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Heart disease in pregnancy

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Abstract

Heart disease are common condition in pregnant women. Even, some of the cases diagnose just during the pregnancy. In this article, we summarize common cardiac disorders, which might encounter during the pregnancy and after birth. Furthermore, we discuss some etiological factors, pathophysiology, clinical manifestation, treatment and outcomes of the cardiac disease in pregnancy.

Keywords: Pregnancy; Cardiac disease; Heart failure; Birth

1. Introduction

Heart disease accounts for about 10% of maternal mortality [1]. In the United States, as the incidence of rheumatic heart disease has dropped significantly, most heart problems during pregnancy are the result of congenital diseases [2]. However, rheumatic heart disease is still common in Southeast Asia, Africa, India, the Middle East, and parts of Australia and New Zealand [3].

Among other diseases, it ranks first in maternal and perinatal morbidity and mortality. Increased requirements for the functioning of the cardiovascular system are required even during normal, physiological pregnancy and childbirth, which is due to: development and growth of the fetus; the appearance of the placental circulation; an increase in body weight of a pregnant woman and other changes occurring in a woman's body [4]. All diseases of the cardiovascular system can be divided into groups: congenital heart defects; diseases of the myocardium, pericardium, endocardium; rheumatic heart disease and rheumatism; the state of "operated heart"; hypotonic illness; hypertonic disease; systemic lupus erythematosus.

During pregnancy, the hormonal status changes. The anterior lobe of the pituitary gland (adenohypophysis) increases 2-3 times and undergoes significant morphological restructuring associated with an increase in the number and size of cells that secrete some hormones [5]. In addition to the hypothalamic-pituitary regulation, which ensures the adaptation of a woman during pregnancy, an important role in this process is played by the corpus luteum of pregnancy, which secretes estrogens and progesterone. Exposure to estrogen correlates with progesterone levels [6]. Throughout pregnancy, the content of progesterone is maintained at a high level, a decrease in its concentration occurs towards the end of pregnancy, when the level of estrogen sharply increases [7]. From the very beginning of pregnancy and before childbirth, β-adrenoreactivity increases and α-adrenergic reactivity decreases, which is a necessary condition for reducing the contractile activity of the myometrium in order to carry a fetus [8]. The density of β-adrenergic receptors under the action of progesterone in the myometrium increases. By itself, the activation of β-adrenergic receptors can stimulate a number of effects: inhibition of erythropoiesis and immunity, as well as an increase in renin secretion by the kidneys and thereby an increase in cardiac output [9]. This contributes to an increase in the incidence of anemia, immunodeficiency states, vegetative-vascular dystonia and hypertension during pregnancy.

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Despite dramatic improvements in the survival and quality of life of patients with severe congenital heart defects and other heart diseases, pregnancy is still not recommended for women with a certain high risk of diseases, such as the following.

- Pulmonary hypertension (pulmonary artery systolic pressure> 25 mmHg) caused by any condition, including Eisenmenger syndrome
- Coarctation of the aorta, if it is not eliminated or is accompanied by an aneurysm
- Marfan syndrome with aortic root diameter> 4.5 cm
- Severe symptomatic aortic stenosis or severe mitral stenosis
- Bicuspid aortic valve with ascending aortic diameter> 50 mm
- Single ventricle and systolic dysfunction (regardless of whether Fontaine surgery was used)
- Cardiomyopathy with an ejection fraction <30% or NYHA class III or IV heart failure (see table New York Heart Association (NYHA) Classification of Heart Failure]).

2. Pathophysiology

Pregnancy makes the cardiovascular system tense, often worsening underlying heart disease; minor heart disease may first appear during pregnancy.

Stress includes a decrease in hemoglobin levels and an increase in circulating blood volume, stroke volume, and heart rate [10]. Cardiac output is increased by 30-50% [11]. These changes peak between 28 and 34 weeks of gestation.

