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Prolonged QTc interval: Peripartum cardiomyopathy

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Abstract

Peripartum cardiomyopathy (PPCM) is an idiopathic, often dilated, cardiomyopathy characterized by left ventricular systolic dysfunction. It occurs in 1:300 to 1:4000 live births, and risk factors include increased age, multiparity, black race, preeclampsia, hypertension, use of tocolytics, low socioeconomic status, twin pregnancy, and obesity. Women diagnosed with PPCM commonly present with signs and symptoms of heart failure with complications such as arrhythmia or thromboembolism. Sudden cardiac death (SCD) can occur due to life-threatening arrhythmias, especially when patients have poor left ventricular systolic function. Delayed diagnosis of PPCM can lead to complications associated with high mortality. We describe a case of PPCM in a 33-year-old female 40 days postpartum presenting with heart failure and typical ECG changes, which were detected early and treated accordingly. The patient was mechanically ventilated, treated for various arrhythmias, then extubated and discharged to the ward.

Keywords

QTc; peripartum cardiomyopathy

Introduction

Peripartum cardiomyopathy (PPCM) is characterized by left ventricular systolic dysfunction (LVSD), which usually occurs between the last month of pregnancy and the fifth month postpartum in patients without preexisting cardiac disease [1].

The etiology and pathogenesis of PPCM is unknown, but involvement of myocarditis, prolactin, 16kDa prolactin Cathepsin D, autoimmunity, or malnutrition has been proposed. Other hypotheses include abnormal response to increased hemodynamic burden of pregnancy, genetic susceptibility, and apoptosis [2]. Demakis et al. defined three diagnostic criteria for PPCM: 1) the development of heart failure during the last month of pregnancy or within five months after delivery, 2) the absence of determinable etiology for heart failure, and 3) absence of heart disease before the last month of pregnancy.

An electrocardiogram (ECG) is recommended for all PPCM patients, and 96% of PPCM patients have abnormal ECGs at presentation, thus making it a valuable tool for screening and estimating prognosis for patients at risk [3]. In this report, we describe a case of peripartum cardiomyopathy detected early by ECG and echocardiograph.

Case Presentation

A 33-year-old female 40 days post-cesarean section was admitted to the emergency department experiencing shortness of breath and chest pain for the preceding two days. The cesarean section had been uneventful, the patient was not known to have any previous illness, and there was no significant family history. Patient had been comfortable at home lactating and feeding the baby. The patient reported coughing with yellow expectoration and no fever. In the emergency department, the patient was tachypneic and placed on high-flow oxygen (15 L/min), after which oxygen saturation was 80%. The patient's pulse was 120/min, and blood pressure was 90/60 mmHg. A chest x-ray (Figure 1) showed bilateral pulmonary infiltrates with bilateral pleural effusion, suggesting pneumonia and pulmonary congestion. The ECG (Figure 2) showed ST-T changes with T-wave inversion in anterior chest leads, and the QTC interval was 514ms. The QT interval was 416ms. Thoracic CT (Figure 3), with contrast showed massive bilateral pleural effusion with bilateral basal collapse consolidation mainly on the left side with basal consolidation of the left upper lobe. Transthoracic echocardiography revealed no abnormality except for an ejection fraction of approximately 55%. Thyroid function was within normal limits. Patient showed no signs of improvement after treatment with high flow oxygen and was started on non-invasive ventilation for hypoxia. Despite treatment, the patient continued to deteriorate and was intubated and connected to mechanical ventilation. After diagnosis of pneumonia with pulmonary edema, patient was treated with antibiotic and furosemide. Antibiotic treatment did not affect the QT interval, and patient was shifted to the ICU. Bedside chest ultrasound showed bilateral Kerley B lines with bilateral effusion and a diagnosis of postpartum cardiomyopathy was made. Furosemide infusion was started. Subsequent ECG (Figure 4) showed ST-T changes with T-wave inversion in inferior and anterior leads. The QT interval increased to 530ms with QTC 546ms. In the ICU, the patient developed various arrhythmias including supraventricular and ventricular and received pharmacological and electrical cardioversion. Potassium and magnesium were 3.8mmol/L and 0.9mmol/L, respectively. Potassium chloride and magnesium sulfate were administered. Patient recovered and was continued on mechanical ventilation. Repeated echocardiography showed mildly impaired left ventricular systolic function with an ejection fraction of 45%, dilated left chambers, and moderate to severe mitral regurgitation. Cardiac enzymes were mildly elevated; troponin was initially 0.18ng/ml and increased to 0.29ng/ml. Metoprolol, furosemide, and ACE inhibitors were administered. Patient was started on methylprednisolone 60 mg 12 hourly. Approximately one liter of clear transudate fluid was removed during therapeutic right pleural drainage. Potassium chloride and magnesium sulfate were administered daily to maintain high-normal levels. After one week of mechanical ventilation, the patient was weaned and extubated successfully. The ECG next day (Figure 5) showed a QT interval of 362ms and a QTC 458ms. Patient was fully conscious on ambient air, hemodynamically stable, and shifted to the medical ward for two days before discharge. Follow-up was advised.

