Left bundle branch block and peripartum cardiomyopathy – review of recent evidence

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Abstract

Peripartum cardiomyopathy (PPCM) is a rare, life-threatening heart disease of still unknown cause manifesting in late pregnancy or within 5 months after delivery in women without prior known cardiac pathology. The outcome of PPCM is highly variable, including clinical recovery, rapid deterioration unresponsive to medical treatment requiring mechanical circulatory support, and even death. Early diagnosis allows for standard heart failure treatment respecting contraindications for some drugs in pregnancy. Confirmation requires echocardiography to reveal left ventricular dysfunction. However, variables from electrocardiography (ECG) such as QRS duration are also important in the initial assessment. This paper presents the current state of knowledge of the diagnostic and prognostic role of the occurrence of left bundle branch block (LBBB) in PPCM.

Introduction

Although peripartum cardiomyopathy (PPCM) was first described in 1880, much remains unknown about it[1]. Recently it has been reviewed in ESC Guidelines[2]. Undoubtedly the ongoing prospective, international and multicenter registry which aims to collect information on 1000 patients with PPCM, as part of the EU Obser-vational Research Programme (EORP) and as an initiative of the Study Group on PPCM of the Heart Failure Association, will provide important data on this condition. The analysis of the first 411 patients were presented by Sliwa in 2017[3].

Peripartum cardiomyopathy (PPCM) is characterized by left ventricular ejection fraction reduced to less than 45% near the end of pregnancy or within the first 5 months postpartum[4]. Important predisposing factors include African ethnicity, advanced or teenage age, multiparity, pre-eclampsia, smoking, diabetes and malnutrition[5]. Although the cause of PPCM is still not fully explained, potential etiologies include a complex interaction of genetic and environmental factors contributing to inflammation, endothelial damage and angiogenic imbalance, which may lead to myocardial dysfunction in susceptible women[6][7]. Nowadays studies indicate that the cleavage of prolactin into an angiostatic N-terminal 16 kDa prolactin fragment (16 kDa Prl) and impaired VEGF signaling because of up-regulated sFlt-1 may initiate PPCM[1].
Peripartum cardiomyopathy has received increasing awareness and attention in the past decade. Recently published studies have focused on the pathomechanism, presentation and management of PPCM. Despite the increasing number of surveys on this issue, there is a lack of research on diagnostic and prognostic role of electrocardiographic variables such as echocardiographic and electrophysiologic features were quite similar between studied regions in the registry on PPCM from the EURObservational Research Programme and the Heart Failure Association of the European Society of Cardiology.

However, QRS duration was longer in ESC countries than in non-ESC countries – mean (SD) 93.8 ms (21.7) vs. 86.8 (20.8) and median (Q1–Q3) 90.0 (80.0–100.0) vs. 80.0 (80.0–90.0), p<0.001. LBBB appeared more often – 23/194 (11.9%) in ESC countries vs. 14/204 (6.9%) in non-ESC countries, but without statistical significance, p=0.086.

Furthermore, interesting data were obtained from the comparison of electrocardiographic characteristics in PPCM and Hypertensive Heart Failure of Pregnancy (HHFP) patients. First, LVH was found to occur more commonly in HHFP than in PPCM. In turn, atrial fibrillation (p = 0.028), QRS abnormalities (p = 0.001), left atrial hypertrophy (p = 0.030), T wave inversion (p < 0.001), longer QRS duration (p < 0.001), and left bundle branch block (p = 0.002) were detected more often in PPCM than in HHFP patients. Occurrence of LBBB may signify greater myocardial injury in patients with PPCM compared to HHFP. However, no ECG characteristics can differentiate between PPCM and HHFP.

Eventually, approximately 23-41% of women recover completely from PPCM. Labidi’s interesting case report reveals that LBBB in pregnancy may be the first sign of developing PPCM but can also be temporary. After the 12-month postpartum intensive heart failure treatment (bromocriptine, ACE inhibitors, diuretics, beta-blockers) the patient showed rapid improvement and normalized cardiac dimension and function and also LBBB had disappeared.

According to the guidelines for severe LV dysfunction during the 6–12 months following the first presentation despite optimal medical therapy for patients with left bundle branch block and QRS >130 ms, implantation of an implantable cardioverter defibrillator and cardiac resynchronization (CRT) therapy are recommended. However, mortality reduction in those women with non-ischemic cardiomyopathy is uncertain.

In the PPCM registry only one woman from the group of ESC countries was qualified for implantation of CRT – 1/410 (0.2%) [3].

Conclusions

Peripartum cardiomyopathy has received increasing awareness and attention in the past decade. Recently published studies have focused on the pathomechanism, presentation and management of PPCM. Despite the increasing number of surveys on this issue, there is a lack of research on diagnostic and prognostic role of electrocardiographic variables such as occurrence of LBBB.

The EURObservational Registry on PPCM and a few ongoing studies will hopefully provide substantial new in-
formation. During that time undoubtedly occurrence of LBBB in a pregnant woman should be taken seriously, as it may be the first symptom of imminent peripartum cardiomyopathy.

References

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