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THE IMPACT OF HEART DEFECTS ON THE COURSE OF PREGNANCY

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Abstract
Among extragenital pathologies, heart defects are the leading cause of maternal mortality. This review article presents current data on the impact of heart defects on the course of pregnancy. Information from Russian-language research literature was processed and analyzed using the electronic search systems cyberleninka.ru and eLIBRARY in the international Scopus database for the period 2020–2025.

Keywords
Congenital heart defects, acquired heart defects, mitral stenosis, rheumatic fever, septal defect, pregnancy, WHO, maternal mortality, diagnosis.

Introduction

Heart defects are divided into two groups: congenital and acquired

A congenital heart defect (CHD) is a structural abnormality of the heart and/or major blood vessels present from birth. Most defects disrupt blood flow within the heart or through the systemic and pulmonary circulatory systems. Heart defects are the most common congenital defects and the leading cause of infant mortality due to developmental abnormalities [1, 2].

Heart embryogenesis occurs between the 3rd and 9th weeks of pregnancy. Exposure of the woman to adverse factors can disrupt heart differentiation and lead to the formation of CHD. Such factors are generally considered to include:

- infectious agents (rubella virus, cytomegalovirus, herpes simplex virus, influenza virus, enterovirus, Coxsackie B virus, etc.);
- hereditary factors—in 57% of cases, CHD is caused by genetic abnormalities, which may occur either in isolation or as part of multiple congenital malformations; the most well-known causes of CHD are point mutations or chromosomal abnormalities in the form of deletions or duplications of DNA segments;
- the mother's somatic diseases, primarily diabetes mellitus, lead to the development of hypertrophic cardiomyopathy and CHD;
- occupational hazards and the mother's harmful habits (chronic alcoholism, computer radiation, mercury or lead poisoning, exposure to ionizing radiation, etc.)

[3].

The most common defects are: ventricular septal defect (VSD) (28.3%); atrial septal defect (ASD) (10.3%); pulmonary artery stenosis (9.8%); Tetralogy of Fallot (TOF) (9.7%); aortic stenosis (7.1%); aortic coarctation (5.1%); transposition of the great vessels (4.9%); tricuspid valve hypoplasia syndrome, patent ductus arteriosus (PDA), and total anomalous venous return are also encountered [1, 2, 4].

Acquired heart defects (AHD) remain a significant problem in cardiological obstetric practice, posing unique risks to both the mother and the fetus. Pregnancy induces significant hemodynamic shifts: an increase in circulating blood volume (CBV) by 40–50%, a 30–50% increase in cardiac output (CO), a 10–20 beats-per-minute increase in heart rate (HR), and a 20–30% decrease in systemic vascular resistance (SVR) by the end of the second trimester [5, 6]. These changes place a significant burden on the compensatory mechanisms of the heart with pre-existing valvular disease. The etiopathogenesis of PPS in women of reproductive age exhibits regional variations: in developing countries, rheumatic heart disease (RHD) predominates [7], whereas in developed countries, the role of degenerative lesions (myxomatous degeneration of the mitral valve, calcified aortic stenosis) and infective endocarditis is increasing [8]. Key pathophysiological mechanisms determining the severity of the condition during pregnancy include: in valve stenosis—the inability to increase cardiac output adequately to meet demand due to a fixed obstruction to blood flow, leading to pressure overload and pulmonary edema (particularly critical in mitral stenosis—MS); in valve insufficiency – relative tolerance to increased volume overload due to reduced afterload (CVC) [5, 9]. The main clinical risks include heart failure, pulmonary hypertension, arrhythmias (particularly atrial fibrillation), thromboembolic complications (especially in mitral stenosis and atrial fibrillation), and increased perinatal morbidity and mortality [5, 8, 10]. The relevance of this issue is underscored by the growing number of pregnant women with CHD, including those who have previously undergone valve surgery [11].

Main Section

Congenital Heart Defects

Prenatal care for women with CHD requires close monitoring from the earliest stages of pregnancy. In some cases, women with decompensated heart defects experience symptoms associated with their condition that are also characteristic of pregnancy: general malaise, nausea, vomiting, and changes in the function and condition of the

reproductive system (amenorrhea, cyanosis of the mucous membrane at the vaginal opening and cervix, and enlargement of the uterus). The symptoms identified in this group of women also occur in the absence of pregnancy, and as a result, erroneous conclusions are often drawn. Many pregnant women delay seeking care at a prenatal clinic out of fear that they will have to terminate the pregnancy [12, 13, 14, 15, 16]. By the time pregnancy occurs, most women consider themselves healthy, but with the onset of pregnancy and a high risk of complications, they require monitoring by an obstetrician-gynecologist, cardiologist, internist, and cardiac surgeon [17].

