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PREGNANCY WITH PREPARTAL CARDIOMYOPATHY

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ABSTRACT

Peripartal cardiomyopathy (PCMP) is an idiopathic cardiomyopathy with left ventricular systolic dysfunction and clinical heart failure that occurs in healthy women at the end of pregnancy or in the first months after delivery. According to the 2016 definition of the European Society of Cardiology (ESC) working group on PCMP, the diagnosis of PCMP can be made by checking the left ventricular ejection fraction (LVEF) by 45%. Other causes of heart failure (pulmonary embolism, congenital or acquired heart disease, dilated cardiomyopathy, etc.) should be ruled out during the diagnosis process.

Keywords: Cardiomyopathy, prepartal, pregnancy, laparoscopic, therapeutic, gynecological, uterine, clinical.

The prevalence of PCMP varies significantly depending on regional and ethnic characteristics. There are no exact statistics, but it is known that PCMP is more common in Nigeria and Haiti - 1 case per 100 births. The prevalence of PCMP in Germany is approximately 1: 1000-1500 births and is comparable to data from South Africa (1: 1000) and the United States (1: 1500). The mortality rate in this pathology is 2% to 30%. To date, several risk factors for PCMP are known: extreme maternal age, multiple pregnancies, in vitro fertilization, African race, long-term tocolytic therapy, hereditary factors, malnutrition during pregnancy.

Arterial hypertension (AH) during pregnancy is also referred to as a risk factor for PKMP. Pregnancy hypertension is 1.5 times more common and Preeclampsia is 4 times more common in patients with PCMP. Animal studies have shown that angiogenic imbalances play an important role in the development of PCMP and preeclampsia. During the last trimester of pregnancy, the placenta releases a large number of anti-angiogenic factors that cause endothelial dysfunction, vasospasm, hypertension, and organ hypoperfusion. However, not all patients with preeclampsia develop PCMP, which is probably a genetic predisposition. In one study, the onset of PCMP was associated

with a history of gestational hypertension, but this did not affect the prognosis and recovery of RF LV systolic function. One of the largest epidemiological studies was conducted in the United States between 2004 and 2011. During this period, 34,219 women aged 15 to 54 years were registered with a PCM diagnosis. The prevalence of this diagnosis was 1 case per 968 live births. According to the registry, the incidence of PKMP was found to increase with age and was highest among women aged 40–54 years. The most common PCMP is found in African-American women, women with hypertension, eclampsia (or preeclampsia), and type 2 diabetes.

To date, the pathogenesis of PCMP is not fully understood; Several mechanisms of PCMP development have been proposed, such as autoimmune, inflammatory theory, viral infections, and low levels of selenium in the blood. However, none of these mechanisms have been confirmed. That theory was first described in an experimental study conducted in 2007 by D. Hilfiker-Kleiner et al. Showed an important role of prolactin during lactation in the mouse model, noting the occurrence of toxic effects on cardiomyocytes and the formation of heart muscle dysfunction as a result. Thus, due to oxidative stress associated with pregnancy and the postpartum period, a 23 kDa prolactin molecule is broken down to form a 16 kDa prolactin fragment, which has an anti-angiogenic, anti-inflammatory effect. This fragment of prolactin induces apoptosis of cardiomyocytes with properties similar to proteolytic enzymes such as cathepsin D and matrix metalloproteinases (MMP). In addition, damage to the prolactin molecule may occur with the formation of a 16KDa fragment. Thus, a 16 kDa fraction of prolactin causes endothelial dysfunction and micro-RNA 146a expression, which has toxic effects on myocytes and potentially leads to the development of reversible systolic heart failure.

Furthermore, an increase in 16kDa in patients with PCMP has been shown to be associated with slow transcription and activation of receptor coactivator gamma (PGC-1a) activated by peroxisome proliferator, which plays an important role in proliferation. and protecting myocytes from death. PGC-a1 receptors are involved in lowering the levels of the antioxidant manganese-dependent superoxide dismutase (MnSOD), which leads to increased production of reactive oxygen species that have a detrimental effect on cells, which has been proven in experimental studies on rodents.

An increase in pro-angiogenic placental growth factor (PLGF) levels was detected in patients with PKMP in the early postpartum period. Efficacy against PLGF-factor has been shown as a method of prevention of myocardial dysfunction. These results highlight the need for further study of the function of the endothelial and proangiogenic

system in PCMP and the search for new effective methods of its specific prevention and treatment. It is known that approximately 15% of patients with PCMP have mutations in genes similar to those in expanded cardiomyopathy, which confirms the role of genetics in the development of PCMP. Several gene mutations, such as cardiac myosin heavy chain (MYH), titin (TSN), and SCN5, have been previously described. In most carriers, the mutations are asymptomatic before pregnancy. Hemodynamic changes (increased heart rate, cardiac output, decreased general peripheral vascular resistance, volume overload of the heart) occur in the late stages of pregnancy, during and in the early postpartum period. This hemodynamic stress may be a trigger for genetic damage activation. In addition, more morbidity in certain ethnic groups and in some parts of the world confirms the role of genetic factors.

The course of the disease can range from mild changes in the patient's condition to very severe forms with cardiogenic shock clinic. Often, before the end of pregnancy and in the early postpartum period, even healthy women begin to complain of general discomfort, weakness, asthenia, shortness of breath, edema syndrome associated with general fluid retention. Complaints of pathological dyspnea should be distinguished from orthopnea and hypotension. In some cases, PCMP begins with cardiogenic shock and lung tumor. Early diagnosis is critical in all cases of PCMP, allowing timely initiation of proper treatment. For the onset of chronic heart failure (CHF), the PKMP clinic should be able to differentiate between treating the patient in the last trimester of pregnancy and in the early postpartum period. Patients complain of severe unexplained weakness, dizziness, heaviness and swelling, swelling of the lower extremities. One study found that only 4% of the 262 patients who complained of shortness of breath during pregnancy were associated with the onset of PKMP, and in 96% of cases, shortness of breath was associated with physiological changes in the respiratory system. In this case, the system of the pregnant woman, respiratory infections, severe anemia can also be observed.

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