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Mitral Stenosis and Pregnancy

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Abstract

Maternal cardiac disease is a major risk factor for nonobstetric mortality and morbidity in pregnancy. Rheumatic heart disease is less common than in the past, but is still a major heart problem associated with pregnancy in developing countries. Mitral stenosis (MS) is most common rheumatic valvular lesions seen in pregnancy and is poorly tolerated. A series of cardiocirculatory changes including increase in blood volume, cardiac output, stroke volume and heart disease, decrease in blood pressure and systemic vascular resistance occur in pregnancy beginning from the early first trimester. These hemodynamic changes may provoke clinical manifestations of cardiac complications during pregnancy. The normal mitral valve orifice area is about 4–6 cm^2. When the valve area is reduced <2 cm^2 the classical symptoms of mitral stenosis start appearing. These symptoms include dyspnea, ortophnea, paroxysmal nocturnal dyspnea and decreased exercise capacity. Cardiac decompensation and pulmonary edema usually occur in second or third trimester as the hemodynamic burden of pregnancy is greatest. Although physical examination, assessment of functional capacity, electrocardiogram and chest X-ray may reveal many clues, echocardiography is the standard imaging modality used to assess patients with MS. The maternal cardiac complications correlate with the New York Heart Association (NYHA) functional classification and the severity of the mitral stenosis. Management depends upon the severity of disease, symptoms and time of diagnosis. The risks associated with pregnancy should be discussed with patients. In pregnancy medical treatment should be the first line of management. If symptoms persist despite optimal medical treatment invasive procedures should be considered. Multiple studies have shown that vaginal delivery under epidural anaesthesia is safe and well tolerated, unless obstetrically contraindicated. Understanding of the physiological changes in pregnancy and the pathogenesis of mitral stenosis and a multidisciplinary team approach with a cardiologist, obstetrician, and obstetric anesthesiologist in management provides decreased mortality and morbidity.

Keywords

Cardiac Disease in Pregnancy, Mitral Stenosis, Valvular Heart Disease in Pregnancy

1. Introduction

Maternal cardiac disease is a major risk factor for nonobstetric mortality and morbidity in pregnancy. About 20% of the pregnants with heart disease are complicated by a cardiovascular event such as heart failure, hypertensive disorder or arrythmia [1]. Rheumatic heart disease is less common than in the past, but is still a major heart problem associated with pregnancy in developing countries as in Turkey. Mitral stenosis (MS) is most common rheumatic valvular lesions seen in pregnancy and is poorly tolerated. Although MS is usually associated with mitral regurgitation, morbidity and mortality is often related to mitral stenosis [2]. We reviewed the recent reports and aimed to provide a practical approach about the risk assessment and the optimal cardiac and obstetric management of MS in pregnancy.

2. Hemodynamic Changes During Pregnancy, Labor and Delivery

A series of cardiocirculatory changes including increase in blood volume, cardiac output, stroke volume and heart disease, decrease in blood pressure and systemic vascular resistance occur in pregnancy beginning from the early first trimester. The cardiac output increases by approximately 40% of the pre-pregnant values. These hemodynamic changes which peak between the second and third trimesters may provoke clinical manifestations of cardiac complications during

pregnancy. The cardiovascular changes of pregnancy resolve by 3-6 months after delivery [3].

The normal mitral valve orifice area is about 4–6 cm^2.When the valve area is reduced <2 cm^2 the classical symptoms of mitral stenosis start appearing. MS prevents blood flow through left ventricle from the left atrium and reduces filling of the left ventricle, resulting in increased left atrial pressure, decreased stroke volume and decreased cardiac output. When the stenosis progresses, the left atrium dilates and the left atrial pressure increases A pressure gradient develops between the left atrium and the left ventricle during diastole. The magnitude of this gradient is the haemodynamic hallmark of MS. Hence, the back pressure on the pulmonary veins causes to pulmonary congestion and, even pulmonary edema in severe cases.

The physiologic changes in pregnancy can lead to an increased pressure gradient across the valve, decreased filling time, increased left atrial pressure and following symptoms of pulmonary edema respectively. These symptoms include dyspnea, ortophnea, paroxysmal nocturnal dyspnea and decreased exercise capacity. Patients with moderate to severe MS are more vulnerable to these hemodynamic changes. Cardiac decompensation and pulmonary edema usually occur in second or third trimester as the hemodynamic burden of pregnancy is greatest. Atrial fibrillation also increases the risk of pulmonary edema in MS. The maternal mortality risk is greatest during labour and the early post-partum period because of sudden increase in the pre-load immediately after delivery. There is a further increase in heart rate and blood pressure due to pain and anxiety. Each contraction during labor as well as lack of inferior vena cava compression lead to an increase in blood volume which can result in pulmonary congestion and heart failure. This preload increase may continue for 24-72 h after delivery and risk of pulmonary edema extends for several days after delivery [4].

3. Diagnostic Evaluation

Although physical examination, assessment of functional capacity, electrocardiogram and chest X-ray may reveal many clues, echocardiography is the standard imaging modality used to assess patients with MS. Echocardiography shows the area of the mitral valve, size and function of the both left atrium and ventricle, presence of thrombus and right-sided chambers. Doppler examination gives information about the severity of the stenosis, and the degree of pulmonary hypertension. But gradients in mitral stenosis in the pregnant women need to be evaluated with caution, as an increased heart rate leads to over-estimate, and impaired systolic function underestimate the degree of stenosis. Cardiac catheterization is necessary only when echocardiography is non-diagnostic. All women with valvular heart disease should ideally have preconception evaluation, including advice on risk prediction and contraception by a cardiac–obstetric team. Patients with symptomatic severe disease should be seen at a minimum of 4–8 week intervals until 36 weeks and then weekly until delivery.