In women with congenital heart defects, the risk of developing serious complications such as heart failure, arrhythmias, cerebrovascular disease, and embolism—potentially leading to death—increases during pregnancy. Prolonged ventricular overload in terms of volume and pressure is possible due to heart valve disease or the presence of an uncorrected shunt, especially in cases of late surgical correction or palliative surgical defects. The development of arrhythmias, most commonly supraventricular, is associated with dilation of the heart chambers, leading to myocardial hypertrophy, fibrosis, scarring, and conduction system abnormalities. Pregnancy can trigger the development of arrhythmias even in a healthy heart due to physiological factors such as hormonal effects, physiological dilation, and changes in the autonomic nervous system [18].

Based on an assessment of the risks of pregnancy and childbirth in women with congenital heart defects, the American Heart Association (AHA/AAS), the American College of Cardiology, and the European Society of Cardiology (ESC) have developed guidelines for optimizing the management of this patient group [17]. When assessing risk factors in patients with congenital heart defects planning a pregnancy, it is necessary to consider the anatomical features of the defect, the type of previous surgical intervention, and to evaluate cardiac function based on medical history and stress test results. One of the main factors contributing to adverse pregnancy and delivery outcomes in women with congenital heart defects is the presence of heart failure, which is diagnosed during exercise testing [19].

To diagnose the location, functional capacity, and hemodynamic characteristics of the heart defect, additional diagnostic methods are required: electrocardiography, echocardiography with assessment of ventricular function, heart valve function, the degree of pulmonary hypertension, and the material from which prostheses and patches are made. In pregnant women with congenital heart defects, computed tomography (CT) and magnetic resonance imaging (MRI) of the heart are

performed, cardiac catheterization is conducted, and the concentration of cardiac biomarkers is determined. All patients with congenital heart defects who are only planning a pregnancy must be advised to undergo genetic testing and consult a geneticist [20].

Pregnancy itself places very high demands on the body's cardiovascular system, especially in women with congenital heart defects, whose compensatory capacity is already reduced. The main clinical symptoms in pregnant women with heart defects are nonspecific. Patients experience rapid fatigue, muscle weakness, heaviness in the legs, drowsiness, palpitations, and shortness of breath, which occur during physical exertion. As hemodynamic disturbances associated with congenital heart defects progress, shortness of breath is observed even at rest [21].

Congenital heart defects are one of the leading causes of maternal mortality during pregnancy and require an accurate diagnosis, close monitoring throughout pregnancy in collaboration with cardiologists and cardiac surgeons, and appropriately tailored medical and surgical management. Heart disease accounts for 28.5% of maternal mortality cases. In pregnant women with congenital heart defects, blood oxygen saturation decreases, leading to circulatory or mixed respiratory-circulatory hypoxia. Existing chronic maternal hypoxia leads to impaired uteroplacental blood flow and, consequently, to chronic fetal hypoxia [18, 22].

A working research group of the European Society of Cardiology proposed using a unified, modified WHO classification to assess the risk of cardiovascular complications for the mother and the unborn child in pregnant women with congenital heart defects and other cardiovascular pathologies. The proposed classification combines all cardiovascular risk factors, underlying heart disease, and comorbidities; it includes potential contraindications to pregnancy, timing of consultations with specialists, additional diagnostic procedures, and the timing and methods of delivery depending on the type of congenital defect and the presence of complications [18].

The modified WHO classification of maternal risk in cardiovascular diseases includes several groups of patients [7]. The first group includes conditions in pregnant women with a WHO risk assessment of I: uncomplicated, mild, or moderate pulmonary artery stenosis and patent ductus arteriosus; successfully repaired congenital heart defects, including atrial septal defect, ventricular septal defect, patent ductus arteriosus, anomalous pulmonary venous drainage, and rare atrial or ventricular extrasystoles. The second group includes conditions in pregnant

women with a WHO risk classification of II or III. WHO Class II includes: unoperated atrial septal defect and ventricular septal defect, corrected tetralogy of Fallot, and virtually all arrhythmias in the absence of other complications. WHO Class III includes: moderate left ventricular dysfunction, hypertrophic cardiomyopathy, valvular heart disease (not classified as WHO Class I or IV), Marfan syndrome not complicated by aortic dilatation, aortic diameter less than 45 mm in combination with a bicuspid aortic valve, and surgically treated aortic coarctation. WHO Class III, regardless of individual characteristics, includes: artificial mechanical valve, systemic right ventricle, Fontan procedure, unoperated heart defects with cyanosis, other complex congenital heart defects, aortic dilation of 40–45 mm in Marfan syndrome, aortic dilation of 45–50 mm with a bicuspid aortic valve. The third group includes conditions in pregnant women with a WHO risk class of IV, in which pregnancy is contraindicated. These include pulmonary arterial hypertension, left ventricular dysfunction with an ejection fraction of less than 30% and heart failure functional class III–IV, previous peripartum cardiomyopathy with residual left ventricular damage, severe mitral stenosis, severe aortic stenosis with subjective symptoms, Marfan syndrome with aortic dilation greater than 45 mm, aortic dilation greater than 50 mm with a bicuspid aortic valve, and severe aortic coarctation [23].

