Unusal presentation of liver dysfunction in the later stages of pregnancy

Gebeliğin geç dönemlerinde ortaya çıkan nadir karaciğer disfonksiyonu olgusu

To the Editor,

Severe liver disorders may be observed in the later stages of pregnancy. Acute fatty liver of pregnancy (AFLP), preeclampsia and HELLP syndrome (hemolysis, elevated liver enzymes and low blood platelet count) are the main causes of severe hepatic failure in pregnancy (1, 2). As these disorders have similar signs and symptoms, distinguishing between them can be difficult. We report herein a patient with acute liver dysfunction who was diagnosed as TTP (thrombotic thrombocytopenic purpura) with very high levels of blood bilirubin.

A 30-year-old pregnant woman (gravida 2, para 1), who was in the 37th week of gestation, was admitted to the Obstetrics and Gynecology Department with a 10-day history of myalgia, vomiting and malaise.

The laboratory tests revealed high levels of blood bilirubin, lactate dehydrogenase (LDH) and uric acid levels, slight rise in transaminase levels and coagulation tests, and moderate increase in blood urea and creatinine levels. Thrombocytopenia was also detected. Serologic tests for human immunodeficiency virus (HIV), hepatitis A, B, C and Epstein-Barr virus (EBV) were negative. The results of laboratory testing at the time of admission are presented in Table 1.

Physical examination revealed scleral icterus, impaired consciousness and mild arterial hypertension. She had no history of arterial hypertension, liver disease or alcohol or drug abuse during the pregnancy. She had a history of diet-controlled gestational diabetes mellitus. Her weight gain was approximately 11 kilograms during the pregnancy period.

On the suspicion of HELLP syndrome, labor was induced with oxytocin infusion, but fetal distress necessitated cesarean section despite the patient's impaired coagulation tests. At the end of the procedure, the patient was admitted to the obstetric clinic in good condition, and magnesium sulfate infusion was started in the immediate postoperative period. The patient's laboratory values and vital signs were stable in the first few hours after the operation; however, at the postoperative sixth hour, she developed anuria and became comatose, clinically jaundiced and hypotensive.

The patient was transferred to the intensive care unit (ICU) and central venous access was obtained. On admission to the ICU, body temperature (axillary) was 37°C, with heart rate of 142 bpm, blood pressure of 60/30 mmHg and SaO2 of 98%. After dopamine and aggressive fluid and electrolyte management, urine output and blood urea and creatinine values returned to normal levels. The patient's consciousness improved. No signs of cholestasis or other liver abnormalities were observed on the abdominal ultrasound.

In the following days, progressive increase in bilirubin levels, thrombocytopenia and high levels of LDH necessitated performing plasmapheresis, which was continued daily over the following 10 days.

On the 10th day, thrombocyte count returned to normal values. Coagulation tests and liver function tests also returned to normal but bilirubin levels remained high. Fragmented red cells were observed in the peripheral smear. Autoimmune antibodies (ANA, anti dsDNA, anti SMA) were detected as negative.

Liver biopsy performed on the 12th postpartum day showed obstructive cholestasis and fibrosis, but the results of the biopsy provided no clues to establishing a definitive diagnosis of the liver disease.

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On the 14th day of the delivery, she was discharged from the hospital with medication including ursodeoxycholic acid. At the two-month follow-up, her total bilirubin level was 4.7 mg/dl and she was in good health.

Progressively increasing bilirubin levels (maximum level of total bilirubin 52.4 mg/dl) and high levels of LDH with thrombocytopenia in the postpartum period were the major complications in this patient, and led us to consider TTP in the differential diagnosis.

An overview of the liver diseases in pregnancy shows that AFLP is a fulminant and progressive disease that can only be diagnosed by liver biopsy (2,). In AFLP and HELLP syndrome, total bilirubin levels usually do not increase to the extent observed in our patient. HELLP and preeclampsia are pregnancy-specific syndromes that usually show full recovery after the delivery is completed (3, 4).

Intrahepatic cholestasis of pregnancy (IHCP) is the most common liver pathology associated with pregnancy. It is usually seen in the third trimester and is characterized by pruritus and mild jaundice. Total bilirubin levels in IHCP are usually less than 5 mg/dl and it also usually resolves after delivery (2).

The combination of elevated liver enzymes and thrombocytopenia accompanied by hemolysis in this patient resembled HELLP syndrome; however, after the delivery, despite first returning to normal levels, her laboratory values remained at pathological levels with severe thrombocytopenia, hemolysis and progressive rise in bilirubin level.

The patient showed some benefit from plasmapheresis, which again pointed to TTP as the initial differential diagnosis. The diagnosis of TTP can be made after further tests that can reveal fragmented red blood cells, as in our patient, and autoantibodies against ADAMTS13, though the latter test is not yet available in our country (5).