4. Predictors of High Maternal and Fetal Morbidity

The maternal cardiac complications correlate with the New York Heart Association (NYHA) functional classification and the severity of the mitral stenosis [5]. In a large study of 80 pregnancies with mitral stenosis the risk for maternal complications were 67% for severe (MVA<1.0 cm^2), 38% for moderate (MVA 1.1 cm2>1.5 cm^2) and 26% for mild disease (MVA>1.5 cm^2). Consistent with the previous reports, a former cardiac event, abnormal functional capacity (NYHA>II) at baseline, impaired left ventricular systolic function (ejection fraction <40%) and left-sided heart obstruction (mitral valve area<2 cm^2, aortic valve area<1.5 cm^2) were defined to be predictors of adverse cardiac events by Silversides et al. [1].

5. Management

Management depends upon the severity of disease, symptoms and time of diagnosis. The risks associated with pregnancy should be discussed with patients. In patient with severe MS or moderate symptomatic stenosis, percutaneous mitral balloon valvuloplasty (PMBV) or valve reapair (if PMBV is not feasible) should be offered if MS is diagnosed prior to pregnancy. In patient with mild MS pregnancy is usually well tolerated. In pregnant patients medical treatment should be the first line of management.

6. Medical Therapy

The primary goal of treatment in mitral stenosis is to reduce heart rate in order to allow improved ventricular filling. Medical treatment includes bed rest, salt restriction, oxygen therapy, judicious use of diuretics and beta-blockers. Selective beta-1 adrenergic drugs should be preferred over nonselective beta blockers to avoid beta-2 mediated uterine relaxation [6]. Due to association with adverse fetal effects with diltiazem, verapamil is preferred calcium channel blockers, Digoxin is generally considered safe and well tolerated with few adverse fetal effects and digoxin may also be preferred for ventricular rate control in patients with atrial fibrillation. Electrical cardioversion can be performed safely during pregnancy if hemodynamic instability occurs or pharmacologic therapy fails to control the ventricular response. Atrial fibrillation requires anticoagulation to prevent systemic embolization. Due to embryopathy risk during the first trimester warfarin should be used during 12–36 weeks of pregnancy. Anticoagulation therapy during pregnancy should be;

- Unfractionated heparin for up to 12 weeks (aPTT 1.5–2.5-times of normal),
- Warfarin with a target INR of 2-3 between week 12-36
- Unfractionated heparin after 36 weeks [7].

Low-molecular weight heparin (LMWH) use instead of unfractionated heparin is very often. Although anti Xa activity is used for monitoring LMWH, no anti-Xa activity-based

guidelines have been published yet [8]. Because of the hypercoagulability in pregnancy prophylactic anticoagulation may be considered for patients with severe left atrial dilation and severe mitral stenosis despite the presence of sinus rhythm. Endocarditis prophylaxis is reserved only for patients with endocarditis history or presence of established infection [9].

If symptoms persist despite optimal medical treatment invasive procedures should be considered. During pregnancy, second trimester should be the preferred period for any invasive intervention. When anatomically suitable valves are present PMBV has a success rate about 90-95% (valve area to >1.5 cm^2 without a substantial increase in mitral regurgitation) and should be the first line invasive procedure [10]. Although the maternal outcome in PMBV and open commissurotomy are the same, the fetal mortality is higher in open commissurotomy, at a ratio of 1:8 [11]. Valve replacement should be reserved for severe cases with calcified valve and in mural thrombus where the maternal mortality is 1.5–5% and the fetal loss is 16–33% [12].

7. Labor and Delivery

Multiple studies have shown that vaginal delivery under epidural anaesthesia is safe and well tolerated, unless obstetrically contraindicated. Caesarean section is indicated for obstetric reasons only. Tachycardia, due to labour pain leads an increase in flow across the mitral valve and causes a sudden rise in left atrial pressure which can result to acute pulmonary edema. Epidural analgesia without significantly altering the patient haemodynamics prevents from this tachycardia.

Hemodynamic monitoring during labor is recommended in symptomatic patients or in patients with moderate to severe MS. Regional anaesthesia has proved to be a safe technique in cardiac patients presenting for caesarean section. Epidural and continuous spinal anaesthetic techniques are attractive options in order to reduce fluctuations in heart rate and cardiac output.

Using intrathecal fentanyl produces well enough analgesia without major haemodynamic changes during the first stage of labour, The uterine contractile force should be allowed rather than the maternal expulsive effort during the second stage of labour. Therefore, the second stage of delivery should be cut short by instrumentation. Supplementary analgesia for instrumentation with slow epidural boluses of fentanyl decreases cardiac pre-load [13].

Fetal heart rate monitoring is necessary during all stages of labour. Supplemental oxygen administration with pulse oximetry monitoring to minimize increases in pulmonary vascular resistance and maintenance of left uterine displacement for good venous return are mandatory.

General anaesthesia has some disadvantages including increased pulmonary arterial pressure and tachycardia during laryngoscopy and tracheal intubation. Furthermore the adverse effects of positive-pressure ventilation on the venous return may provoke cardiac complications as well [14].

8. Conclusion

In conclusion mitral stenosis in pregnancy is still a frequent cause of maternal death. Understanding of the physiological changes in pregnancy and the pathogenesis of mitral stenosis and a multidisciplinary team approach with a cardiologist, obstetrician, and obstetric anesthesiologist in management provides decreased mortality and morbidity.

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