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Akut Hepatit Nedeniyle Tanı Konulan Peripartum Kardiyomyopati

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ÖZET: Giriş:Şok karaciğeri, diğer olası nedenlerinin yokluğunda serum aminotransferaz seviyelerinin önemli ölçüde yükselmesi ile karakterizedir. Peripartum kardiyomiyopati (PPCM) gebeliğin son ya da postpartum dönemin ilk 5 ayında belirlenebilir kalp hastalığı yokluğunda meydana gelen idiopatik kalp yetmezliği durumudur.Olgu Sunumu: Kronik karaciğer ve kalp hastalığı öyküsü olmayan 27 yaşında bir kadın, olaysız spontan vajinal doğum sonrası 2 ayda akut hepatit ile hastanemize başvurdu. Nefes darlığının değerlendirilmesi için ekokardiyogram çekildi, hastanın ciddi kardiyomyopatisi olduğu ve ejeksiyon fraksiyonunun m-mod ekokardiyograf ile % 23, alan uzunluk ejeksiyon fraksiyonu ile % 22 olduğu saptandı. Uygun tedavi başlandıktan sonar akut hepatit ve dilate kardiyomiyopati tablosu ortadan kalktı.Tartışma ve Sonuç: İskemik hepatit akut hepatit tedavi edilebilir nedenlerinden biridir ve nedeni bilinmeyen akut hepatit ayırıcı tanısına her zaman dahil edilmelidir.

ANAHTAR KELIMELER: Akut hepatit, iskemik hepatit, peripartum kardiyomyopati, dilate kardiyomyopati.

ABSTRACT: Introduction: Shock liver is characterized by a marked elevation of serum aminotransferase levels in the absence of other potential causes of the liver injury. Peripartum cardiomyopathy (PPCM) is idiopathic heart failure occurring in the absence of any determinable heart disease during the last month of pregnancy or the first 5 months postpartum. Case Presentation: A 27-yr-old woman, 2 months postpartum from an uneventful spontaneous vaginal delivery, with no previous chroronical liver and heart disease history was admitted to our hospital with acute hepatitis. Echocardiogram was obtained for evaluation of dyspnea, patient was found to have severe cardiomyopathy with area length ejection fraction 22%, ejection fraction by m-mode echocardiography 23%. After initiation of appropriate treatment of dilated cardiomyopathy, acute hepatitis resolved. Discussion and conclusion: Ischemic hepatitis is one of the treatable causes of acute hepatitis and must always be included in the differential diagnosis of acute hepatitis with unknown pathogenesis.

KEY WORDS: Acute hepatitis, ischemic hepatitis, peripartum cardiomyopathy, dilated cardiomyopathy.

1. Introduction

Shock liver is characterized by a marked elevation of serum aminotransferase levels in the absence of other potential causes of the liver injury [1]. Peripartum cardiomyopathy (PPCM) is idiopathic heart failure occurring in the absence of any determinable heart disease during the last month of pregnancy or the first 5 months postpartum [2]. Hepatic dysfunction in congestive heart failure is due to hepatic congestion from venous hypertension and decreased oxygen delivery from an impaired cardiac output [3].

Case Presentation

A 27-year-old woman who was 2 months postpartum from an uncomplicated pregnancy and uneventful spontaneous vaginal delivery was transferred with complaints of cough, dyspnea, orthopnea,