Diagnosis of congenital heart defects includes not only anamnestic indications of the presence of a defect and heart murmurs from birth or childhood, but also a physical examination. Palpation of the cardiac region, percussion of the heart and vascular bundle, and auscultation of heart sounds are mandatory. Laboratory tests are performed during the pre-pregnancy preparation phase at 10–11, 26–28, and 32 weeks of gestation, as well as after delivery, and must include an assessment of the coagulation system. Electrocardiography of the heart allows for the detection of signs of hypertrophy and overload in various parts of the heart, depending on the type of defect and the presence of hemodynamic abnormalities. In most cases, Doppler echocardiography and echocardiography allow for the detection of signs characteristic of heart defects, the assessment of the degree and severity of intraventricular hemodynamic disturbances, and the functional status of all heart chambers [18, 19, 22].

Acquired Heart Defects

Mitral Heart Defects

Among acquired heart defects in pregnant women, mitral heart defects account for

75–90% of cases, with isolated mitral stenosis (MS) occurring in only one-third of cases of rheumatic mitral heart defects. Most patients have a combined mitral valve defect: MS and mitral valve regurgitation (MVR). A heart defect is considered combined when stenosis and regurgitation of a single valve are present, and mixed when multiple valves are affected. If multiple defects are present, they are listed, with the most severe defect listed first. Maternal mortality in mild MS is less than 1%, in severe cases—5%, and rises to 17% with the onset of atrial fibrillation (AF) [23].

Mitral Stenosis

Mitral stenosis is a narrowing of the left atrioventricular orifice, leading to impaired diastolic blood flow from the left atrium to the left ventricle. It is the most common defect in young women, including pregnant women. MS is most often associated with a history of rheumatic fever; less commonly, the cause is infective endocarditis (IE) or systemic connective tissue diseases (SCTD): systemic lupus erythematosus (SLE) and rheumatoid arthritis (RA). Congenital MS is rare and may be associated with an atrial septal defect (ASD). The hemodynamic changes characteristic of pregnancy are particularly detrimental for this defect, to the extent that MS can take a very unfavorable course. It is believed that pregnancy itself increases the severity of MS by one stage. During pregnancy, the transmitral gradient increases, especially in the second and third trimesters. Tachycardia contributes to a further increase in left atrial pressure due to a shortened diastole. The risk of pulmonary edema, heart failure, arrhythmia, and fetal growth restriction is high when the mitral valve orifice area is less than 1.5 cm^2 (or 1 cm^2 per 1 m^2 of body surface area). According to the current classification (adopted at the 6th Congress of Cardiologists and Cardiac Surgeons of Ukraine, Kyiv, 2000), five stages of mitral stenosis are distinguished [23].

Stage I — Compensation: Hemodynamic disturbances are caused by a slight narrowing of the mitral orifice (its area is 2–2.5 cm^2). Pressure in the left atrium is elevated to 10–15 mm Hg.

Stage II — pulmonary congestion is characterized by a narrowing of the mitral orifice to 1.5–2 cm^2, pressure in the left atrium rises to 20–30 mm Hg, and pulmonary artery pressure increases accordingly.

Stage III — right ventricular failure. Characterized by persistent hypertension in the pulmonary circulation with the formation of a “second barrier,” increased workload on the right ventricle, and the development of right ventricular failure. Sclerosis of

the pulmonary vessels and a reduction in pulmonary blood flow lead to a decrease in or disappearance of episodes of cardiac asthma and pulmonary edema. Dystrophic changes in the parenchymal organs are moderately pronounced. The area of the mitral orifice is 1–1.5 cm².

Stage IV — dystrophic. The area of the mitral orifice is less than 1 cm². It is characterized by marked circulatory impairment in the systemic and pulmonary circulations [23].

