A case of peripartum cardiomyopathy presenting with complete heart block

Tam kalp bloku ile görülen peripartum kardiyomiyopati: Olgu sunumu

İlknur Can, M.D., Akif Düzenli, M.D., Bülent Behlül Altunkeser, M.D., Ahmet Soylu, M.D.

Department of Cardiology, Medicine Faculty of Selçuk University, Konya

A 33-year-old woman presented with unconsciousness and complete heart block 20 days postpartum. On admission, her blood pressure was 70/50 mmHg and heart rate was 30/min. Immediately, a temporary transvenous pacemaker was inserted via the right femoral vein and her blood pressure increased to 100/70 mmHg with restoration of consciousness. Echocardiographic examination showed reduced left ventricular systolic function. The following day, complete heart block resolved spontaneously to sinus rhythm with right bundle branch block. Ten days later, right bundle branch block resolved together with improvement in left ventricular ejection fraction. To our knowledge, a case of peripartum cardiomyopathy presenting with complete heart block has not been reported. Key words: Cardiomyopathies/etiology; heart block/etiology; postpartum period; pregnancy complications, cardiovascular.

Otuz üç yaşında kadın hasta doğumdan 20 gün sonra bilinç kaybı ve tam kalp bloku ile başvurdu. Başvuru sırasında kan basıncı 70/50 mmHg, kalp hızı 30/dk ölçüldü. Hastaya vakit kaybetmeden sağ femoral ven yoluyla geçici transvenöz pacemaker takıldı. Hastanın kan basıncı 100/70 mmHg'ye yükseldi, bilinci geri geldi. Ekokardiyografik incelemede sol ventrikül sistolik fonksiyonlarının bozuk olduğu izlendi. Bir gün sonra, tam kalp bloku kendiliğinden düzelerek ritim sağ dal bloklu sinus ritmine döndü. On gün sonra sağ dal bloku kaybolurken sol ventrikül ejeksiyon fraksiyonunda da iyileşme görüldü. Bilgilerimize göre, peripartum kardiyomiyopatisi ile tam kalp blokunun bir arada görüldüğü bir olgu bildirilmemiştir.

Anahtar sözcükler: Kardiyomiyopati/etyoloji; kalp bloku/etyoloji; postpartum dönemi; gebelik komplikasyonu, kardiyovasküler.

Peripartum cardiomyopathy (PPCM) is a rare cardiac disorder associated with high rates of mortality during the peripartum period. It is recognized as a distinct entity, separate from preexisting cardiomyopathies that are worsened by the stressors of pregnancy. Similarities between the symptoms of normal late pregnancy and early congestive heart failure presents a challenge in recognizing and diagnosing PPCM. Normal late pregnancy may be associated with symptoms of dizziness, dyspnea, fatigue, or pedal edema, all of which are present in patients with congestive heart failure. There are currently no specific clinical criteria to help differentiate between the symptoms of late normal pregnancy and heart failure. Therefore, the diagnosis of PPCM relies on a high index of suspicion in conjunction with the timing of symptoms and echocardiographic identification of new left ventricular systolic dysfunction, which includes depression of both fractional shortening and ejection fraction.^[1]

CASE REPORT

A 33-year-old postpartum woman was admitted to the coronary care unit because of complete heart block and unconsciousness. On admission, her blood pressure was 70/50 mmHg and heart rate was 30/min. She was cyanotic. The electrocardiogram at the coronary care unit revealed complete heart block with ventricular rate 27/min (QRS was wide) and atrial rate 150/min (Fig. 1). Her temperature was 36.6 °C. The high atrial rate was due to atropin administered in the emergency room. Immediately,

178 Türk Kardiyol Dern Arş

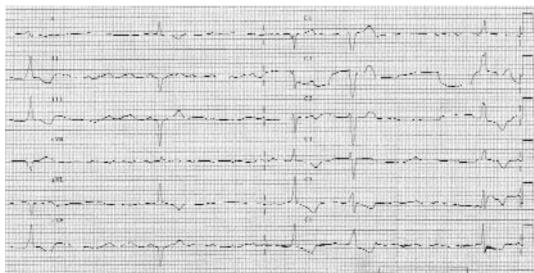


Figure 1. Complete heart block at the time of presentation.

a temporary transvenous pacemaker was inserted via the right femoral vein and her blood pressure increased to 100/70 mmHg with restoration of consciousness. She recalled that she was feeling weak and feverish about two or three days before her admission. Her medical history included no previous cardiac disease and two uneventful pregnancies. Twenty days before her presentation, she had her third successful vaginal delivery. Echocardiographic examination revealed reduced left ventricular ejection fraction (LVEF) (25%) with a left ventricular enddiastolic diameter of 5.0 cm with global hypokinesis. Grade I mitral regurgitation and grade II tricuspid regurgitation were noted. Pulmonary artery systolic pressure was 50 mmHg. Laboratory parameters were: hemoglobin 11.1 g/dl, thrombocyte count 171,000/ mm³, and leukocytosis (21,500/mm³). Both the liver enzymes (SGOT 4307 U/I, SGPT 1196 U/l) and total bilirubin level (3.06 mg/dl) were elevated. Creatinine level was 1.8 mg/dl. Cardiac

enzymes were also elevated (CK-MB 10 ng/ml and troponin I 1.4 ng/ml)

The following day, the ECG showed sinus rhythm with right bundle branch block (Fig. 2). Liver enzymes, total bilirubin level and cardiac enzymes progressively improved. Ten days after her admission, her liver enzymes (SGOT 34 U/l, SGPT 83 U/l), creatinine (0.9 mg/dl), cardiac enzymes (CK-MB 5.1 ng/ml, troponin I 0.83 ng/ml), and leukocyte count (11,000/mm³) almost returned to normal. Right bundle branch block also resolved on her electrocardiogram and sinus rhythm was maintained with widespread T-wave inversion (Fig. 3). The patient remained asymptomatic under bisoprolol and enalapril treatment. Echocardiographic evaluation revealed marked restoration of LVEF (50%).