Discussion

We describe a case of PPCM in a 33-year-old female 40 days postpartum who presented with heart failure and demonstrated typical ECG changes and echocardiograph results, which were observed early to assist in providing appropriate treatment.

The diagnosis of PPCM requires development of heart failure, exclusion of other causes of cardiomyopathy, and confirmation by echocardiographic assessment of LVSD. An ECG is recommended for all PPCM patients: a recent study found 96% of PPCM patients have abnormal ECGs at presentation and suggested its potential usefulness in screening and assessing prognosis for patients at risk (3). Left ventricular recovery predominantly occurs within the first six months after diagnosis, but can extend beyond 12 months [4]. Women of African ethnicity present with a more severely reduced left ventricular ejection fraction (LVEF) and minimal recovery [5]. It is difficult to estimate, if ever, when a patient with PPCM may be considered fully "recovered" and her heart failure medications safely discontinued. Normalized left ventricular systolic function after treatment may not represent true recovery. There are, as such, no guidelines about cardiac medication duration. Some experts recommend slow weaning of cardiac medications with follow-up imaging to ensure that LVEF remains stable. Poor compliance to medications can lead to relapse of PPCM.

Echocardiography is not sensitive enough to detect recovery at the myocardial cellular level, rendering the patient vulnerable to future complications [6]. Cardiac magnetic resonance imaging has been used in a few PPCM patients to assess cardiac function and detect mural thrombi or myocardial fibrosis [6], and can also predict subsequent recovery of LVEF. The prognosis for PPCM patients varies geographically. In the United States, the mortality rates associated with PPCM have varied between 0–19%, while incidence of cardiac transplantation has ranged from 6–11% [7]. In PPCM, sudden cardiac death, which is usually due to ventricular tachyarrhythmia, contributes to 25–39% of all mortality. Though the literature on bradyarrhythmias in PPCM is minimal, it has been reported that up to 7% of patients require permanent pacing [8]. Holter ECG monitoring helps in diagnosing tachy- and bradyarrhythmias.

In PPCM, a 12-lead ECG is a valuable tool to detect the most common waveform abnormalities, including T-wave inversion and prolongation of the QTc interval. T-wave inversion at the time of diagnosis is associated with poor systolic function (LVEF <35%). QTc prolongation (i.e., corrected QT interval >460 ms) at presentation is an independent predictor of poor long-term outcomes in PPCM [9]. Extreme care must be taken when treating patients with PPCM to avoid drugs that can lead to QT prolongation. Bundle branch blocks are rare in PPCM, whereas their incidence in other forms of dilated cardiomyopathy is approximately 25-30% [9]. Our patient had prolonged QTc interval at admission with global hypokinesia and reduced left ventricular function. No drugs were administered, which would have affected the QT interval. Anti-failure drugs, beta-blockers, methylprednisolone, and frequent electrolyte replacement lead to rapid and dramatic recovery of the patient with normalization of QTc duration.