During labor, with each contraction, cardiac output increases by about 20% [12]. Other stressors include pushing during the second stage of labor and increased venous return to the heart from the contracting uterus. Cardiovascular disorders return to their pre-pregnancy state only a few weeks after delivery.

3. Clinical manifestations

Signs suggestive of heart failure (mild dyspnea, systolic murmurs, dilated jugular veins, tachycardia, edema, slight enlargement of the heart on a chest x-ray) are usually seen in normal pregnancy or may be a manifestation of heart disease. Diastolic or presystolic heart murmurs are more common in heart disease.

Heart failure can be a cause of premature birth or arrhythmias [13]. The risk of maternal or fetal death is correlated with a functional classification by the New York Heart Association (NYHA) based on the intensity of physical activity that causes symptoms of heart failure.

The risk is increased if only the symptoms

- Appears with light exercise (NYHA class III)
- Appears with minimal physical activity or at rest (NYHA class IV)

4. Diagnostics

- Clinical evaluation
- Typically, echocardiography

Diagnosis of cardiac dysfunction during pregnancy is usually based on clinical examination and echocardiography.

Heredity can influence the risk of developing heart disease, therefore, women with congenital heart disease should be offered genetic counseling and fetal echocardiography.

5. Treatment

- Elimination of warfarin, Angiotensin Converting Enzyme (ACE) inhibitors, angiotensin II receptor blockers (ARBs), aldosterone antagonists, thiazide diuretics and some antiarrhythmic drugs (amiodarone)
- In class III and IV heart failure (NISA) after 20 weeks, exercise restriction and even bed rest are indicated
- Common treatment options for heart failure and arrhythmias

Frequent prenatal consultations, good rest, avoidance of excessive weight gain and stress, treatment of anemia [14]. An anesthesiologist familiar with cardiac disease in pregnant women should be present during labor and should ideally advise the patient prior to delivery [15]. Pain and anxiety are treated aggressively to minimize tachycardia during labor. The woman is closely monitored immediately after childbirth and is observed several weeks after the birth by a cardiologist.

Before women with Class III and IV deficiency (NYHA) become pregnant, their medical conditions must be treated with medication or, if indicated (for example, with heart valve disease), surgically [16]. Women with class III or IV heart failure or other high-risk disorders (listed above) may be offered early termination [17].

Some women with heart disease and impaired heart function need digoxin 0.25 mg orally per day, and from 20 weeks on, bed rest or activity restriction [18]. Cardiac glycosides (digoxin, digitoxin) cross the placental barrier, but newborns (and children) are relatively resistant to their toxicity [19]. ACE inhibitors [20] and ARBs [21] are contraindicated because can cause kidney damage in the fetus. Aldosterone antagonists (spironolactone, eplerenone) are excluded because they can cause feminization of the male fetus [22]. Other drugs for the treatment of heart failure (non-thiazide diuretics, nitrates, inotropins) can be continued during pregnancy [23], weighing the severity of the disease and the risk to the fetus, which is determined by the cardiologist and perinatologist.

6. Arrhythmias

Atrial fibrillation can be a manifestation of cardiomyopathy or valvular disease [24]. Heart rate regulation is usually similar to that in non-pregnant patients and is done with beta-blockers, calcium antagonists, or digoxin [25]. Certain antiarrhythmics (amiodarone) should be avoided [26]. If a pregnant patient has newly developed atrial fibrillation [27] or hemodynamic instability, or if medical control of ventricular rate is ineffective, cardioversion may be used to restore sinus rhythm.

Anticoagulants may be needed because the relative hypercoagulability of pregnancy makes atrial blood clots (and then systemic and pulmonary embolism) more likely [28]. Standard or low molecular weight heparin is used. Neither standard heparin nor low molecular weight heparins cross the placental barrier, but low molecular weight drugs have a lower risk of thrombocytopenia. Warfarin crosses the placenta and can cause fetal malformations [29], especially when taken in the 1st trimester. However, the risk is dose-dependent and very low incidence rates are observed at doses ≤ 5 mg per day [30]. Using warfarin in the last month of pregnancy is risky. Rapid cessation of the anticoagulant effect of warfarin may be difficult to achieve and may be necessary due to traumatic intracranial hemorrhage in the fetus or newborn, or maternal bleeding (due to trauma or emergency caesarean section).

