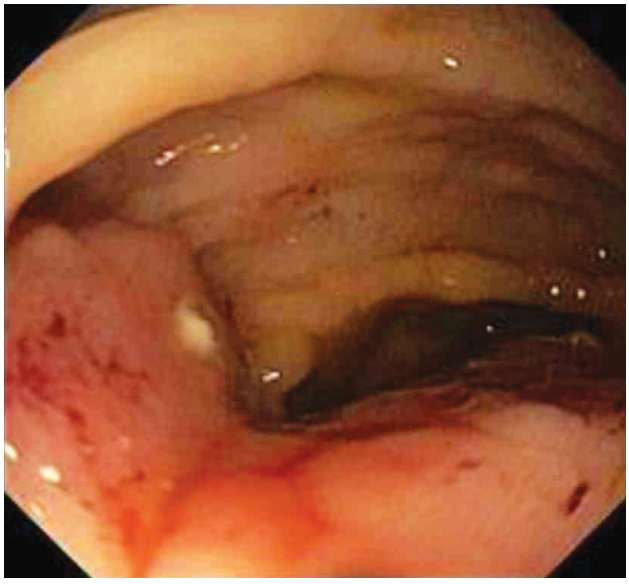

Figure 1. A polyploidy mass in the second part of duodenum.

shown to be non-existent by tomography imaging. After completion of treatment, the patient followed –up in remission for 2 years.

The true etiology and pathogenesis of germ cell tumors in the GIS is yet to be determined. Investigators have accepted the retro-differentiation theory, which shows a retro differentiation of the adenocarcinoma cells to the level of the embryonic ectoderm, and subsequently, a metaplasia or de-differentiation to trophoblastic precursor cells (2). The most common manifestations of GIS metastasis are intestinal obstructions and/or gastrointestinal bleeding (4). Some biochemical markers are important in the diagnosis and treatment of GCT, including HCG, AFP, and LDH. However in our case with a duodenum GCT, serum AFP was high. GIS GCTs are more frequently seen in young patients. Generally, primary GCTs have a good prognosis. GCTs should be kept in mind in the differential diagnosis of young male patients that have malignant evidence presenting with GIS bleeding. The treatment of GIS GCTs should be a multidisciplinary approach including and not limited to surgery, chemotherapy, and radiotherapy.

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HLA subtypes and *Helicobacter pylori* infection in an infant with celiac crisis

Çölyak krizli bir infantta HLA subtipleri ve Helicobacter pylori enfeksiyonu

To the editor,

The term celiac crisis has been used to describe the acute, fulminant form of celiac disease (CD) that is associated with hypoproteinemia and edema (1). Factors regarding the frequency of disease and types of presentation are unknown. In this letter we present an infant with CD whose initial

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presentation was consistent with celiac crisis, and we discuss probable relationships between human leukocyte antigen (HLA) tissue typing, *Helicobacter pylori* infection, and celiac crisis.

A thirteen-month-old male patient was admitted with acute onset severe diarrhea, abdominal distension, and weight loss. On physical examination the patient showed signs of tachycardia, hypotension, abdominal distention, and severe dehydration. Laboratory tests were consistent with mild anemia, hypokalemia, hyponatremia, hypocalcemia, hypoalbuminemia, metabolic acidosis and a prolonged coagulation profile. Blood and stool cultures were negative. Microscopic examination of the stool was normal, however steatorrhea was detected. Dilated intestinal loops were found through plain radiography and ultrasound. Serum immunoglobulin levels and sweat test were both normal. Anti-endomysial, anti-gliadin antibodies, and tissue transglutaminase were all positive.

After these results, celiac crisis was diagnosed and 2 mg/kg/day prednisolone treatment was initiated. Total parenteral nutrition was initiated instead of oral nutrition. After corticosteroid therapy, clinical symptoms and laboratory findings improved gradually. At the fifth day of treatment, oral nutrition with elemental formula was administered. Corticosteroid therapy was slowly tapered over the course of three weeks. Upper gastrointestinal endoscopy was performed afterwards. Duodenal mucosa was observed to be edematous and pale. The results of biopsies obtained from duodenal and gastric mucosa demonstrated Marsh 3c destructive duodenum, confirming the diagnosis of celiac disease with *H. pylori* positive chronic gastritis (Figure 1). Eradication therapy for *H. pylori* infection was then started.

In HLA typing analysis HLA-ABC class I: A*24, B*50, B*58, CW*03, CW*06, HLA, DQ, DR class II: DRB1*07, DRB1*11, DRB3, DRB4, DQB1*02, DQB1*03 were determined. On the 25th day since admission to the hospital, the patient was discharged from the hospital with a gluten free diet.

Celiac crisis can not only be treated through the regulation of the diet (1,2). As supportive therapy goes on, it may be necessary to add corticosteroid therapy. The dose and duration of corticosteroid therapy remain controversial (1-3). We used 2 mg/kg/day of prednisolone until the patient responded to the therapy.

