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Valve disease in pregnancy

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ABSTRACT

Maternal cardiac disease is a major cause of non-obstetric morbidity and accounts for 10–25% of maternal mortality. Valvular heart disease may result from congenital abnormalities or acquired lesions, some of which may involve more than one valve. Maternal and fetal risks in pregnant patients with valve disease vary according to the type and severity of the valve lesion along with resulting abnormalities of functional capacity, left ventricular function, and pulmonary artery pressure. Certain high-risk conditions are considered contraindications to pregnancy, while others may be successfully managed with observation, medications, and, in refractory cases, surgical intervention. Communication between the patient's obstetrician, maternal–fetal medicine specialist, obstetrical anesthesiologist, and cardiologist is critical in managing a pregnancy with underlying maternal cardiac disease. The management of the various types of valve diseases in pregnancy will be reviewed here, along with a discussion of related complications including mechanical prosthetic valves and infective endocarditis.

Maternal cardiac disease is a major cause of non-obstetric morbidity and accounts for 10–25% of maternal mortality.[1] Cardiac disease complicates up to 4% of pregnancies in the United States.[2] Changes in maternal physiology during pregnancy can worsen or even unmask previously undiagnosed cardiac disease. Congenital heart disease is emerging as the most common etiology of maternal cardiac disease since treatment advances over the past few decades have allowed more affected children to reach their childbearing years.[3] Acquired heart disease from medical conditions such as hypertension, diabetes, and hypercholesterolemia is also encountered more often as social factors, and assisted reproductive technology may postpone childbearing into the fourth, fifth, and even sixth decades of life.

Communication between the patient's obstetrician, maternal–fetal medicine specialist, obstetrical anesthesiologist, and cardiologist is critical in managing a pregnancy with underlying maternal cardiac disease. Ideally, a combined clinic setting can be established where obstetric and cardiac assessment may be accomplished during the same visit, thus facilitating a unified management approach and reducing the need for travel among patients at risk of functional deterioration. Cardiac risk level will determine the frequency of cardiac evaluation during pregnancy. For example, the 2011 European Society of Cardiology (ESC) guidelines recommend monthly or bimonthly follow-up for moderate to severe mitral stenosis, but only in the preconception period and every trimester for those with mild mitral stenosis.[4]

Physiology of normal pregnancy

Adaptive circulatory changes beginning within the first 5–8 weeks of pregnancy are designed to maximize oxygen

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delivery to maternal and fetal tissues. Cardiac output rises 30–50% above baseline through a combination of a 50% rise in blood volume,[3] a 15–20 bpm increase in maternal heart rate, and a 20–30% increase in stroke volume.[5–8] The low resistance within the uteroplacental circulation plus systemic vasodilation related to changes in progesterone, angiotension II, and norepinephrine all contribute to reduced systemic vascular resistance and an approximately 20% decrease in blood pressure by 24 weeks.[9–15]

These physiologic changes place additional stress on the maternal heart, which may prompt patients with underlying heart disease to decompensate with advancing gestational age. Distinguishing the normal symptoms and signs of pregnancy from those reflecting cardiac disease in the mother can be challenging, as many of these symptoms overlap. For example, signs and symptoms of normal pregnancy include decreased exercise tolerance, peripheral edema, mildly elevated heart rate, and a systolic ejection flow murmur less than grade III/VI.[16] Dyspnea that worsens with gestational age or that occurs at rest, angina, syncope, and most diastolic murmurs, on the other hand, warrant further investigation into cardiac function.[17] An electrocardiogram can be a useful objective tool, although subtle changes such as shortening of the PR and QT intervals, slightly leftward deviations in the frontal lead axis, and nonspecific abnormalities of the ST segment and T waves occur in 4–14% of normal pregnancies.[18–20]