Treatment for acute supraventricular tachycardia or ventricular tachycardia is the same as for non-pregnant patients.

7. Arterial hypertension

Arterial hypertension (AH) is a condition in which a systolic blood pressure (SBP) ≥140 mm Hg is recorded in pregnant women and/or diastolic blood pressure (DBP) ≥90 mm Hg Level increase needs to be confirmed blood pressure (BP), at least two measurements with an interval at least 15 minutes on the same hand [31]. About the presence of hypertension with self-measurement of blood pressure of a pregnant woman at home or during daily blood pressure monitoring (ABPM) indicates a blood pressure level of ≥135/85 mm Hg.

8. Prevention of endocarditis

For pregnant women with structural heart disease, the indications for endocarditis prophylaxis are the same as for non-pregnant women [32]. The American Heart Association's Guidelines of the Year discourage endocarditis prophylaxis in both vaginal and caesarean delivery because of the low incidence of bacteremia. However, in patients at highest risk (eg, patients with prosthetic materials, a history of endocarditis, unrepaired congenital cyanotic heart disease, or a heart transplant with valvulopathy), prophylaxis is often considered for amniotic fluid drainage, although evidence of benefit is lacking [33].

If patients with structural heart disease develop chorioamnionitis or another infection (e.g., pyelonephritis) that requires hospitalization, the spectrum of antibiotics used to treat the infection should cover the pathogens commonly associated with endocarditis.

9. Stenosis

9.1. Valvular stenosis and insufficiency in pregnancy

During pregnancy, stenosis and regurgitation (insufficiency) most often occur in the mitral and aortic valves. Mitral stenosis is the most common valvular dysfunction during pregnancy [34]. During pregnancy, auscultatory murmurs of mitral and aortic stenosis increase, while murmurs in mitral and aortic regurgitation decrease. During pregnancy, unexpressed mitral and aortic regurgitation is easier to tolerate, stenosis are more difficult to tolerate and predispose to maternal and fetal complications. Mitral stenosis is especially dangerous. Tachycardia, increased blood volume and cardiac output during pregnancy aggravate the course of this disease, which is manifested by increased pressure in the capillaries of the lungs, leading to edema. Atrial fibrillation is also common.

9.1.1. Treatment

- In mitral stenosis, prevention of tachycardia, treatment of pulmonary edema and atrial fibrillation, and in some cases, valvotomy is used
- For aortic stenosis, if possible, surgical correction is used before pregnancy

Ideally, valvular disease should be diagnosed and treated with medication before conception, and surgical treatment is often indicated for severe disorders. In some situations (for example, for the prevention of endocarditis), prophylactic antibiotic therapy is necessary.

9.2. Mitral stenosis

The patient should be closely monitored throughout the pregnancy as mitral stenosis can progress rapidly. However, open heart surgery increases the risk to the fetus. Commissurotomy is safe enough during pregnancy if needed. To maximize diastolic flow through a stenotic mitral valve, tachycardia should be prevented.

Most often it manifests itself as signs of heart failure. Signs begin to appear from 12-20 weeks of pregnancy, hemodynamic recovery occurs 2 years after childbirth. Stages/degrees of mitral stenosis.

9.2.1. Stage I

Full compensation of blood circulation. There are no complaints, no shortness of breath even with physical exertion.

9.2.2. Stage II

Relative circulatory failure. After physical exertion, symptoms of circulatory disorders appear in a small circle in the form of shortness of breath, at rest there is no shortness of breath.

9.2.3. Stage III

The initial stage of severe insufficiency. The appearance of congestion in the lungs, increased venous pressure, enlarged liver, no edema, no atrial fibrillation.

