high C-reactive protein (CRP, 15 mg/dl), erythrocyte sedimentation rate (ESR, 62 mm/hour) and alanine aminotransferase (ALT, 45 U/L). Multi-slice computed tomography (CT) and magnetic resonance imaging (MRI) showed a thick-walled spherical mass containing centrally cystic areas and smooth margins, without evidence of peripheral invasion, in segment 6 of the posterior right hepatic lobe (Figure 1). Due to atypical CT and MRI findings, percutaneous biopsy of the mass was performed. Inflammatory pseudotumor (IPT) was diagnosed based on the histopathological findings. Treatment with antibiotic (cefuroxime axetil 500 mg) and anti-inflammatory (naproxen sodium 550 mg) drugs was ordered for two weeks. At the follow-up visit at the end of the first month, the mass was 60-70% smaller, and the borders of the mass were less apparent on ultrasonography.

Hepatic IPT is an idiopathic, rare, benign, and tumor-like lesion. It is a granulomatous reaction containing plasma cells, chronic inflammatory cells and sometimes histiocytes around a fibrous stroma (1), and it is usually seen in the lungs. Involvements of the orbita, oral cavity, parotid gland, pleura, liver, stomach, ovary, and retroperitoneum have been reported in the literature less frequently (2). The etiology of IPT is still unknown; infections, autoimmunity and biliary obstruction are the known possible causes. It is more common in males, and the average age of onset is 35 years (3-5).

Clinical symptoms of IPT are fever, epigastric pain, vomiting, general weakness, weight loss, and jaundice. Leukocytosis and increased ESR may be seen (4,5). In the differential diagnosis of IPT from other tumors, one of the most important findings is negative tumor markers, which was valid for our patient as well (5). The mass was diagnosed incidentally in our case. Except for higher levels of CRP and ESR in our case, there were no clinical symptoms or abnormal laboratory findings. As a result, although IPT is usually symptomatic, in some rare cases, it may be asymptomatic and incidentally detected. If an atypical contrast pattern is seen in a mass of the liver, IPT should be kept in mind.

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## Hepatitis A-associated immune thrombocytopenia

Hepatit A-immün trombositopeni birlikteliği

To the Editor,

Immune thrombocytopenia (ITP) is the most frequently encountered autoimmune disorder related with blood cells, and the acute form is generally seen in children following viral infection or vaccination. Epstein-Barr virus, parvovirus, human immunodeficiency virus (HIV), and hepatitis C

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**Table 1.** Laboratory parameters of the patients

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Days	Hb (g/dl)	WBC (/mm³)	Plt (/mm³)	AST (IU/L)	ALT (IU/L)	T. Bil mg/dl	D. Bil mg/dl	Anti HAV Total	Anti HAV IgM
Case 1 Onset	9.3	8900	10000	470	350	8.2	4.5	1.2	14 (+)
Day 3	9.1	7600	50000	640	830	9.4	5.4		
Day 30	9.6	7800	254000	28	34	1.1	0.4	16	0.4
Case 2 Onset	10.7	4400	4000	1143	1262			4.4 (+)	10.2 (+)
Day 7	9.7	8100	7000	1065	1204	1.7	1.4		
Day 12	9	13000	35000	48	231				
Day 30	11.1	9400	206000	22	11			12.06	00.22

Hb: Hemoglobin, WBC: White blood cells. Plt: Platelets. AST: Aspartate aminotransferase. ALT: Alanine aminotransferase. T. Bil: Total bilirubin. D. Bil: Direct bilirubin. HAV: Hepatitis A virus.

Case 1. Day 1: IVIG treatment 1 g/kg/day.

Case 1. Day 3: The patient was discharged. Case 2. Days 2-5: IVIG treatment 1 g/kg/day.

Case 2. Day 8: Onset of steroid treatment.

Case 2. Day 12: The patient was discharged.

and B viruses (HCV, HBV) are among the viruses causing ITP. Hepatitis A virus (HAV)-associated ITP is a rare condition (1). Two cases of ITP associated with hepatitis A infection will be presented herein.

An eight-year-old boy was admitted with diffuse ecchymosis on the both extremities. The platelet count was 10,000/mm<sup>3</sup> in the hemogram. Alanine aminotransferase (ALT) and aspartate aminotransferase (AST) were 350 and 470 IU/L, respectively. HAV-IgM positivity was detected. The patient was given 1 g/kg intravenous immunoglobulin (IVIG) treatment for three days, and his platelet count reached 50,000/mm<sup>3</sup> on the third day. The second case, a four-year-old boy, was admitted due to ecchymosis measuring 3x4 cm on the left frontal area and subcutaneous hematoma. In the hemogram, platelet count was 1,000/mm<sup>3</sup> (Table 1). ALT and AST were 1262 and 1143 IU/L, respectively. HAV-IgM positivity was also detected in this patient. Acute hepatitis A infection with associated ITP was diagnosed. IVIG 1 g/kg/day was administered for three days, but there was no change in his platelet count. After bone marrow aspiration with increased immature megakaryocytic cells, oral methylprednisolone 2 mg/kg was started. His platelet count reached 30,000/mm<sup>3</sup> on the third day of the treatment.

Immune thrombocytopenia (ITP) developed in our patients during the HAV infection. In the literature, a limited number of cases have been reported (2-4). Response to IVIG facilitates the treatment process, but as seen in our second case, steroid treatment should be considered and planned in those unresponsive to IVIG. Hepatitis A has a benign process, but fulminant cases have been reported as well (5). In the second case, no response was obtained with daily IVIG 1 g/kg for three consecutive days. When transaminase was at the reduction threshold, steroid was initiated, and the platelet count increased. Another case report with the same clinic was managed with high-dose steroid treatment, and hematological recovery of the thrombocytopenia was detected. However, at the admission, serum transaminase levels of this child were about two-fold normal levels, probably at the time of recovery of hepatitis, such that the timing of the steroid treatment was comparable with the treatment in our second case (6).

It is concluded that ITP cases are rarely associated with acute hepatitis A infection. HAV- associated ITP is not a rare situation in our country, in which HAV infection is frequent. We report that this rare disease can be treated with steroids if the patient is resistant to IVIG.

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