Patients with MS, like all pregnant women with CVD, require constant monitoring by a cardiologist (internist) and an obstetrician. Routine hospitalization of pregnant women with heart defects is usually performed three times (more often if necessary): before 12 weeks—for an initial examination, confirmation of the diagnosis, and a decision on the feasibility of carrying the pregnancy to term; at 28–32 weeks—the period of maximum hemodynamic stress—for a repeat comprehensive examination; at 36–38 weeks—for comprehensive prenatal preparation.

Stage V—terminal. The clinical picture corresponds to Stage III according to the classification by N.D. Strazhesko and V.H. Vasilenko. It is characterized by severe circulatory disorders with irreversible degenerative changes in internal organs (liver, kidneys) and ascites, as well as atrophy of the muscular system [23].

Drug therapy is prescribed as indicated. When the left atrial pressure exceeds 50 mm Hg, even in the absence of any clinical symptoms, cardioselective β-blockers (metoprolol, bisoprolol, betaxolol) are indicated, with doses determined by left atrial pressure, heart rate, and blood pressure. If there are signs of congestion in the pulmonary circulation, diuretics (thiazide or loop) are prescribed. Spironolactone is contraindicated due to the risk of fetal feminization. If signs of heart failure and/or pulmonary hypertension exceeding 50 mm Hg persist despite adequate medical therapy, surgical correction of the defect is indicated [23].

Mitral regurgitation

Isolated mitral regurgitation of rheumatic origin accounts for approximately 10% of all acquired heart defects. In addition to rheumatic heart disease, the causes of mitral regurgitation include congenital abnormalities (valve cleft, prolapse); SLE (SLE, systemic sclerosis—SS, nonspecific aortoarteritis); IE; valvular apparatus pathology (chordae rupture, papillary muscle rupture as a complication of valve replacement). It should be noted that in this defect, pregnancy significantly lessens the severity of heart disease if the defect is not accompanied by marked regurgitation or heart failure. With normal left ventricular dimensions, pregnancy does not increase the

risk of complications for the mother and fetus. An increase in BCC and cardiac output leads to an increase in volume overload, which is a consequence of valvular regurgitation; however, a decrease in SVR reduces the degree of regurgitation, thereby compensating for the volume overload. Five stages of MI are distinguished [23].

Clinical variants of mitral regurgitation 1.

Secondary mitral regurgitation arises due to left ventricular dilation and dysfunction of any etiology, leading to structural and mechanical changes. These changes include: an increase in the diameter of the mitral annulus, a change in the direction of papillary muscle traction, and a decrease in the contractility of the LV myocardium and papillary muscles, which play an important role in maintaining normal mitral valve function.

Rheumatic mitral regurgitation.

Mitral regurgitation is a common manifestation of acute rheumatic carditis, which usually resolves over time. Chronic rheumatic disease more often presents as mitral stenosis; however, valve deformation frequently causes mitral regurgitation as well. Sometimes mitral regurgitation predominates over stenosis. In cases of combined mitral valve disease with predominant stenosis, the stages and indications for surgical treatment

— are the same as for mitral stenosis;

— with a predominance of regurgitation: the staging and indications for surgical treatment are the same as for mitral regurgitation; — without a clear predominance: the staging and indications for surgical treatment are the same as for mitral regurgitation [23].

Aortic insufficiency

Aortic insufficiency (AI) in pregnant women may result from valve disease: congenital bicuspid valve, rheumatic fever, infective endocarditis, valvulitis, trauma; due to aortic root disease: hereditary connective tissue disorders, syphilis, dissecting aortic aneurysm, SSA (ankylosing spondylitis, aortitis). Five stages of the defect are distinguished [23].

Tricuspid Valve Defects

Tricuspid valve defects, whether in pregnant women or in the general population, are virtually never seen as an isolated, independent heart condition. Furthermore, the extent of valve damage is usually not hemodynamically significant. The clinical presentation, prognosis, and treatment are generally determined by the involvement

of the mitral and aortic valves. The severity of tricuspid valve defects is classified into three grades: Grade I—mild; Grade II—moderate; Grade III—severe [23].

Conclusion

A review of the literature shows that both congenital and acquired valve defects increase the risk of heart failure, arrhythmias, pulmonary hypertension, and thromboembolic complications. Among acquired defects, mitral stenosis is the most dangerous: changes in hemodynamics during pregnancy sharply worsen the patient's condition. Valvular insufficiency against a background of reduced vascular resistance is better tolerated.

Early risk assessment using the modified WHO classification plays a key role. It allows determining whether pregnancy is permissible and planning the timing of hospitalizations, the scope of examinations, and the method of delivery.

A favorable outcome is possible with an accurate diagnosis, continuous monitoring by an obstetrician-gynecologist, cardiologist, internist, and cardiac surgeon, as well as timely medical and, if necessary, surgical intervention.

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