CASE REPORT

Arrhythmic manifestation of peripartum cardiomyopathy— Case report

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We report a case of 26-year-old woman, with arrhythmic manifestation of peripartum cardiomyopathy with moderate heart failure. Ventricular arrhythmia recorded in ambulatory Holter ECG (premature ventricular contractions) was most severe during pregnancy, reduced when beta-blocker therapy was initiated and almost completely resolved after delivery. Then, 1 year after hospitalization in Cardiology Department, recurrence was observed with recorded short episode of nonsustained ventricular tachycardia.

KEYWORDS

heart failure, peripartum cardiomyopathy, pregnancy, ventricular arrhythmia

1 | CASE DESCRIPTION

A 26-year-old pregnant woman, without previous history of cardiac disease, was admitted to the Cardiology Department in gestation week 29 because of impaired exercise tolerance, palpitations, and occasional chest pain occurring over the preceding 4 weeks. Patient did not complain of syncope or dizziness. Blood pressure on admission was 120/75 mmHg, and physical examination was normal.

The electrocardiogram on admission revealed regular sinus rhythm, 72 bpm, rSr' complexes, and negative T waves in lead III; PR interval was 140 ms, QRS complex 100 ms, QT interval 380 ms (QTc according to Bazett's formula was 416 ms).

Transthoracic echocardiography revealed moderately dilated (58/68 mm) left ventricle (LV) with systolic dysfunction—ejection fraction (EF) 37%, decreased global longitudinal strain (GLS): −12% and concomitant systolic dysfunction of nondilated right ventricle (low TAPSE of 15 mm, see Figure 1). At 6-min walk test patient reached a distance of 500 m. In laboratory tests NT-proBNP was 197 pg/ml (borderline abnormal) and troponins were not elevated. Serum iron level: 40 µg/dl; and serum ferritin: 9 ng/ml were low. Results of complete blood count, inflammatory markers (C-reactive protein), electrolytes, serum creatinine, liver enzymes, and thyroid hormones were in normal ranges.

Ambulatory Holter ECG recording performed before hospitalization showed sinus rhythm with mean heart rate 90 bpm (range

50–141 bpm). During the recording 8,508 ventricular complexes occurred, including 21 couplets and accelerated idioventricular rhythm composed of 9 complexes with a rate 95 bpm. In Holter ECG monitoring repeated during a hospitalization at Cardiology Department 12,834 monomorphic ventricular complexes were recorded (LBBB morphology), including 345 episodes of ventricular bigeminy (Figure 2). The longest episode lasted 10 min.

Carefully taken heart family history revealed no trace of dilated cardiomyopathy, sudden cardiac death, or malignant arrhythmias in other family members including first- and second-degree relatives.

Considering clinical symptoms and results of additional tests, the diagnosis of peripartum cardiomyopathy (PPCM) was made. Beta-blocker therapy was initiated (metoprolol 100 mg daily) and iron supplementation was started.

One month after the hospitalization ambulatory Holter ECG was repeated. It revealed 7,683 ventricular complexes, including 643 episodes of ventricular bigeminy. The longest episode lasted 3 min. Transthoracic echocardiography showed stable, reduced left ventricular ejection fraction (LVEF 38%), and low GLS (−9.5%)—Figure 3. The patient reported relief of symptoms. After consultation in interdisciplinary team of cardiologists and obstetricians, delivery by cesarean section in week 40 was induced without any complications. After the labor, bromocriptine (2.5 mg twice daily) and angiotensin-converting enzyme inhibitor (ramipril 2.5 mg daily) were added to therapy.

FIGURE 1 Low TAPSE value (15 mm) in first echocardiography. TAPSE, tricuspid annular plane systolic excursion

FIGURE 2 Ventricular bigeminy in Holter ECG recording during hospitalization

The Holter ECG recording performed 2 months after delivery showed only 30 ventricular complexes, including two episodes of ventricular bigeminy. Transthoracic echocardiography showed sustained moderate dilatation (57/68 mm) of left ventricle with systolic dysfunction and ejection fraction (EF) 41% and low GLS (−11%)—Figure 4.

