

CASE REPORT

An Unusual Case of Postpartum Anasarca

¹Jai Inder Singh, ²Randhir Puri, ³KG Kiran

¹Major, Graded Specialist, Medicine, Military Hospital, Belgaum, Karnataka, India

²Colonel, Department of Obstetrics and Gynecology, Military Hospital, Belgaum, Karnataka, India

³Colonel, Commanding Officer, Military Hospital, Belgaum, Karnataka, India

Correspondence: Major, Jai Inder Singh, Medical specialist, Military Hospital, Belgaum Camp, Karnataka-590009, India
Phone: +919343979290, +918312423852, e-mail: jai_11@mail.com

Abstract

A 21-year-old lady, primipara presented with breathlessness on exertion and generalized swelling of three weeks duration. Clinical examination revealed anasarca and features of cardiac failure. After evaluation, a diagnosis of peripartum cardiomyopathy was established based on echocardiographic findings of dilated cardiac chambers and poor left ventricular function. She responded well to treatment. The case is being reported for the diagnostic dilemma and rarity.

Keywords: Anasarca, peripartum cardiomyopathy, systolic dysfunction, echocardiography.

INTRODUCTION

Peripartum cardiomyopathy (PPCM) is a type of dilated cardiomyopathy in women with no past history of cardiac disease and requires a high index of suspicion for diagnosis. It is a disease of uncertain etiology and can worsen during future pregnancies. Symptomatic patients should receive therapy for cardiac failure. Contemporary literature regarding this disease in Asian countries is scarce and requires a large multicentric study into the incidence, etiology, natural history and treatment of the disease. We present a case of PPCM diagnosed and managed at a peripheral center.

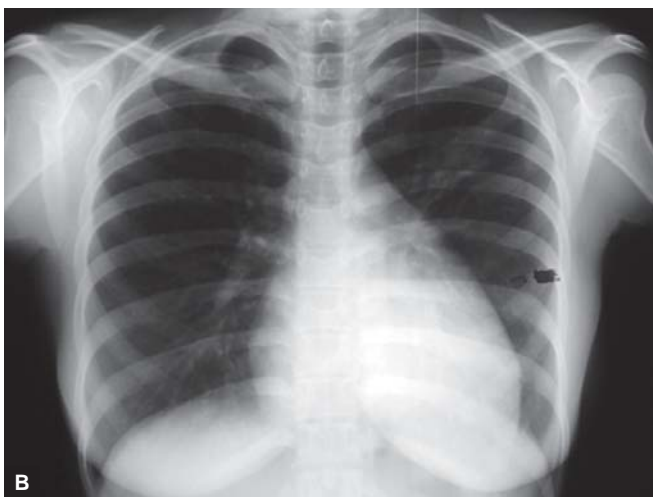
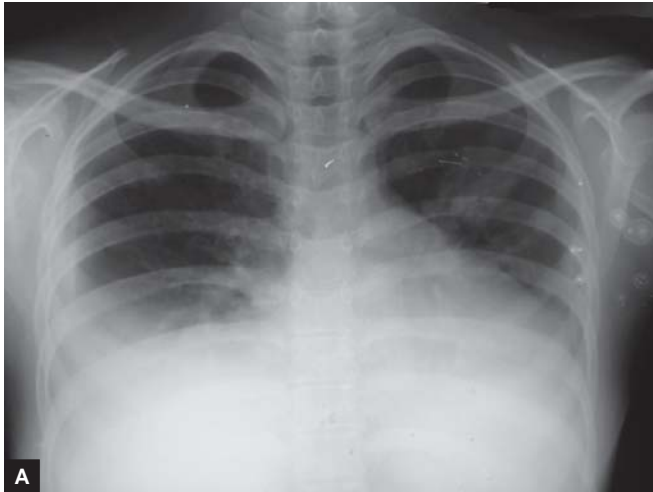
CASE REPORT

A 21-year-old lady presented with dry cough, breathlessness on exertion, generalized swelling of three weeks duration. There was no history of fever, rash, hemoptysis, chest pain, palpitations, orthopnea, oliguria, dysuria or hematuria, frothy urine, jaundice, headache or visual blurring and joint pains. She denied past history of any cardiac, renal, hepatic or connective tissue disorder. She had delivered her first child normally two months ago at term in the same hospital and had no puerperal complications. Physical examination revealed an ill looking young lady with facial puffiness, pallor and pedal edema. Tachycardia (HR = 104/min), tachypnea (RR = 24/min) were present and BP was 94/70 mm Hg. She was afebrile and other general physical examination was not contributory. There were no signs of chronic liver disease or immunocompromised state. Heart sounds were normal and grade II systolic murmur was heard at the left parasternal area. She had signs of bilateral

pleural effusion, ascites and mild hepatomegaly. Laboratory examination revealed microcytic hypochromic anemia (Hb = 8.2 gm/dl). Urine analysis showed presence of albumin 2+, 8-10 pus cells and 4-6 RBC's/hpf. Twenty four hour urine protein was 1.12 gm and urine culture was sterile. Renal/liver function tests, serum proteins, albumin and cholesterol were within normal limits. Chest X-ray showed cardiomegaly and bilateral pleural effusion (Figs 1A and B).

