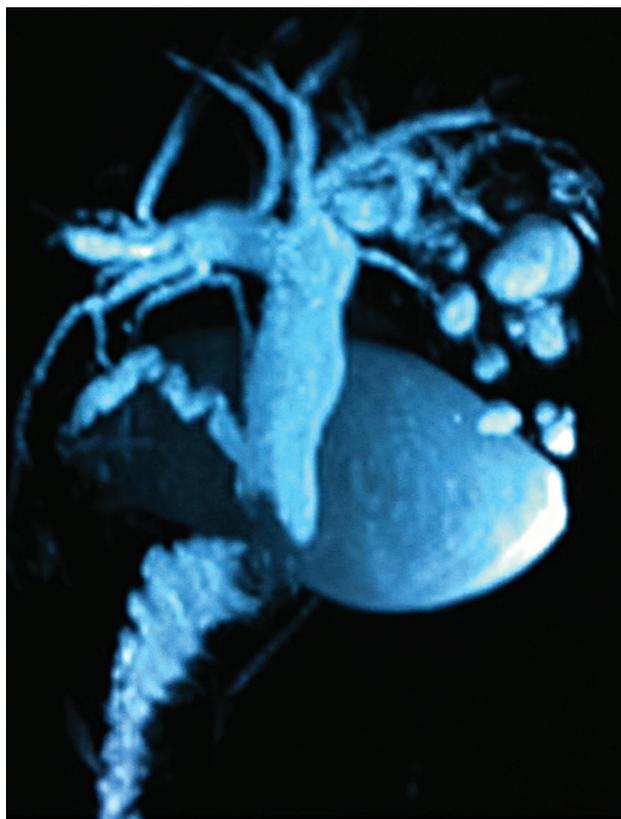


## Cystic hydatid disease of the gallbladder

*Safra kesesinin hidatik kist hastalığı*

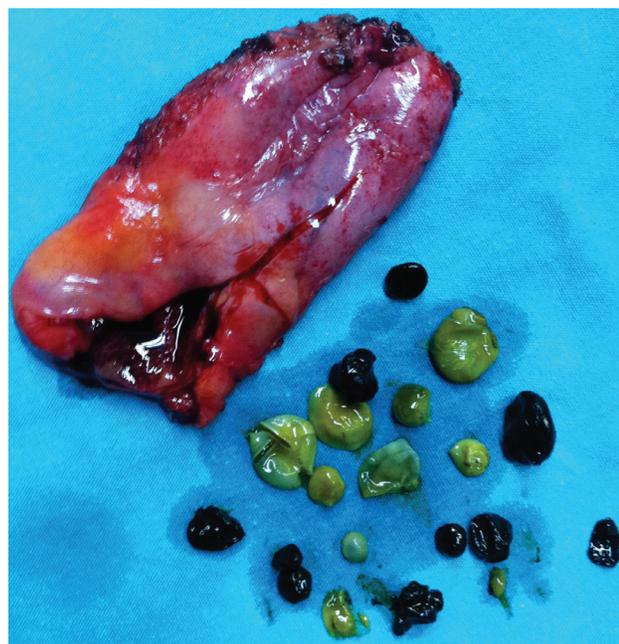
To the Editor,

A 48-year-old male was admitted to our hospital complaining of abdominal pain and jaundice. His body temperature was 36.7 °C, blood pressure 120/70 mmHg and heart rate 76/min. His physical examination revealed mild tenderness in the upper right quadrant of the abdomen and visible jaundice. He had peptic ulcer disease and had been placed on oral antacid treatment. His liver function tests revealed: aspartate aminotransferase (AST): 108 IU/L, alanine aminotransferase (ALT): 186 IU/L, alkaline phosphatase (ALP): 220 IU/L, total bilirubin: 7.1 mg/dl, direct bilirubin: 5 mg/dl, and white blood cell count 13,000 /mm<sup>3</sup>. Gallbladder distension and dilated choledochus were seen



**Figure 1.** Magnetic resonance cholangiopancreatography revealed the obstruction in the distal choledochus.

in ultrasonography. Additionally, there was a cystic, 3 cm lesion in segment 4b that was interpreted as an inactive hydatid disease. His serology was positive for *Echinococcus granulosus* and tumor markers were negative. He denied any *E. granulosus* diagnosis or treatment in his previous medical history. Computed tomography revealed that he had an inactive calcified hydatid cyst in liver segment 4b and choledocholithiasis, and one of the bile stones was noted to be in the neck of the gallbladder. A significant obstruction in the distal choledochus was shown in the magnetic resonance cholangiopancreatography (MRCP) (Figure 1). In the endoscopic retrograde cholangiopancreatography (ERCP), sludge and millimetric bile stones were extracted from the duodenal papilla after sphincterotomy, and the procedure was ended with plastic biliary stenting. His bilirubin levels normalized after ERCP under parenteral antibiotic therapy. He underwent laparoscopic cholecystec-



**Figure 2.** Hydatid cysts were extracted from the lumen of the gallbladder after laparoscopic cholecystectomy.

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tomy. The calcified lesion was compatible with inactive hydatid cyst. The cholecystectomy was finished uneventfully. Before sending the specimen to the pathology laboratory, we cut the wall of the gallbladder and saw the hydatid cysts (Figure 2). The patient was discharged on postoperative day one and placed on 400 mg/day albendazole treatment. Chronic inflammation and hydatid cyst membranes were observed in the histopathological evaluation of the gallbladder.

*E. granulosus* is responsible for hydatid disease, most commonly in the liver. This infection is usually seen in sheep- and cattle-raising regions. Cholangitis with obstructive jaundice is a rare and dreadful complication of hydatid cysts ruptured into the biliary tract. In addition to the diagnostic capabilities of ERCP, it is a treatment tool for obstructive pathologies of the bile duct (1,2). ERCP

shows a cystobiliary relationship, and we did not observe any communication between the gallbladder and the calcified cystic lesion in the liver (3). A false-positive result of the serologic analysis should be kept in mind in patients with previous hydatid disease and no evidence of the disease currently (4). In this case, choledocholithiasis had been concluded as a main cause of the obstructive jaundice after ERCP preoperatively. In such patients, laparoscopic cholecystectomy is the best treatment strategy for preventing further cholangitis attacks. After surgery, macroscopic and pathologic findings revealed a cyst hydatid disease of the gallbladder in the patient, which is a very rare clinical entity (5). ERCP is used to diagnose and treat biliary pathologies. Removal of the gallbladder reduces further risks causing obstructive jaundice and helps in the definitive diagnosis.

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## A rare PRSS1 mutation in a Turkish family with hereditary chronic pancreatitis

*Hereditær kronik pankreatiti olan bir Türk ailede nadir bir PRSS1 mutasyonu*

*To the Editor,*

Hereditary pancreatitis (HP) is an autosomal dominant-inherited disorder characterized by recurrent attacks of pancreatitis with the development of chronic pancreatitis in the absence of known etiologic factors (1,2). R122H and N29I mutations

are the most common PRSS1 mutations that play a causal role in chronic pancreatitis worldwide (3-5). Herein, we report a rare PRSS1 mutation causing typical HP in a Turkish family. The polymorphism is called D21A and results in a substi-

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