PND and palpitation which began 1-wk after the delivery and progressed until the admittance. She had no medical history of chronic liver and heart disease; hepathotoxic medicine intake and herbal medicine usage. At admittance the patient had a temperature of 36.4 ºC, a pulse rate of 112 beats/min, a blood pressure of 104/73 mmHg, and a respiratory rate of 24 breaths/min. Findings on physical examination were inspiratory crepitations, S3 gallop rhythm; enlarged liver with no palpable spleen, no apparent ascites and no stigmata of chronic liver disease. Oculer examination of the patient showed no Kaiser-Fleischer rings.Laboratory testing revealed an increase in hepatic transaminases (AST: 1673 u/L, ALT: 2073 u/L), coagulopathy (INR: 2.0) and a decrease in hepathic synthesis function (ALB: 2.5g/dl). Viral hepatitis serologies were negative. ANA, ASMA, LKM 1 ab's and soluble liver ag were all undetectable. Telecardiography revealed an increase at the cardiothoracic index and ECG left bundle branch block. Abdominal USG showed an enlarged liver with patent, irregular, dilated hepatic vessels, without spleen enlargement and ascites. On echocardiography ALEF was 22%, MODEF was 23%. Patient was determined as ischemic hepatitis due to PPCM. Captopril 3X6.25 mg p.o, carvedilol 2X6.25 mg p.o, furosemide 2X40 mg i.v and dobutamine infusion 5μg/kg/min and vitamin K was administered and by day 5, cardiac output had improved, urine output was > 2L/day, liver enzymes were normalized and INR was < 1.5. After an 18-day hospitalization, the patient was discharged with ASA 1X100 mg p.o, carvedilol 2X6.25 mg p.o, perindopril 1X2mg p.o, and spironolactone 25 mg + hydrocholrothiazide 25 mg p.o. treatment. Three months after discharge she had no dyspnea, orthopnea or S3 gallop, with normal daily activities, liver enzymes, INR and echocardiography findings.