Treatment of PPCM in cases such as the one presented here should consist of a combination of beta-blockers, ACE-i/ARBs, and diuretics. The benefit of using digoxin in addition to these treatments is

controversial. The use of a prolactin-blocker such as bromocriptine can be considered and should be accompanied by anticoagulants to reduce the risk of thromboembolic events [10]. Sustained ventricular tachycardia (VT) or ventricular fibrillation should be electrically cardioverted or defibrillated without any delay. Intravenous amiodarone is used in cases of refractory VT [11].

The European Society of Cardiology recommends implantable cardioverter-defibrillator (ICD) as a primary prevention in PPCM with LVEF <35%, despite optimal medical therapy and as secondary prevention in PPCM with episodes of ventricular arrhythmias [12]. As there is a high chance of left ventricle recovery rate associated with PPCM, the decision to implant an ICD should be made with extreme caution.

Regarding subsequent pregnancies after PPCM, LVEF is the strongest predictor of adverse outcomes among patients in whom the LVEF did not normalize. Women with persistent left ventricular dysfunction (LVEF <50%) had a 50% risk of acute heart failure with worsening cardiomyopathy with a 25-50% risk of mortality in some sub-series of South Africa. After an episode of PPCM, women with normal LVEF had a 20% risk of deteriorating left ventricular systolic function in subsequent pregnancies, which lasted for an extended period in 20-50% of patients [13].