DISCUSSION

Peripartum cardiomyopathy is a rare form of cardiac failure occurring late in pregnancy or in the postpar-

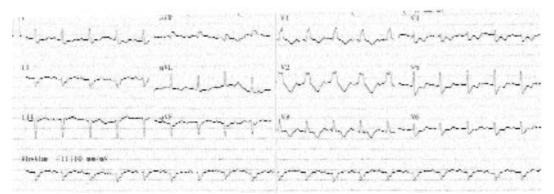


Figure 2. The following day, complete heart block reverted to sinus rhythm with right bundle branch block.

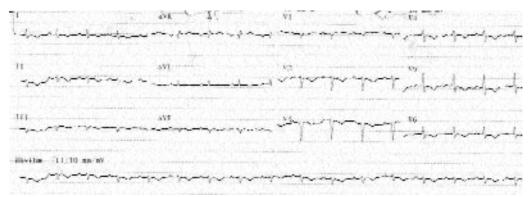


Figure 3. Ten days after her presentation, ECG showed sinus rhythm with T-wave inversion.

tum period. In April 1997, the National Heart, Lung, and Blood Institute (NHLBI) held a multidisciplinary review of PPCM in an effort to review current information and develop recommendations for further research and education. Definition of PPCM was based on the following four criteria: development of cardiac failure in the last month of pregnancy or within five months after delivery; absence of a demonstrable cause for cardiac failure; absence of demonstrable heart disease before the last month of pregnancy, and documented systolic dysfunction. [2] Echocardiography is the most widely used procedure for diagnosis and it usually shows a dilated left ventricle with marked systolic dysfunction. High parity, twin gestation, age beyond 30 years, conduction defects on electrocardiography, and late onset of symptoms after delivery are unfavorable prognostic factors.[3] Many studies reported poor prognosis in PPCM, with mortality rates ranging from 26% to 80%.[4] In contrast, Felker et al.[5] reported a lower incidence of death (7%) and the need for heart transplantation (7%) in the long-term (>8 years) follow-up. Return to normal size and function of the left ventricle usually occurs within six months after delivery in 50% of the cases. The likelihood for normalization of cardiac function is significantly higher in patients with an LVEF greater than 30%.[6]

In a young patient presenting with complete heart block, other etiologies including lyme disease, systemic lupus erythematosus, drug overdose (e.g. verapamil), hyperkalemia, history of mediastinal irradiation and use of herbs should be considered in the differential diagnosis.^[7-10] In our patient, careful history taking, laboratory analysis, and the absence of systemic manifestations of lupus or lyme disease suggested PPCM as the main cause of complete heart block.

To our knowledge, coexistence of complete heart block and PPCM has not been reported. The present patient fulfilled the criteria of NHLBI,^[2] and despite adverse prognostic factors on presentation (LVEF <30%, age>30 years) had an uneventful recovery with restoration of LVEF and resolution of complete heart block within a short time. Elevated cardiac enzymes on presentation improved progressively on the following days. This finding was probably due to the high prevalence of myocarditis (62%) in patients with PPMC.^[4] Elevation in liver enzymes was probably due to the hypotensive period before her presentation to the emergency room.

REFERENCES

- Pearson GD, Veille JC, Rahimtoola S, Hsia J, Oakley CM, Hosenpud JD, et al. Peripartum cardiomyopathy: National Heart, Lung, and Blood Institute and Office of Rare Diseases (National Institutes of Health) workshop recommendations and review. JAMA 2000;283: 1183-8.
- 2. Ro A, Frishman WH. Peripartum cardiomyopathy. Cardiol Rev 2006;14:35-42.
- 3. Ravikishore AG, Kaul UA, Sethi KK, Khalilullah M. Peripartum cardiomyopathy: prognostic variables at initial evaluation. Int J Cardiol 1991;32:377-80.
- 4. Homans DC. Peripartum cardiomyopathy. N Engl J Med 1985;312:1432-7.
- 5. Felker GM, Jaeger CJ, Klodas E, Thiemann DR, Hare JM, Hruban RH, et al. Myocarditis and long-term survival in peripartum cardiomyopathy. Am Heart J 2000; 140:785-91.
- 6. Elkayam U, Akhter MW, Singh H, Khan S, Bitar F, Hameed A, et al. Pregnancy-associated cardiomy-opathy: clinical characteristics and a comparison between early and late presentation. Circulation 2005; 111:2050-5.
- 7. Rosenfeld ME, Beckerman B, Ward MF, Sama A. Lyme carditis: complete AV dissociation with episodic asystole presenting as syncope in the emergency

180 Türk Kardiyol Dern Arş

- department. J Emerg Med 1999;17:661-4.
- 8. Maier WP, Ramirez HE, Miller SB. Complete heart block as the initial manifestation of systemic lupus erythematosus. Arch Intern Med 1987;147:170-1.
- 9. Kim NH, Oh SK, Jeong JW. Hyperkalaemia induced
- complete atrioventricular block with a narrow QRS complex. Heart 2005;91:e5.
- 10. Onrat E, Kaya D, Barutçu İ. Verapamil ile birlikte bal tüketimine bağlı geliştiği düşünülen atriyoventriküler blok: Olgu sunumu. Anadolu Kardiyol Derg 2003;3:353-4.