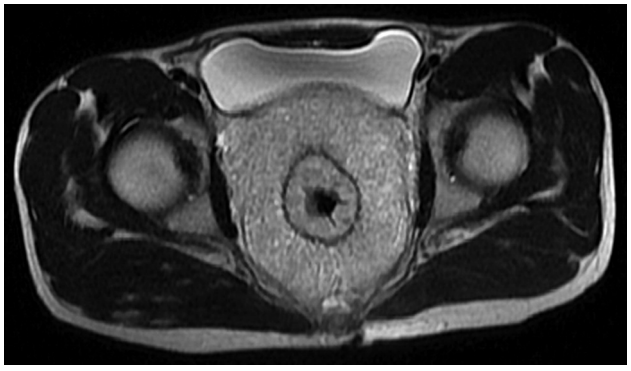

Figure 1. Axial T2-weighted image of the rectosigmoid colon wall and perirectal mass shows high signal intensity.

mucosal ulceration overlying the hemangioma usually cause the rectal bleeding (3). Massive hemorrhage is the major cause for the 50% mortality rate associated with this condition (4).

There are seven reports in the literature that describe the MR imaging findings (1,2). The MR imaging features of this disease are very characteristic, as there is no known disease that presents hyperintensity of the rectosigmoid wall and perirectal fat on the T2WI, as in this disease. Hemorrhoid should be considered in the differential diagnosis. Hemorrhoids usually involve only the anal region. Perirectal fat abnormalities and marked bowel wall thickening are not seen in hemorrhoids, and are easily shown by MR imaging. T2WI hyperintensity may be seen in severe inflammatory conditions such as Crohn's disease and ulcerative colitis. These diseases accompany significant colon wall enhancement.

Thus, computerized tomography and MR provide information about the border of the mass and relations to adjacent structures, which is necessary before surgical treatment. MR imaging findings of diffuse cavernous hemangioma of the rectosigmoid colon are reliable, specific, and valuable in the diagnosis.

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Rabeprazole-induced acute cholestatic liver injury

Rabeprazolün indüklediği akut kolestatik karaciğer hasarı

To the Editor,

Proton pump inhibitors are widely used drugs in the treatment of peptic ulcer and gastroesophageal reflux disease (GERD). In addition to their well-documented efficacy, these drugs are generally well tolerated. There are only a few case reports concerning omeprazole-, pantoprazole- and lansoprazole-related hepatotoxicity (1). There is also only one report in the English literature about rabeprazole-associated hepatotoxicity (2). Herein, we present a new case of rabeprazole-induced acute hepatotoxicity.

A 46-year-old male was admitted to the gastroen-

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terology outpatient clinic with heartburn and regurgitation for more than a year. His medical history was unremarkable; there were no administrations of any other medications, herbal products or alcohol. Coulter blood count, liver enzymes and renal function tests were in normal limits on admission. Upper gastrointestinal endoscopy revealed lower esophageal sphincter relaxation, and rabeprazole 20 mg/day was prescribed for non-erosive GERD. One week after the treatment was started, he admitted again with jaundice. Biochemical parameters performed on readmission were as follows: alanine transaminase (ALT): 374 IU/ml (normal: 0-40 IU/ml), aspartate transaminase (AST): 298 IU/ml (normal: 0-40 IU/ml), alkaline phosphatase: 470 IU/ml (normal: 0-270 IU/ml), γ-glutamyl transpeptidase: 148 IU/ml (normal: 0-38 IU/ml), total bilirubin: 8.3 mg/dl (normal: 0.1-1 mg/dl), and direct bilirubin: 5.2 mg/dl (normal: 0.1-0.3 mg/dl). Albumin and prothrombin time were within the normal ranges. Serological markers for acute viral hepatitis were negative. In addition, laboratory tests for autoimmune hepatitis, hemochromatosis, thyroid diseases, and Wilson's disease were also unremarkable. Abdominal ultrasonography showed nothing pathological. After cessation of rabeprazole, liver enzymes and bilirubin levels returned to normal within the 15th day. However, one month later, he restarted rabeprazole himself because of his repeated GERD symptoms. Jaundice recurred three days after restarting the drug, and he admitted to the hospital again. In the laboratory analysis, liver enzymes and bilirubin level had increased again (AST: 88 IU/ml, ALT: 110 IU/ml, total bilirubin: 4.8 mg/dl, direct bilirubin: 2.9 mg/dl). We confirmed again that he had not used any other medication, herbal product or alcohol, and rabeprazole was ceased again. Bilirubin and liver enzymes gradually normalized in four weeks.

Drug-induced hepatotoxicity is a frequent cause of liver injury, and accounts for less than 5% of the cases with jaundice or acute hepatitis (3). Patients with drug-induced liver injury can have a similar presentation to that of other causes of hepatobiliary disease (3,4). The first case in the literature with rabeprazole-induced hepatotoxicity was also receiving terbinafine, which was also hepatotoxic. Later, in a correspondence letter, rabeprazole was defined as the lower- potential hepatotoxic drug in that case report (5). In our patient, the absence of evidence of other liver disease or other hepatotoxic agents together with the normalizing of liver enzymes and bilirubin levels after discontinuation of the drug on two occasions led us to the diagnosis of rabeprazole-induced hepatotoxicity.

In conclusion, rabeprazole is an effective drug for the treatment of peptic ulcer and GERD, but physicians should be aware of the potential for hepatotoxicity due to rabeprazole.

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