Figures

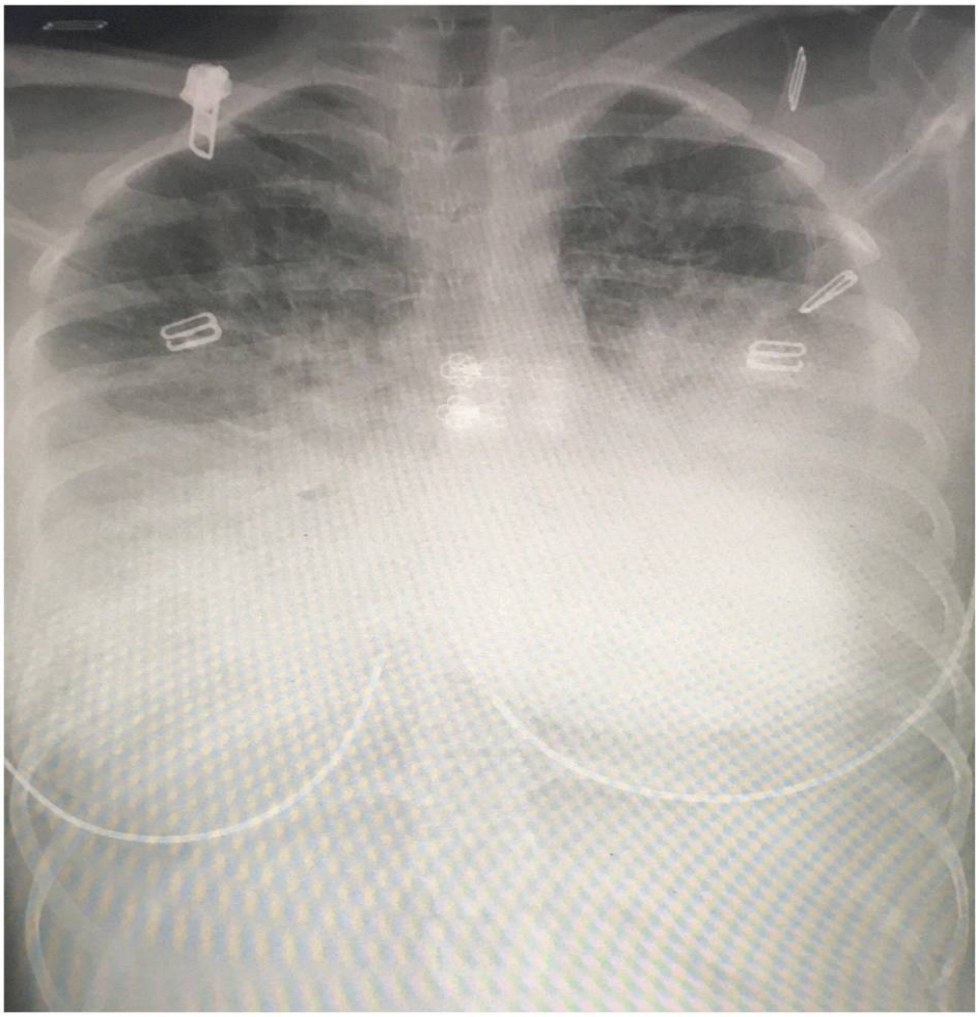

Figure 1: Chest x-ray showed bilateral pulmonary infiltrates with bilateral pleural effusion.

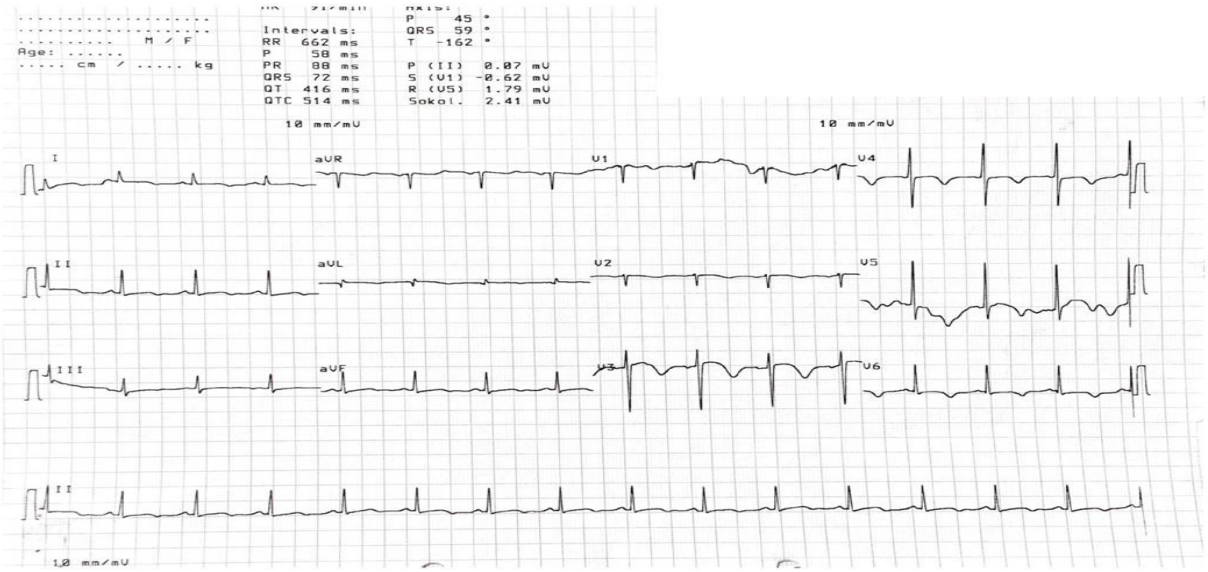

Figure 2: ECG in emergency department showed ST-T changes with T wave inversion in anterior chest leads and the QTC interval was 514ms.