A control Holter performed 10 months after delivery recorded 2,654 ventricular complexes, including one episode of nonsustained ventricular tachycardia composed of three complexes with a rate 110 bpm. In TTE LV was slightly smaller (51/62 mm), with continuous

systolic dysfunction and EF 38% but normalization of right ventricular function occurred.

The Holter ECG monitoring that was made 16 months after delivery showed sinus rhythm with mean heart rate 62 bpm with sinus bradycardia in the nighttime (minimal heart rate 38 bpm), 1,387 ventricular complexes occurred, including eight couplets and 204 episodes of ventricular trigeminy. The patient presented with functional NYHA class I, had no syncope episodes and physical examination was normal. Her previous medical treatment of heart failure was maintained.

FIGURE 3 Remodeled LV and low GLS value 1 month after hospitalization. GLS, global longitudinal strain; LV, left ventricle

FIGURE 4 Remodeled LV 2 months after delivery. LV, left ventricle

2 | DISCUSSION

Peripartum cardiomyopathy (PPCM) is a nonfamilial form of peripartum heart failure. It is idiopathic cardiomyopathy with heart failure caused by left ventricular dysfunction, that occurs in the end of pregnancy or several months after delivery, when no other causes of heart failure can be found. Clinical features of PPCM are similar to DCM, although left ventricle is not always dilated, however, in almost all cases LVEF is <45% (Sliwa et al., 2010).

The incidence of PPCM varies depending on the geographical region. High frequency is described on Haiti (1:299) (Fett, Christie,

Carraway, & Murphy, 2005), in South Africa it is 1:1,000 (Desai, Moodley, & Naidoo, 1995). In Western countries the incidence of the disease is increasing. For example, in USA increase in incidence was described from 1993 (1:4,350) to 2002 (1:2,229). Potential causes of this phenomenon are pregnancies in older age, fertility-assisted treatment, multifetal pregnancies, better diagnostic tools, and better knowledge and awareness (Haghikia et al., 2013; Mielniczuk et al., 2006).

There are various clinical features of PPCM and they are similar to that in heart failure caused by other causes. Symptoms may resemble those encountered during physiological pregnancy, for

example, lower extremity edema, dyspnea on exertion, cough, paroxysmal nocturnal dyspnea, and orthopnea. Other symptoms that can occur are chest pain, palpitations, abdominal discomfort, and dizziness (Sliwa et al., 2010). In most patients (78%) symptoms occur within 4 months after delivery, in 9% during the last month of pregnancy and in 13% PPCM begins before or after above mentioned period (Lampert and Lang, 1995; Sliwa et al., 2010).

In pregnancy increased incidence of arrhythmias is observed, even in women without structural heart disease. Physiological changes in women's organism, such as increased circulating blood volume, altered vegetative system response and increased estrogen and progesterone levels which increase cardiomyocyte susceptibility to endogenous catecholamines may predispose to this condition. Potential triggers include electrolyte imbalance, antiarrhythmic drugs, stress, and physical activity. Premature ventricular contractions are common disorder in pregnant women without structural heart disease. Depending on publication, their frequency may range from 50% to even 90% during labor. Other types of ventricular arrhythmias, such as ventricular tachycardia, can also be observed in patients without structural heart disease. In their course cases of cardiac arrest were also described, but they are very rare (Kurpesa & Krzemińska-Pakuła, 2007; Świątecka, 2006).

In patients with PPCM arrhythmias are not uncommon, however, precise data are not available. Reported incidence varies depending on publication. Diao et al. (2004) performed Holter ECG recordings in 19 patients with diagnosed PPCM. Statistical analysis showed that in 89.4% of patients sinus tachycardia occurred, in 21% atrial premature complexes were recorded, in 36.8% premature ventricular complexes, in four patients episodes of nsVT and in one case first-degree atrioventricular block.

Incidence of arrhythmias in patients with initial diagnosis of PPCM was also evaluated in study carried out by Mallikethi-Reddy et al. (2017). They analyzed data from 9,841 cases of hospitalization in period from 2007 to 2012 in numerous centers in USA. Mean age of patients was 30.05 ± 6.69 years. Arrhythmias were present in 18.7% of patients. The most common disorder was ventricular tachycardia (4.2%), in 1.7% of patients bundle branch block was reported, atrial fibrillation was present in 1.3% of cohort, in 1% ventricular fibrillation occurred and in 0.1% atrial or ventricular premature complexes were observed. Cardiac arrest occurred in 2.2% of patients. 0.3% of analyzed group required electrical cardioversion, 1.9% catheter ablation, and in 6.8% implantation of ICD was performed.