Fibrosis with hepatocanalicular cholestasis was observed in the liver biopsy. This may indicate that TTP might have been superimposed on her old asymptomatic liver pathology. Ursodeoxycholic acid resulted in improvement in laboratory values, which also supports the fact that there was some ongoing cholestatic pathology of the liver (2).

Hemolytic uremic syndrome (HUS) should also be remembered in the differential diagnosis. Generally, HUS presents with renal failure that usually occurs immediately after the delivery or within a few weeks. However, it is not always possible to make an exact distinction between TTP and HUS in clinical diagnosis (6). This patient's clinical symptoms suggested a disease mostly likely related with a liver pathology.

Our literature review also revealed an unusual case of liver disease that was found to be caused by leptospirosis; however, the test for leptospirosis in this case was negative (1).

Most often, liver pathologies seen in the third trimester of pregnancy resolve after delivery; however, progressive diseases that can be accompanied by multi-system organ failure may lead to maternal morbidity and mortality. Definitive diagnosis is sometimes difficult, but supportive therapy should be the main target of the treatment and must begin as soon as possible.

Table 1. Biochemical parameters of the patient

	Prepartum	**1st day**	**2nd day**	**10th day**	**Postpartum 1st month**	**Postpartum 2nd month**
Urea (mg/dl)	30	30	33	9	10	10
Creatinine (mg/dl)	1.6	1.8	1.7	0.2	0.4	0.6
Uric acid (mg/dl)	10.1	9.8	10.1	7	4	3
Bilirubin, total (mg/dl)	16.9	16.1	16.54	52.4	22.1	4.99
Bilirubin, direct (mg/dl)	11.4	10.3	11.0	31.1	15.3	3.4
AST (U/L)	40	132	112	79	154	45
ALT (U/L)	72	87	75	59	132	62
GGT (U/L)	248	81	92	105	252	144
Hb (g/dl)	16.1	7.87	10	10.2	11.8	14.1
Hct (%)	34.2	21.2	27.8	27.3	31.5	39.6
Platelet count (x 109/L)	91	56	55	41	290	283
INR	2.21	2.57	2.17	2.0	1.2	1
APTT (s)	48.4	66.3	42.6	40.4	26	24
PT (s)	24.7	28.9	24.4	22.4	13	12.5
LDH (U/L)	716	1163	1201	1002	200	128

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A rare cause of cholangitis: *Fasciola hepatica*

Nadir bir kolanjit nedeni: Fasciola hepatica

To the Editor,

Fascioliasis is a zoonotic disease caused by liver flukes (1.) Although Turkey is regarded as mesoendemic for the disease, there are few cases reported (2, 6, 7).

The disease has a broad clinical spectrum ranging from an acute hepatic phase to a chronic biliary phase. The diagnosis of fascioliasis can be established when the eggs of *Fasciola hepatica* are found in feces, duodenal aspirates or bile or by using various serological tests (3, 14, 15). Imaging studies are also helpful (8, 16-18).

A 58-year-old female patient admitted to the emergency department with fatigue, malaise, loss of appetite, and abdominal pain. Her medical history revealed hypertension and hyperlipidemia for two years. On physical examination, she had pain on the right upper quadrant and her scleras were icteric. Laboratory investigation results were as follows: glucose: 127 mg/dl, aspartate aminotransferase: 541 U/L, alanine aminotransferase: 886 U/L,

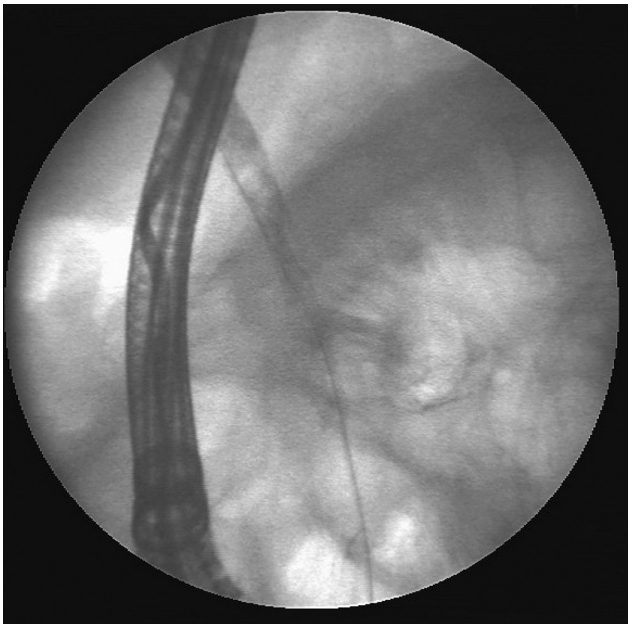

Figure 1. ERCP demonstrated dilatation in intrahepatic bile ducts and common bile duct.

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