ECG showed T-wave inversion in inferior and lateral leads (Fig. 2). Cardiac enzymes were normal, antistreptolysin O titre, anti nuclear antibody (ANA) and viral markers (HIV, HBV and HCV) were negative. Ascitic fluid and pleural fluid analysis was suggestive of a transudate. USG abdomen showed congestive hepatomegaly, ascites and normomorphoc kidneys. Two days after admission she developed orthopnea and clinical features of congestive cardiac failure (raised jugular venous pulse and gallop rhythm). Echocardiography (Fig. 3) revealed severe left ventricular systolic dysfunction (Ejection Fraction = 20%), dilated left atrium (LA)/left ventricle (LV), marked generalized hypokinesia, mild mitral and tricuspid regurgitation, pulmonary pressure gradient of 50 mm Hg, a rim of pericardial effusion and no clot.

In view of foregoing she was diagnosed to have peripartum cardiomyopathy (PPCM) with systolic heart failure and managed on Inj. Furosemide, Tab spironolactone, digoxin, carvedilol and Inj Enoxaparin. She showed rapid clinical response to treatment. Repeat echocardiography after six months was recommended. She was counselled about risk during future pregnancies and discharged three weeks later on beta blockers, diuretics, digoxin and anticoagulants.



Figs 1A and B: A. Chest X-ray on admission showing cardiomegaly and bilateral pleural effusion, B. Chest X-ray after five days of treatment showing clearance of pleural effusion

DISCUSSION

PPCM is a dilated cardiomyopathy of uncertain etiology that is defined as¹ (1) development of cardiac failure in the last month of pregnancy or within 5 months after delivery, (2) absence of a demonstrable cause for the cardiac failure, (3) absence of demonstrable heart disease before the last month of pregnancy, and (4) documented systolic dysfunction by classic echocardiographic criteria, such as depressed shortening fraction or ejection fraction. PPCM remains a diagnosis of exclusion. The incidence of peripartum cardiomyopathy ranges from 1 in 1300 to 1 in 15,000 pregnancies in different parts of the world.¹ The incidence of PPCM in India has not been extensively studied but was found to be 1 case per 1374 live births in one study.² PPCM may some times be unrecognized, due to the non-specific nature of the symptoms leading to underestimation of the true incidence. The mean \pm SD age at presentation was 31 ± 5 years and mean parity was 2.6 ± 1 in one study from India.³ The study also reported that 37% patients were diagnosed antepartum and 63% postpartum compared to 17% and 83% respectively in a Chinese study.⁴ The etiology of PPCM is unknown, but viral, autoimmune,⁵ and idiopathic causes may contribute. Risk factors for PPCM are given in Table 1. Toxemia of pregnancy was not identified as a risk factor in one study from Haiti.⁶

The most common presentation of PPCM is with symptoms and signs of systolic heart failure⁷ and unusual presentations

Table 1: Risk factors for PPCM

- Advanced maternal age
- Multiparity
- African race
- Twinning
- Pre-eclampsia/eclampsia or postpartum hypertension
- Long-term tocolysis
- Selenium deficiency
- Maternal cocaine abuse