Ventricular arrhythmias are a significant problem among PPCM patients, especially since they may be potentially life threatening. Sudden cardiac death is often described as a cause of death, which may indicate a significant contribution to arrhythmias. In retrospective study carried out by Goland et al. sudden cardiac death was the cause of 38% of deaths (Goland et al., 2009). In other study authors analyzed 19 cases of patients with PPCM, which had implanted ICD in primary prevention and 60 control subjects with dilated cardiomyopathy (DCM). During 3-year follow-up in 37% of patients with PPCM appropriate ICD therapy was reported. In this group

ventricular arrhythmias were significantly more frequent than in patients with DCM (Arany and Elkayam, 2016).

Due to the real risk of serious arrhythmias from a potentially reversible cause, a wearable cardioverter/defibrillator (WCD) may be an attractive solution. Duncker et al. (2014) investigated usefulness of WCD as a temporary protection against life-threatening ventricular arrhythmias. The study group included seven patients with PPCM and reduced left ventricular ejection fraction ($LVEF \leq 35\%$). A median follow-up was 81 days. In three of patients four episodes of ventricular fibrillation occurred with appropriate and successful WCD therapy.

In the other study of the same authors (Duncker et al., 2017) incidence of arrhythmias was evaluated in 49 patients with PPCM and $LVEF \leq 35\%$ which received a WCD. Mean follow-up was 15 ± 10 months. In six patients (12%) eight episodes of ventricular arrhythmia were recorded: five episodes of ventricular fibrillation, two episodes of sustained ventricular tachycardia, and one episode of nonsustained ventricular tachycardia. Authors indicate that patients with PPCM and reduced LVEF are at higher risk of serious ventricular arrhythmias. They also mention that in this group use of WCD should be considered for 3–6 months to prevent sudden cardiac death caused by ventricular tachyarrhythmias.

Prognosis in PPCM depends on many conditions. Factors associated with poor prognosis are older maternal age, multiple pregnancies, severely reduced LVEF ($<25\%$), late diagnosis, and occurrence of arrhythmias (Duncker et al., 2017; Świątecka, 2006). Mortality is reported from 7% to 50% depending on publication. 50% of deaths occur in the first months after diagnosis (Whitehead, Berg, & Chang, 2003). In study carried out by Krishnamoorthy et al. (2016) 4,871 cases of patients with PPCM were analyzed. There was a higher mortality rate after delivery (2.1%) compared to perinatal (0.5%).

In subsequent pregnancies PPCM recurrence is observed in 50% of patients with left ventricular dysfunction and in 20% of patients with complete recovery after delivery. In those cases mortality may reach even 100%. Therefore, patients with PPCM should be informed about the risk associated with the next pregnancy. Sustained left ventricular dysfunction is considered as a contraindication for another pregnancy. In case of subsequent pregnancy optimal pharmacotherapy should be implemented and therapeutic decisions should be made in interdisciplinary team of cardiologists, obstetricians, anesthetists, and neonatologists (Hilfiker-Kleiner et al., 2015; Świątecka, 2006). Since left ventricular systolic function often tends to deteriorate in the third trimester, regular echocardiographic monitoring during this period (e.g., every 2 weeks) seems to be helpful (Kim and Shin, 2017).

The main limitation of our case is lack of MRI performed and genetic assessment toward dilated cardiomyopathy due to absence of patient's consent.

In conclusion, this case illustrates less typical manifestation of PPCM with dominant arrhythmic symptoms, moderate heart failure, and good response to beta-blockers therapy. Ventricular arrhythmia (premature ventricular contractions) was most severe during pregnancy, reduced when beta-blocker therapy was

initiated and almost completely resolved after delivery. Then, 1 year after hospitalization in Cardiology Department, recurrence was observed with recorded short episode of nonsustained ventricular tachycardia.

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