Coexistence of celiac disease and HLA-DQ2, DQA1*0501, DQB1*02, or DQ8 (DQA1*03, DQB1*0302) has been reported (4,5). In literature, there is no report about the relationship between celiac crisis and HLA. However, it has been reported that patients who are homozygous for the allele of DQB1*0201 have presented with more severe clinical symptoms, a late onset of disease, severe atrophy of the villi, severe diarrhea, and lower levels of hemoglobin (4). In our presented case, the patient was heterozygous for the allele of HLA-DQB1*0201. This may be related to the development of celiac crisis.

In our case, celiac crisis has been found to be associated with *H. pylori* infection as well. There is no reported literature regarding this association. However, there have been reports about preventation

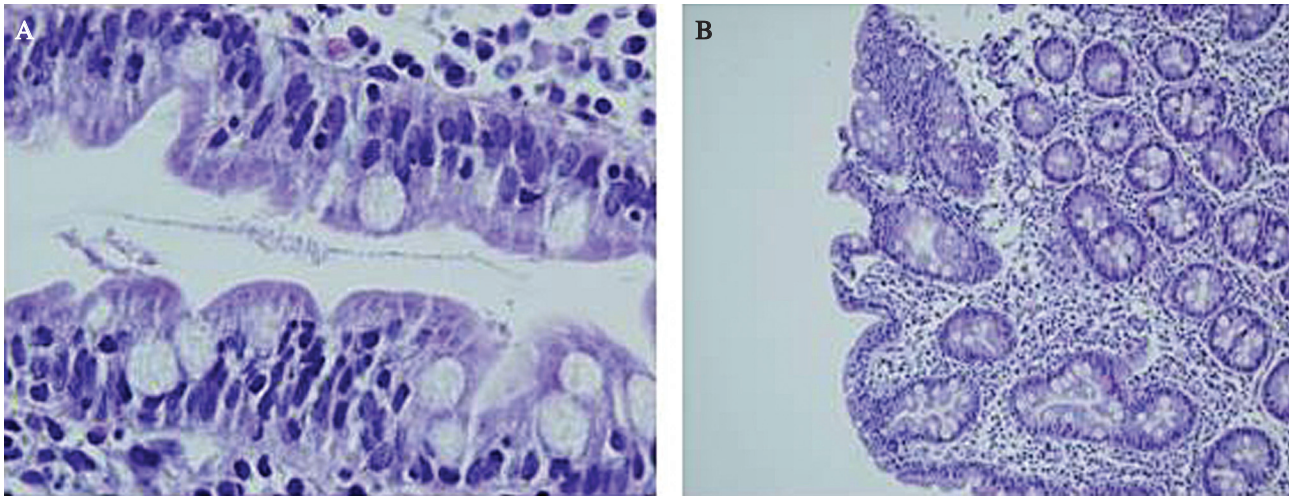

Figure 1. A. demonstrates intrepithelial lymphocytic infiltration. **B.** demonstrates villous atrophy and cryptic hyperplasia.

from *H. pylori* infection in patients with a history of celiac disease (6,7). Unlike developed countries in the world, the incidence of *H. pylori* infection is higher at younger ages in developing countries like Turkey (8). As seen in our case, *H. pylori* infection may deteriorate the clinical status, or it may have been found incidentally.

In conclusion, it should be remembered that celiac disease may present as a celiac crisis in infancy. In the case of acute severe diarrhea with metabolic acidosis, hypokalemia, and hypoalbuminemia resistant to supportive treatment, celiac crisis should be considered in the differential diagnosis. Further investigations are needed to determine the relationships between HLA typing, *H. pylori* infection, and celiac crisis.

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A rare cause of elevated liver enzymes: Addison's disease

Nadir bir karaciğer enzimleri yüksekliği nedeni: Addison hastalığı

To the Editor,

Mildly elevated serum aminotransferase levels (<250 IU/L) occasionally may be due to endocrine diseases and disorders. Diabetes mellitus, hypothyroidism, and hyperthyroidism are well-recognized endocrine diseases that may cause increased serum aminotransferase activity. Olsson et al. first reported that Addison's disease, another endocrine disease, may be another one of the reasons we see mildly elevated serum aminotransferase levels (1). To date, a total of 14 cases have been published in the literature (1-8). It is not clear why liver enzymes increase in Addison's disease. Boulton et al. have suggested that chronic hypo-perfusion may be the underlying mechanism for the abnormal liver biochemistry values (2). Another hypothesis by Rizvi et al. has proposed that the liver test abnormalities may be due to an immunologic reaction within hepatic tissue (4). An alternative explanation is that apoptosis and necrosis of hepatocytes, induced by local release of cytokines by infiltrating lymphocytes, may occur. This phenomenon can be reversed by glucocorticoid replacement therapy (5). Here, we report a patient with Addison's disease presenting with high serum liver transaminase levels, and normalization of these transaminases after administration of corticosteroid treatment.

A 49 year old woman was admitted to the hospital with a two month history of weakness, fatigue,

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