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CASE REPORT AND REVIEW OF LITERATURE ON ANAESTHESIA MANAGEMENT OF A CASE OF LSCS WITH RHD WITH MILD PAH WITH MODERATE MS.

Medicine

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ABSTRACT

Rheumatic heart disease with mild Pulmonary Arterial Hypertension (PAH) in pregnancy is a grave situation, present with high maternal morbidity and mortality. In this case report, we describe the management of such a case which was even more difficult in combination with moderate mitral stenosis , mild pulmonary artery hypertension and with grade 1 MR with trivial TR.

We successfully did this case in epidural anesthesia in situ in close monitoring of cardiac physicians .

KEYWORDS

PAH , pregnancy, MS

INTRODUCTION

CARDIAC disease in pregnancy remains an important etiology of maternal and fetal morbidity and mortality.[1] Although the incidence of rheumatic heart disease (RHD) has decreased in developed countries, it still accounts for most of the cardiac disease–related maternal mortality in developing countries—as well as in immigrants to the United States from these nations.[2]

Mitral stenosis is the most commonly acquired valve lesion encountered in pregnant women and is almost invariably caused by RHD. Pregnancy and the peripartum period represent a physiologic burden that may worsen symptoms in even moderate degrees of cardiac disease. Consequently, many women are first diagnosed with cardiac disease during pregnancy. The need to provide labor analgesia or anesthesia for a Cesarean section to a woman with cardiac disease is not infrequent and can be challenging.

In this case report, we describe the management of such a case which was even more difficult in combination with moderate mitral stenosis , mild pulmonary artery hypertension and with grade 1 MR with trivial TR.

CASE REPORT

A 24-yr-old woman (gravida, 2; para, 1) with a history of Cesarean section delivery presented at 28 weeks gestational age with complete placenta previa, possible placenta accreta, and mild dyspnea. Although the patient complained of dyspnea with moderate exertion before pregnancy (class II symptoms, New York Heart Association Functional Classification system; (table 1), she was now unable to lie flat and even became dyspneic when doing minimal activity (class IV). Her symptoms improved with heart rate control using 25 mg metoprolol orally twice daily. Physical examination revealed an arterial blood pressure of 100/60 mmHg, a regular heart rate of 107 beats/min, and a respiratory rate of 20 breaths/min. There were diastolic murmurs noted. The patient's lungs were clear to auscultation bilaterally, and she had mild pedal edema. Electrocardiogram showed sinus tachycardia and left atrial enlargement. Preoperative transthoracic echocardiogram revealed moderate mitral stenosis, grade 1 mitral regurgitation, and mild pulmonary hypertension with estimated pulmonary artery systolic pressure of 38mmHg. There was trivial tricuspid regurgitation. Left and right ventricular systolic function were normal.

Table 1. New York Heart Association Functional Classification of Heart Disease Class Functional Description

- I Asymptomatic except during severe exertion
- II Symptomatic with moderate activity
- III Symptomatic with minimal activity
- IV Symptomatic at rest

The patient was admitted to the hospital for observation and management.. Multiple studies have shown that vaginal delivery is well tolerated in most patients with valvular heart disease.[3]Cesarean section is usually performed for obstetrical indications only. Because our patient had placenta previa, an elective repeat Cesarean section was planned at 36 weeks gestational age in an operating room with cardiopulmonary bypass capabilities with cardiac on standby.

As prophylaxis against acid aspiration, 30 ml sodium bicitrate was administered orally. While American Society of Anesthesiologists standard monitors were placed, the patient was positioned in the left uterine displacement position. Fetal heart rate monitoring was performed by one of the obstetricians from the time of entry into the operating room until surgical site preparation. The patient's initial systemic arterial blood pressure was 125/65 (mean 76) mmHg. Her heart rate was 105 beats/min. cvp was 8-9 cm of H2o

A graded epidural anesthesia was planned to maintain hemodynamic stability and to maintain optimum systolic BP, diastolic BP, HR, and prevent further rise in pulmonary vascular resistance (PVR). Epidural catheter was inserted through L1-L2 interspace with the patient seated and was placed 4 cm into the epidural space. Position was confirmed following administration of a test dose of 3 ml of 2% lignocaine with adrenaline. The patient was placed in the supine position with a left tilt of 15° to prevent aortocaval compression and a sensory block to T6 dermatome was achieved by 12 ml of 0.5% bupivacaine in fractionated doses of 3 ml over a period of 20 min with 50 µg fentanyl. Oxygen was administered by a face mask at 6 l/min throughout the intraoperative period. IV fluid infusion was guided by continuous monitoring of CVP. Arterial cannula for invasive BP monitoring was not instituted due to logistic constraints. It should be preferably used, if possible.