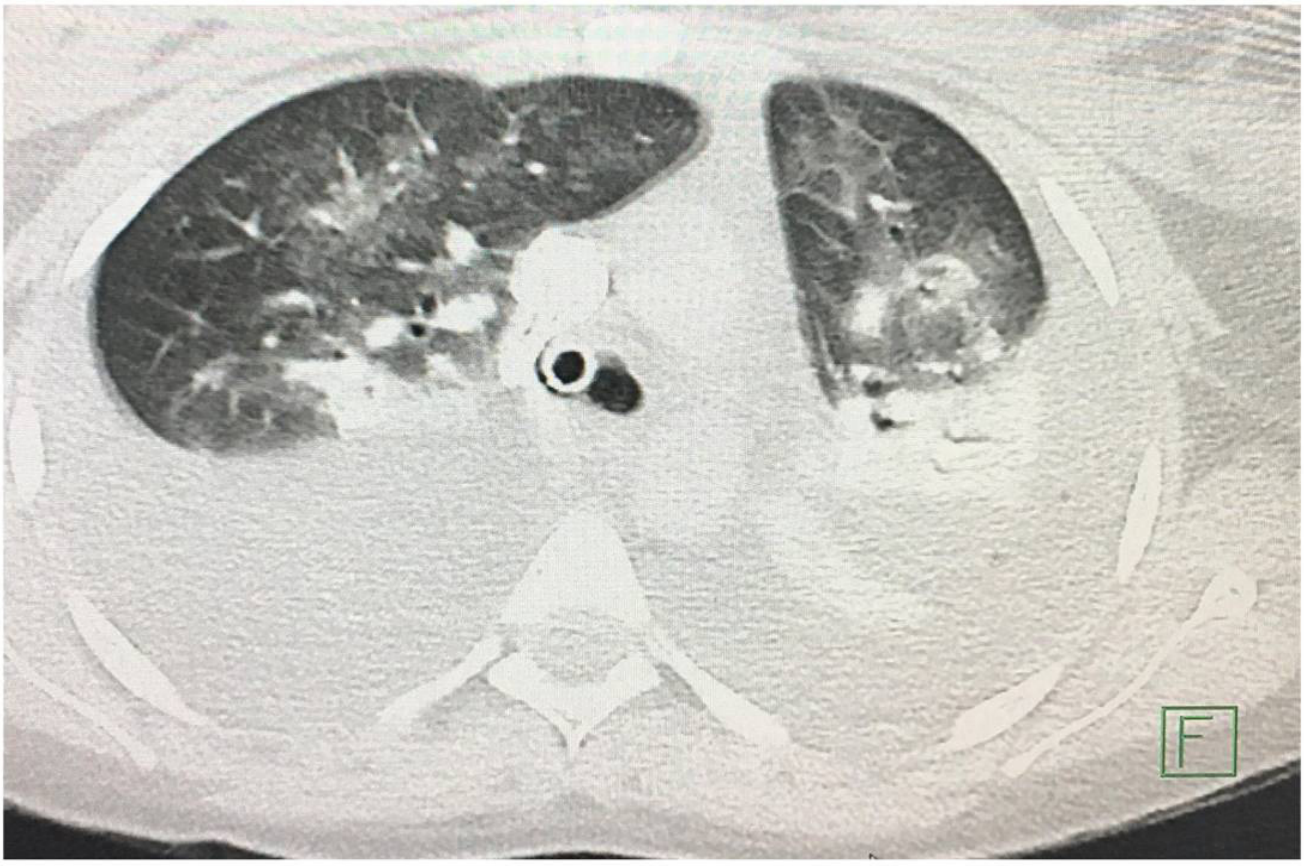

Figure 3: CT Thorax with contrast was carried out which showed massive bilateral pleural effusion with bilateral basal collapse consolidation mainly on left side with basal consolidation of left upper lobe.