Marked fluctuations in cardiac output occur during normal labor and delivery. Pain and anxiety increase sympathetic tone. Each contraction redistributes and autotransfuses approximately 500 mL of intermyometrial blood, resulting in an increased cardiac output of approximately 50%, from 7 L/min at term to 11 L/min at the end of the first stage of labor.[2] Abrupt relief of vena cava obstruction after delivery similarly results in a 50% increase in cardiac output and 60–70% increase in cardiac stroke volume 1 h after delivery.[21]

If there are concerns about cardiac function, labor may be induced under controlled conditions. Decisions about induction timing involve many factors, including the cervical exam, gestational age, cardiac status, and availability of various members of the inter-disciplinary team. Most pregnant women with cardiac disease should be allowed a trial of labor, since cesarean delivery is associated with more blood loss, increased risk of wound infection, postoperative immobility, and thrombosis. However, a long induction in a woman with an unfavorable cervix should be avoided. Strict monitoring of fluid intake and urine output, supplemental oxygen administration as needed, and cardiac telemetry if the patient is prone to an arrhythmia should be employed during labor. Once in the second stage, the undesirable circulatory effects of the Valsalva maneuver may be avoided by assisting the delivery with either low forceps or vacuum extraction.

Lumbar epidural anesthesia lowers pain-induced increases in sympathetic tone, reduces the urge to push, and provides excellent anesthesia for vaginal delivery or operative vaginal delivery as needed. Slow dosing of anesthetic agents and appropriate hydration according to the patient's cardiac lesion can maintain stable hemodynamics and adequate uteroplacental perfusion. Opiates such as fentanyl may be used in patients sensitive to preload and afterload, as they do not lower peripheral vascular resistance.[22]

Postpartum methergine should be avoided in patients with valve disease due to the potential risk of coronary artery vasoconstriction and elevation of systemic blood pressure. Postpartum fluid shifts affect preload and afterload and may result in heart failure. As such, patients with significant disease should be monitored in an intensive care setting for at least 12–24 h following delivery. Cardiac status gradually returns to baseline by 2–6 weeks postpartum.

Overview of maternal and fetal risks during pregnancy

Pregnancy-related complications in women with heart disease, including pulmonary edema, arrhythmia requiring treatment, stroke, cardiac arrest, or death, occurred in 13% of 252 pregnancies in a retrospective study of 221 women, with 55% of complications occurring in the antepartum period.[23] Maternal mortality varies depending on the specific lesion and the degree of myocardial dysfunction. The major risk of complications, including mortality greater than 25%, is associated with pulmonary hypertension, complicated aortic coarctation, and Marfan syndrome with aortic root involvement.[3]

The history, physical examination, electrocardiogram, and echocardiogram form the foundation of objective cardiac evaluation. A patient's individual risk can be assessed based on the severity of her cardiac lesion and the degree of ventricular dysfunction.

A multicenter, prospective study observed 562 pregnant women with heart disease from the second trimester through 6 months postpartum.[24] Four predictors of maternal complications were identified, including the following:

- A prior cardiac event, such as heart failure, transient ischemic attack, stroke, pulmonary edema before pregnancy, or arrhythmia.
- Poor pre-pregnancy functional class [New York Heart Association (NYHA) class greater than II], or the presence of cyanosis.
- Left heart obstruction defined as a mitral valve area $<2\ cm^2$, aortic valve area $<1.5\ cm^2$, or an aortic outflow gradient >30 mmHg by echocardiography.
- Left ventricular ejection fraction $<40\%$.

The authors suggest using a risk index to predict maternal cardiac morbidity where zero, one, or greater than one of these factors correlate with a 5%, 27%, or 75% risk of a cardiac event in pregnancy, respectively. Neonatal outcome also seems to correlate with this maternal risk score, likely related to preterm delivery indicated by maternal status.

An additional tool used to follow-up pregnant women with heart disease is serial measurement of brain natriuretic peptide (BNP). A prospective study from Toronto evaluated BNP levels during pregnancy in 66 women with heart disease and 12 healthy women and found that those with heart disease had significantly higher BNP levels than those