A male baby of 2.2 kg with Apgar score 9 and 10 at 1 min and 5 min, respectively, was delivered. Following delivery of the baby, 5 units of oxytocin was administered intramuscularly followed by 5 units in 500 ml of lactated ringer solution (RL) infused over 1 h. Hypotension (BP 90/52 mmHg,) occurred after oxytocin infusion was corrected by intermittent bolus of 50 µg of phenylephrine to a total of 300 mcg . Total 1 L of RL was infused maintaining a CVP of 5-7 cm of H_2O .

Duration of surgery was 45 min. Following surgery, BP 112/58 mmHg, MAP 73 mmHg, PR 110/min, SpO_2 100% in room air, and CVP 8 cm H_2O were recorded. The patient was transferred to the critical care unit for observation for 48 h. Analgesia was maintained with 0.125% bupivacaine at 5 ml/h infusion and paracetamol infusion (1 g in 100 ml) for 6 hourly. The patient was shifted to the ward after 48 h. Postoperative period was uneventful. Prescribed cardiac drugs were continued throughout the perioperative period. The patient was discharged from the hospital on 7th postoperative day after obtaining cardiological consultation for further management of underlying cardiac disease.

DISCUSSION

Normal pregnancy results in dramatic changes to the cardiovascular system (table 2). Pregnancy produces a 30 –50% increase in blood volume and cardiac output with physiologic anemia as a result of a greater increase in blood volume than red cell mass.[4-6] The increase in cardiac output is primarily the result of an increase in stroke volume with a smaller contribution from an increase in heart rate. Pregnancy reduces systemic vascular impedance. Anemia decreases blood viscosity with resultant decrease in systemic vascular resistance.

At the time of labor and delivery, pain and anxiety increase catecholamine release with resultant increases in heart rate, arterial blood pressure, and cardiac output. [4-6]Autotransfusion of up to 500 ml with each contraction increases preload and, hence, cardiac output. [7] After delivery, there is an additional increase in venous return as a result of autotransfusion from the contracting uterus as well as from the loss of fetal compression of the inferior vena cava.[8]

Rheumatic Mitral Stenosis

Acute rheumatic fever is an immune-mediated, multisystem inflammatory disease that is a sequella of group A streptococcal infection.[9] The pathogenesis of acute rheumatic fever is believed to involve the triad of a genetically susceptible individual, infection with a rheumatogenic strain of group A streptococcus, and an aberrant host immune response.[10] Rheumatic fever is characterized by carditis, valvulitis, arthritis, chorea, erythema marginatum, and subcutaneous nodules. Although the long-term effects of acute rheumatic fever on most tissues are minimal, its effects on the heart may be devastating. Inflammation leads to neovascularization, which enables further recruitment of T cells, leading to granulomatous inflammation and the establishment of chronic RHD.12 Carditis occurs in 30 – 80% of patients with acute rheumatic fever, and at least 60% of untreated patients develop chronic RHD.

Mitral Stenosis in Pregnancy

In general, mitral regurgitation is well tolerated by pregnant patients because the reduction in systemic vascular resistance reduces regurgitant flow. Mitral stenosis, however, is generally less well tolerated. In the normal adult, the mitral valve has an area of 4 – 6 cm2 . Among patients who do not have mitral valvular stenosis, there are no clinically significant increases in transmitral valvular gradient due to pregnancyinduced increases in cardiac output. With worsening mitral stenosis, however, increases in blood volume and cardiac output associated with pregnancy are less well tolerated. When mitral valve area is reduced to less than 2.0 cm2 , a pressure gradient develops across the mitral valve. The magnitude of this gradient depends on stenosis severity and the amount of blood flow across the valve. Thus, as cardiac output increases during pregnancy, the gradient across the diseased mitral valve increases. This increase in left atrial pressure is reflected back into the pulmonary venous circulation and increases the risk of pulmonary edema. Untreated, this progression results in pulmonary arterial hypertension that may lead to increases in right ventricular pressures and, possibly, to right ventricular failure.[11] The etiology of the pulmonary hypertension is usually passive and hence reversible after intervention; however, endothelial changes and vascular remodeling may affect its course.[12] Pulmonary hypertension is associated with extremely high maternal and fetal mortality.[13]