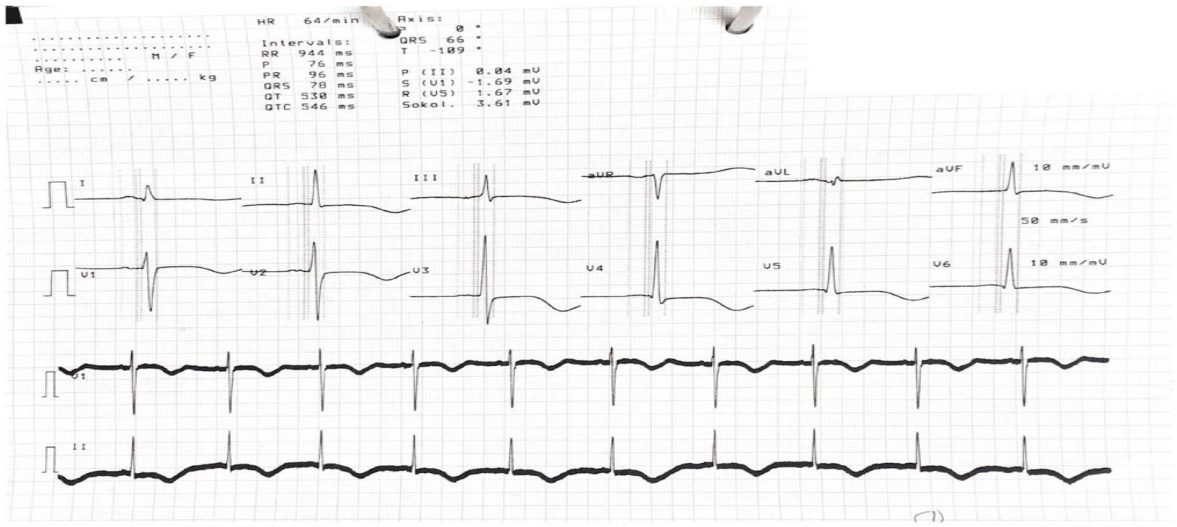

Figure 4: Subsequent ECG was carried out which showed ST-T changes with T wave inversion in inferior and anterior leads. The QT interval increased to 530ms with QTC 546ms.

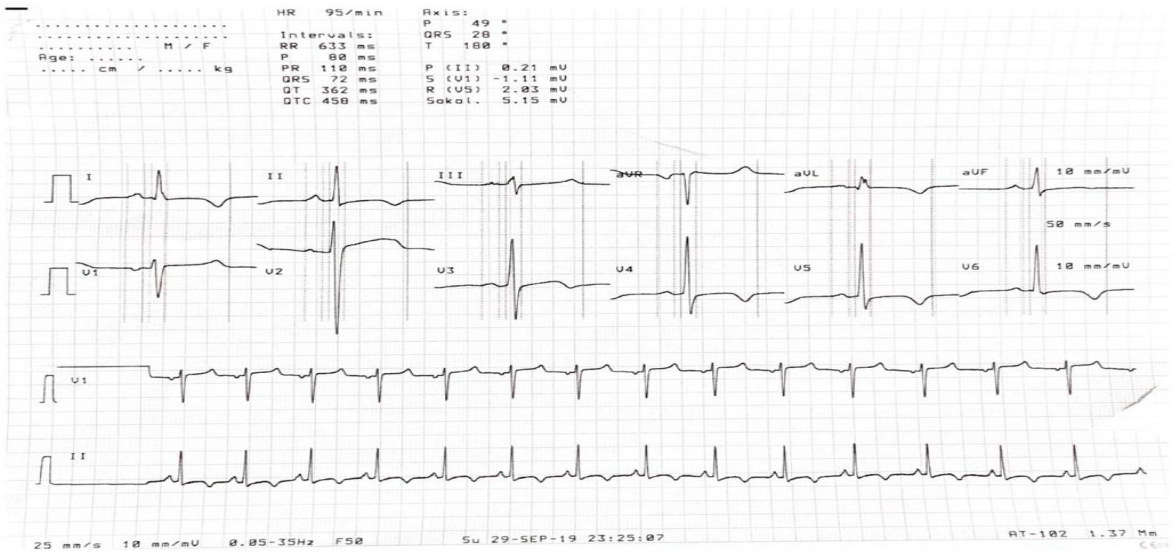

Figure 5: The subsequent ECG showed QT interval of 362ms and QTC 458ms

Conclusion

The course of PPCM can be unpredictable, and those who experience a good recovery may have subclinical dysfunction that places them at risk for future complications. PPCM can be catched earlier in the course of the disease by 12 lead ECG changes which commonly shows T-wave inversion and QT prolongation. Cardiac magnetic resonance imaging helps estimate complete myocardial recovery and, thus, risk of relapse in subsequent pregnancies. Counseling PPCM patients regarding subsequent pregnancies must involve a multidisciplinary approach, which should include an obstetrician, cardiologist, and pediatrician.

References

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2. Hilfiker-Kleiner D, Sliwa K. Pathophysiology and epidemiology of peripartum cardiomyopathy. Nat Rev Cardiol 2014; 11(6): 364-70.