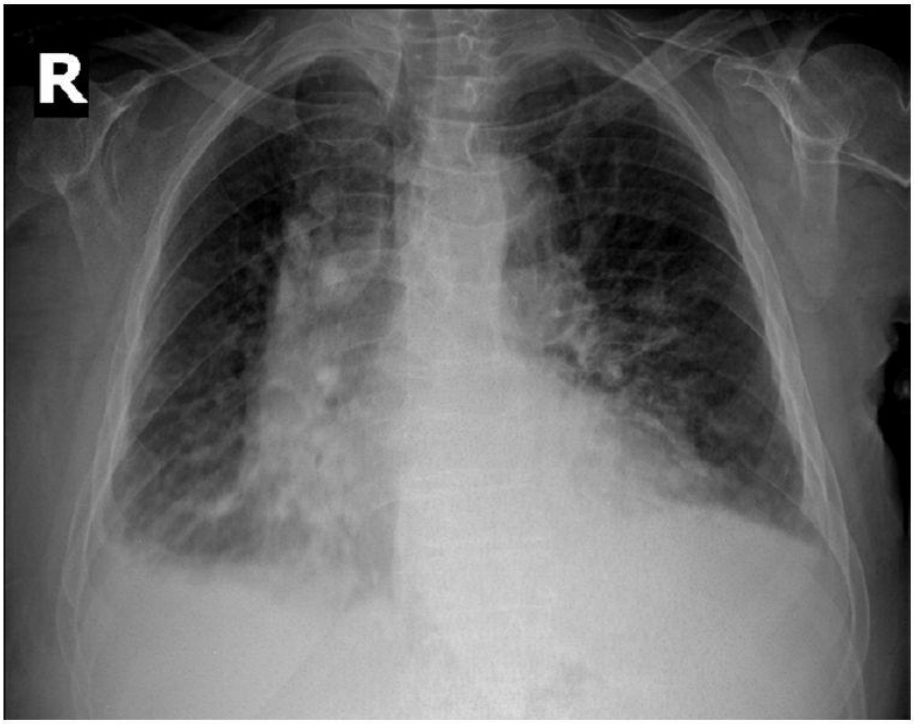

Figure 1. *Telecardiographic findings of the patient*

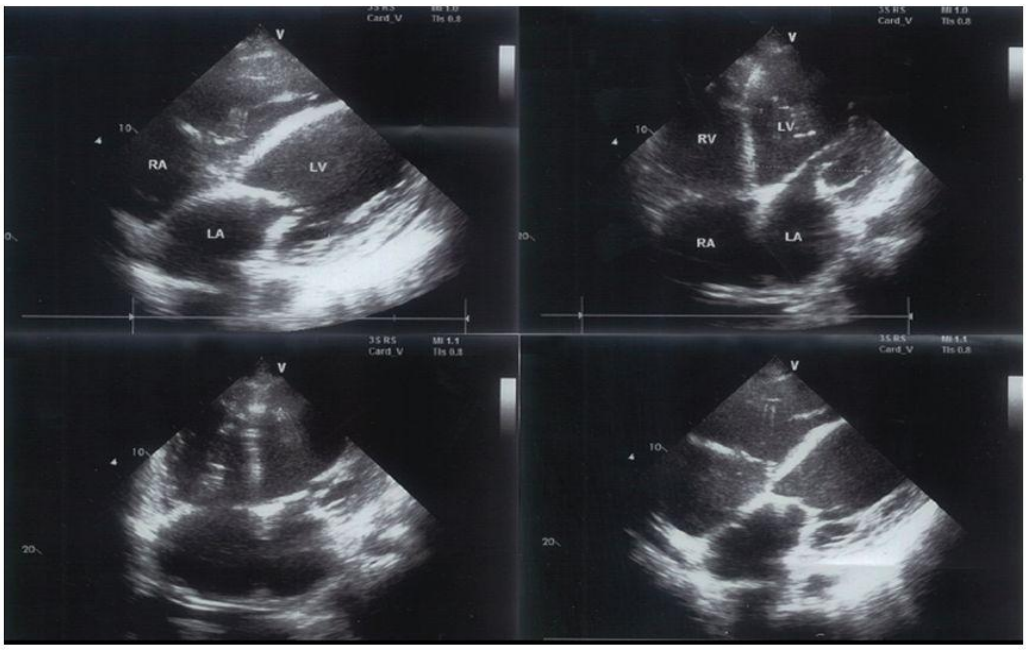

Figure 2. *Echocardiographic findings of the patient*

2. Discussion and Conclusion

Ischemic hepatitis is characterized by centrilobular liver cell necrosis as consequence of hypoperfusion with subsequent ischemia and passive congestion of the liver, severe systemic arterial hypoxemia, and/or impaired hepatic oxygen extraction. Diagnostic criteria are: clinical setting of acute cardiac, circulatory, or respiratory failure, dramatic but transient increase in serum aminotransferase activity reaching at least 20 times the upper limit of normal, exclusion of other putative causes of liver cell necrosis, such as viral, autoimmune, metabolic or drug-induced hepatitis [4]. The goal of the treatment is improvement of cardiac functions [5]. Incidence of PPCM is 1:2400 to 1:15000 pregnancies in the USA. Risk factors include advanced age, multiparity, black race, multiple gestation, obesity, preeclampsia and chronic hypertension. Although etiology of PPCM is unknown, suspected causes are infections, autoimmunity and nutritional defects [6]. The diagnosis of PPCM is based on four criteria, development of cardiac failure in the last month of pregnancy or within five months after delivery, absence of demonstrable cause for the cardiac failure, absence of demonstrable heart disease before the last month of pregnancy and documented systolic dysfunction [7]. Management of PPCM does not differ from other forms of congestive cardiac failure; sodium and fluid restriction, after load reduction, inotropic agents, anticoagulation and antiarrhythmic. Immunosuppressive therapy and cardiac transplantation can be considered in the cases refractory to conventional therapies [8]. In different studies mortality rates are between 7-50%. In > 50% of patients with PPCM the left ventricular ejection fraction normalizes generally during first six month of the presentation; among patients with no improvement in the left ventricular ejection fraction within six months of presentation prognosis is poor and mortality rate is 85% for five years [9].

Ischemic hepatitis is one of the treatable causes of acute hepatitis and must always be included in the differential diagnosis of acute hepatitis with unknown pathogenesis.

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