A mitral valve area greater than 1.5 cm2 usually does not cause symptoms at rest. However, as the severity of stenosis increases, patients develop decreased exercise tolerance, orthopnea, cough, paroxysmal nocturnal dyspnea, or pulmonary edema.[14] Furthermore, atrial arrhythmia associated with ventricular rate acceleration is a common cause of worsening symptoms. Although the clinical presentation of rheumatic mitral stenosis is usually associated with symptoms of congestive heart failure, patients may also present with hemoptysis and chest pain from pulmonary hypertension.[12]

Overall maternal mortality associated with mitral stenosis has been reported at 1%, but this number increases to 7% with worsening symptoms.[15]Desai et al.[11] found the most common complication to be pulmonary edema. The most significant risk factors for the prediction of maternal pulmonary edema were the severity of mitral stenosis, late antenatal presentation, moderate to severe symptoms outside of pregnancy, and cardiac disease diagnosed for the first time in the index pregnancy. Silversides et al.[16] reported that pulmonary edema and arrhythmia were the most common maternal complications; prematurity and intrauterine growth retardation were the most common fetal complications.

patients with mitral stenosis who develop worsening or recurrent heart failure despite medical therapy can be effectively managed with PBMV in experienced centers. An echocardiographic score developed by Wilkins et al[17] is used to select patients who may be appropriate candidates. Four variables of mitral valve morphology are assessed, as shown in table 4. Each variable is graded on a 5-point (0 – 4) scale. Total echocardiographic score is then derived as the sum of the points assigned for each variable, yielding a score between 0 and 16. Patients with an echocardiographic score of 8 or less have the best results from PBMV, whereas a score higher than 11 is associated with increased incidence of poor outcomes. Echocardiographic scores of 9 –11 are less predictive for good outcomes, but patients with extensive subvalvular disease tend to have poorer outcomes.

Anesthetic Goals.

With the increase in transmitral gradient associated with clinically significant mitral stenosis, more time for diastolic filling is necessary to ensure adequate preload. Whereas heart rate is inversely proportional to diastolic filling time, low heart rates are recommended. The decrease in diastolic left ventricular filling time associated with pregnancy-induced tachycardia further increases left atrial pressures. The main hemodynamic goal in mitral stenosis is to avoid tachycardia to optimize left ventricular diastolic filling time. The increase in transmitral gradient can be further complicated during labor. Tachycardia resulting from pain and -agonist tocolytic therapy further decreases diastolic left ventricular filling time. Vasodilation from neuroaxial blockade can also lead to reflex tachycardia. Other hemodynamic goals are the maintenance of normal to high preload, afterload, and contractility. Significant decreases in systemic vascular resistance, which result in reflex tachycardia, are poorly tolerated. Accordingly, in case of hemodynamic instability, the vasopressor choice should be tailored to these hemodynamic goals. Epinephrine should be avoided as it may induce tachycardia. Phenylephrine may restore stable hemodynamics with little or no unwanted effect on uteroplacental perfusion

CONCLUSION

Keeping the underlying pathophysiology in view, we aimed to maintain hemodynamic stability by maintaining an optimum SVR, preload, heart rate, sinus rhythm, and avoiding myocardial depression and increase in pulmonary vascular resistance. This was achieved by epidural anesthesia in a graded manner using small fractionated doses of local anesthetic to ensure a gradual onset of block and minimize hemodynamic changes resulting from sympathetic autonomic blockade. We chose to avoid general anesthesia in our patient to prevent the rise in pulmonary vascular resistance and worsening of pulmonary hypertension resulting from sympathetic stimulation during laryngoscopy, intubation, and nitrous oxide inhalation and to prevent myocardial depression in response to anesthetics. We administered oxytocin by intramuscular route and by slow intravenous infusion to avoid tachycardia and diastolic hypotension.[18]

Epidural anesthesia provides a safer alternative to general anesthesia in parturient with complex valvular lesions. However, it is easier said than done. Successful management necessitates strict vigilance and an extremely cautious approach to maintain the hemodynamic stability throughout the peripartum period.

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