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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Table 1. Laboratory data of the patient during follow-up

	Admission	2 weeks after treatment	3 months after discharge
Cortisol (4.3-22.4 ug/dL)	5,9	-	-
ACTH (0-46 pg/mL)	> 1250	> 1250	11.8
AST (5-34 IU/L)	120	33	16
ALT (5-49 IU/L)	87	21	9
Total bilurubin (0.3-1.2 mg/dl)	0.6	0.6	0.4
Direct bilurubin (0.0-0.2 mg/dl)	0.2	0.2	0.1
ALP (25-129 IU/L)	112	81	52
GGT (1-38 IU/L)	118	58	23
LDH (0-190 IU/L)	240	224	192
Na (135–145 mmol/l)	120	138	141
K (3.5–5.5 mmol/l)	5.9	5.0	4.4
BUN (10–50 mg/dl)	48	34	32
Creatinine (0.6–1.3 mg/dl)	1.2	0.8	0.8
Glucose (70–115 mg/dl)	78	86	88

ACTH: Adrenocorticotropic hormone. AST: Aspartate aminotransferase. ALT: Alanine aminotransferase. ALP: Alkaline phosphatase. GGT: Gamma glutamyl transferase. LDH: Lactate dehydrogenase. Na: Sodium. K: Potassium. BUN: Blood urea nitrogen.

and increased skin pigmentation. On physical examination, she had hyperpigmentation on her hands, feet, and knees. Her laboratory tests showed low serum cortisol, 5.9 ug/dL (4.3-22.4 ug/dL) and increased adrenocorticotropic hormone (ACTH), >1250 pg/mL (0-46 pg/mL). Serum aspartate aminotransferase and alanine aminotransferase were 120 IU/L (5-34 IU/L) and 87 IU/mL (5-49 IU/L), respectively. Also, serum aldosterone level was 40 pg/mL (70-300 pg/mL), renin level was 11 ng/mL (0.2-1.6 ng/mL). Increased skin pigmentation, low serum cortisol, and high ACTH levels suggested Addison's disease and the diagnosis was confirmed by lack of cortisol response to 250 mcg ACTH stimulation test. 21-hydroxylase antibody was found to be positive. She was also evaluated for hypertransaminasemia. Serum alkaline phosphatase, gamma-glutamyl transferase, total bilirubin and albumin levels were all normal. Also, all viral hepatitis markers were negative. Tests for anti-smooth muscle antibody, anti mitochondrial antibody, and antinuclear antibody were all negative. Abdominal computed tomography showed no pathology within the liver and adrenal glands. After confirmation of Addison's disease by ACTH stimulation test, corticosteroid therapy was initiated. Nearly two weeks after corticosteroid replacement therapy aminotransferase activity returned to normal, and continued to normalize for three months after replacement therapy (Table1). For patients with constantly elevated liver enzymes, extra-hepatic diseases have to be considered. Although rare, Addison's disease should be kept in mind.

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