3,2 mg/dL. Throat, blood and urine cultures showed no growth. Ultrasonographic (US) examination of the abdomen demonstrated a markedly thickened, edematous gallbladder wall with pericholecystic fluid and no evidence of gallstones or dilatation of the biliary tract. IgG antibodies for Epstein–Barr viral capsid antigen (VCA) were positive at a level of 62.21 (reference: <9), IgG antibodies for Epstein-Barr nuclear antigen (EBNA) were positive at a level of 87 (reference: <5), EBV DNA was detected by real-time polymerase chain reaction (PCR) and quantified in plasma with a viral load of 74.000 copies/mL confirming the diagnosis of reactive Epstein-Barr

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