9.2.4. Stage IV

Severe circulatory failure. Significant congestion in the small and large circle, increased venous pressure, significant increase and density of the liver, edema, ascites, enlarged heart, myocardial dystrophy.

+ V stage - dystrophic period of circulatory failure, the huge size of the liver, heart, ascites, anosarca.

If pulmonary edema develops, loop diuretics may be prescribed.

If atrial fibrillation occurs, anticoagulant therapy and heart rate monitoring are necessary [35]. Heart rate control is usually the same as in non-pregnant patients and includes beta-blockers, calcium channel antagonists, or digoxin.

During labor, the use of regional anesthesia (such as a slow epidural infusion) is most preferred.

9.3. Aortic stenosis

Aortic stenosis should be treated prior to pregnancy if possible, as surgical correction during pregnancy carries more risks and catheter valvuloplasty is not very effective [36].

During labor, local anesthesia is preferred, but general anesthesia is given if necessary. Regional (block) anesthesia should be avoided because it reduces the filling (preloading) pressure, which may already be reduced by aortic stenosis at this point.

Pushing, which can drastically reduce filling pressure and disrupt cardiac output, is not recommended in stage 2 of labor; vaginal delivery may be preferred. When indicated for obstetric reasons, a caesarean section is performed.

10. Mitral valve prolapse

Mitral valve prolapse is more common in young women and is often familial [37]. Mitral valve prolapse, as a rule, is an isolated pathology that has no clinical consequences; however, patients may also have some degree of mitral regurgitation. Rarely, mitral valve prolapse occurs with Marfan syndrome or atrial septal defect.

Women with mitral valve prolapse and associated mitral regurgitation usually tolerate pregnancy well. The relative enlargement of the ventricles of the heart during normal pregnancy reduces the disparity between the disproportionately large mitral valve and the ventricle.

Beta blockers are indicated for recurrent arrhythmias. Thrombosis and systemic embolism (due to concomitant atrial fibrillation) rarely occur, requiring anticoagulant therapy.

11. Congenital heart disease

For most patients who do not show symptoms of the disease, the risk does not increase during pregnancy [38]. However, patients with Eisenmenger syndrome (currently rare), primary pulmonary hypertension, or possibly isolated pulmonary artery stenosis are predisposed, for unknown reasons, to sudden death during childbirth, in the postpartum period (within 6 weeks of delivery) or after abortion at> 20 weeks gestation. Pregnancy is contraindicated for these patients. If such a patient becomes pregnant, during delivery, she should be closely monitored with a catheterized pulmonary artery and / or an established arterial line.

In patients with intracardiac shunts, the goal is to prevent right-to-left shunting by maintaining peripheral vascular resistance and minimizing pulmonary vascular resistance.

Patients with Marfan syndrome have an increased risk of dissection and rupture of aortic aneurysms during pregnancy. Requires bed rest, use of beta-blockers, avoidance of increased intrauterine pressure, and measurement of aortic diameter using echocardiography.

12. Perinatal cardiomyopathy

Heart failure arising from an unexplained cause (for example, myocardial infarction, valvular disease) can occur between the last month of pregnancy and 6 months after delivery in patients without a history of heart disease [39]. Risk factors include:

- Multiple births
- Age ≥ 30
- Multiple pregnancy
- Preeclampsia

The five-year mortality rate is 50% [40]. Relapse is likely in subsequent pregnancies, especially in patients with residual cardiac dysfunction, so pregnancies are not desirable.

Treatment is the same as for heart failure. Taking ACE inhibitors and aldosterone is contraindicated in most cases, but they can be used in cases where the expected benefit clearly outweighs the potential risk

13. Conclusion

Author should provide an appropriate conclusion to the article. Write conclusion as single para. Conclusion should be concise, informative and can be started with summarizing outcome of the study in 1-2 sentence and ended with one line stating: how this study will benefit to the society and way forward.

Compliance with ethical standards

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Disclosure of conflict of interest

The authors have no potential conflict of interest to disclose.

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