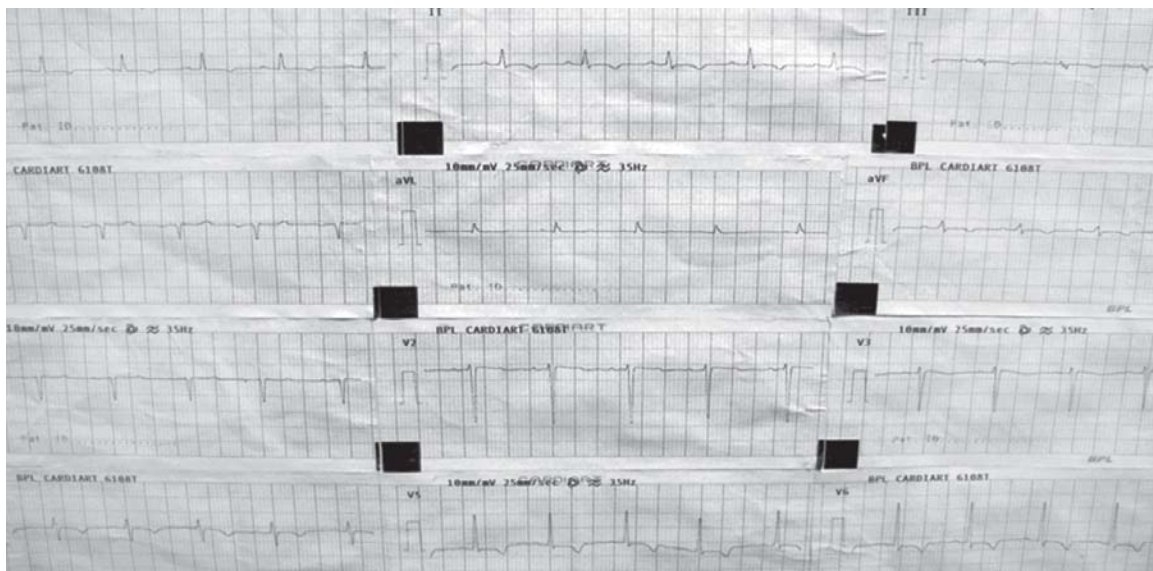


Fig. 2: 12 lead ECG showing T-wave inversion in leads I, II, III, aVF and V4 to V6

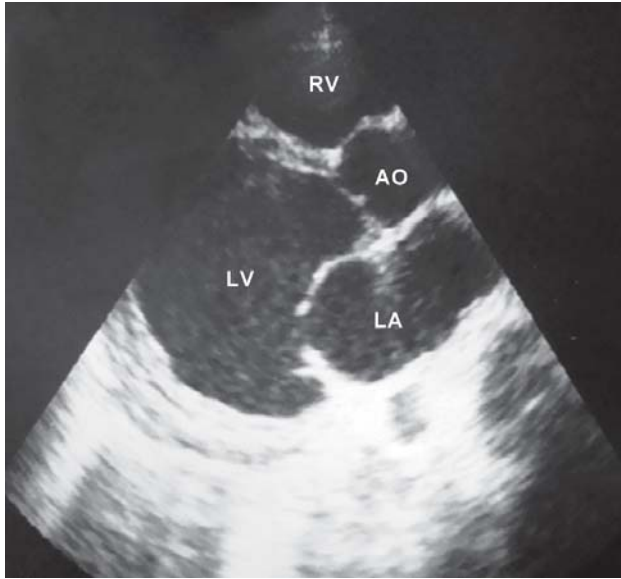


Fig. 3: 2D Echocardiography showing dilated LA/LV

relating to thromboembolism. Pulmonary edema was a presenting symptom in all 106 patients in a 2007 study in China.⁴ Clinical examination of 97 patients seen in South Africa⁸ over 4 years revealed a displaced hypodynamic apical impulse in 72% and gallop rhythm in 92% of the patients. Functional mitral regurgitation was present in 43%. Left ventricular hypertrophy by electrocardiographic voltage criteria was present in 66% and ST-T-wave abnormalities in 96% of the patients. Echocardiography is essential for diagnosis. Other differential diagnosis to consider are myocarditis, inherited cardiomyopathy, pre-eclampsia, systemic lupus erythematosus, anthracycline treatment. Treatment aims to reduce after load and preload, and to increase contractility. Angiotensin-converting enzyme (ACE) inhibitors are used if PPCM occurs after pregnancy. Hydralazine (with or without nitrates) should replace ACE inhibitors during pregnancy. Beta-blockers are recommended for peripartum cardiomyopathy, as they improve symptoms, ejection fraction, and survival. Nonselective beta-blockers such as carvedilol and selective ones such as metoprolol succinate have shown benefit. Diuretics are given for symptom relief. Spironolactone or digoxin is used in patients who have New York Heart Association class III or IV symptoms. The lowest daily dose of digoxin is given (serum digoxin level < 1.0 ng/mL). Inotropic support (Dobutamine infusion) may be required in severe case. Because of the high incidence of thromboembolism, the use of heparin is also considered necessary, followed by warfarin in those with left ventricular ejection fractions of less than 35%. Salt and water restriction are also important. The best time to discontinue ACE-inhibitors or β -blockers is unknown; however, one or both of these medications should be continued for at least one year.

Subsequent monitoring includes a follow-up echocardiogram in the first several weeks and at six month intervals until recovery is confirmed or a plateau is reached. In one study⁴ serum cardiac

troponin T (cTnT) concentration measured within 2 weeks of the onset of PPCM was correlated negatively with LVEF at follow-up. A cTnT concentration of > 0.04 ng/ml predicted persistent left ventricular dysfunction with a sensitivity of 54.9% and a specificity of 90.9%. About half the patients of PPCM recover without complications.^{6,9} Estimated maternal mortality ranges from 10 to 50%. Persistence of disease after six months indicates irreversible cardiomyopathy and portends worse survival. Recovery is more likely in those with an ejection fraction greater than 30% at diagnosis.⁹ Subsequent pregnancy should be discouraged, especially, if systolic function did not recover. Patients who recover normal left ventricular function and have normal left ventricular contractile reserve after dobutamine challenge may undertake another pregnancy safely, but they should be warned of the risk of recurrence even with fully recovered left ventricular function.^{10,11} Patients with severe cardiac dysfunction and decompensation should be evaluated for cardiac transplantation.

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