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A STUDY ON PERIOPERATIVE OUTCOME OF PARTURIENTS WITH MITRAL STENOSIS UNDERGOING ELECTIVE CAESAREAN SECTION UNDER EPIDURAL ANAESTHESIA

Dr. S. Ramadevi M.D; D.A; Assistant Professor, Department of Anaesthesiology, Govt. Chengalpattu Medical College and Hospital, Chengalpattu.

Dr. C. Chitra* M.D; D.A; Assistant Professor, Department of Anaesthesiology, Govt. Chengalpattu Medical College and Hospital, Chengalpattu.*Corresponding Author

ABSTRACT

Rheumatic heart disease is the commonest cardiac disease complicating pregnancy in developing countries. Heart disease accounts for 15% pregnancy related mortality. In the presence of maternal heart disease, the circulatory changes of pregnancy may result in exacerbation of the haemodynamic perturbations due to complex cardiac valvular lesions. Determining the ideal anesthetic technique for Caesarean section in presence of such cardiac conditions remains a much debated topic. General anaesthesia is associated with a further increase in pulmonary pressure in response to laryngoscope and intubation along with myocardial depression by anaesthetic agents. Neuraxial blockade may lead to decrease in SVR and Cardiac output. In this article we present the successful anaesthetic management of a thirty parturient suffering from Rheumatic heart disease with mitral stenosis under epidural anaesthesia with good maternal and neonatal outcome. Successful management requires vigilant perioperative monitoring and thorough knowledge of the haemodynamics of cardiac valvular disease.

introduction

Cardiac disease in pregnancy remains an important etiology of maternal and fetal morbidity and mortality. Prevalence of heart disease in pregnancy varies from 0.3-3.5%. 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.

We hereby discuss the anaesthetic management for Elective Caesarean section of thirty parturient suffering from Rheumatic heart disease with mitral stenosis in our tertiary care teaching hospital.

STUDY METHODOLOGY:

We started to register all parturient suffering from Rheumatic heart disease with mitral stenosis in our antenatal outpatient department and all patients were given regular cardiologist and obstetrician follow up. Our registration period was one year. Out of total 78 patients enrolled,32 patients were planned for elective caesarean section for various obstetric indications.

Inclusion criteria:

1. Rheumatic Heart Disease with mitral stenosis
2. Mild pulmonary hypertension
3. Mitral valve area >1.5 cm2
4. Sinus rhythm
5. Post balloon mitral valvotomy
6. Post closed mitral commissurotomy
7. No other comorbid illness
8. NYHA class 1 and 2
9. Patients with Informed consent

Exclusion criteria:

1. Atrial fibrillation or other arrhythmias
2. Moderate to Severe pulmonary hypertension
3. Left atrial clot
4. Previous history of embolic phenomenon
5. Mitral valve replacement
6. Patients on Anticoagulants
7. Abnormal coagulation profile
8. NYHA class 3 and 4
9. Other comorbid illness
10. Non cooperative patients

Out of 32 patients 3 patients were excluded from our study for reasons of atrial fibrillation, hyperthyroidism and pregnancy induced hypertension respectively. A detailed medical history was elicited thereafter for all study patients prior to surgery.

In the holding area we gave 0.3 M sodium citrate 30 ml, Inj.Ranitidine 50 mg IV as antiaspiration prophylaxis. Infective Endocarditis prophylaxis was given with 2 gm of Inj.Ceftriaxone IV. High risk informed consent was obtained. Ventilator was reserved for standby in postoperative ICU.

All Emergency drugs including drugs like Esmolol, Metoprolol, Phenylephrine,Lignocard, Amiodarone, Frusemide were kept ready to manage tachycardia,hypotension, arrhythmias and pulmonary edema. Defrillator kept ready for emergency cardioversion if needed so.

All the equipments and drugs for emergency intubation and conversion to General Anaesthesia were arranged. ECG, NIBP, SPO2 and urine output were monitored in all patients.

Intra operative management:

Our goal is to maintain normovolemia, avoid tachycardia, hypotension, hypoxia, hypercarbia and volume overload. Titrated IV fluid infusion was started.

With the patient in sitting position under strict aseptic precautions, Epidural needle was inserted through L1-L2 inter space and epidural space was identified with loss of resistance technique and epidural catheter was placed 6 cm into the epidural space. Patients were turned supine and a wedge kept under her right hip for left uterine displacement and to prevent aortocaval compression and supine hypotension. Position was confirmed following administration of a test dose of 3 ml of 2% lignocaine with adrenaline. A sensory block to T6 to L2 dermatome was achieved with 12 ml of 2% lignocaine with adrenaline in fractionated doses of 4ml over a period of 15 minutes.Oxygen was administered by face mask at 6 liter/min throughout the intra operative period. Elective Caesarean was carried out by experienced obstetricians in all patients. Duration of surgery was 30 to 45 minutes.

Heart rate was maintained between 70 to 80 per minute with intermittent bolus of IV Esmolol 10 mg to a maximum of 30mg during surgery. Even minimal hypotension was rapidly corrected with intermittent bolus of 50 microgram of Phenylephrine to a total of 300 mcg . IV fluid infusion was carefully titrated so as to maintain near normal to the baseline blood pressure. Total of 800ml to 1000ml of intravenous fluid was given to the patient during surgery. Following delivery of the baby 10units of Oxytocin was administered intramuscularly and 10 units of Oxytocin IV infusion started. Neonatal outcome of all the study patients was good.

We avoided Methyl ergometrine, ProstaglandinF2alpha, and manual removal of placenta in these patients to prevent sudden uterine contraction and rapid auto transfusion of blood from the

uterus to the heart. Analgesia was maintained with epidural top up of 50 microgram of fentanyl at the end of procedure. Intraoperative period was uneventful in all patients.

Patients were shifted to the ICU for intensive care. Head up position with fluid infusion of 50 ml per hour was adviced. Paracetamol infusion (1 gm in 100ml) was given every 8TH hourly. After 6 hours IV fluids were stopped and oral liquid was started. On the night of surgery we gave 10ml of 0.0625% Bupivacaine epidurally. Epidural catheter was removed after 24 hours. Post operative period was uneventful. Prescribed cardiac drugs were continued throughout the perioperative period and she was put on appropriate antibiotics. Patient was discharged from the hospital on 7THpost operative day after obtaining cardiologist consultation for further management of underlying cardiac disease.

DISCUSSION:

Cardiac disease in pregnancy remains an important etiology of maternal and fetal morbidity and mortality. Mitral stenosis is the most common acquired valvular 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 Caesarean section to a woman with cardiac disease is not infrequent and can be challenging.

PHYSIOLOGICAL CHANGES IN PREGNANCY ON CVS:(1)

Variable	Peak Change, %
Blood volume	+35
Plasma volume	+45
Heart rate	+20
Stroke volume	+30
Cardiac output	+40
Contractility	Variable
Central venous pressure	Unchanged
Pulmonary capillary wedge pressure	Unchanged
Systemic vascular resistance	−15
Systemic blood pressure	−5

Normal pregnancy results in dramatic changes to the cardiovascular system. 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.(2)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. Autotransfusion of up to 500 ml with each contraction increases preload and, hence, cardiac output. 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.

PATHOPHYSIOLOGY OF MITRAL STENOSIS:

Mitral stenosis results in accumulation of blood in the left atrium and accompanying increases in the left atrial pressure. The normal left atrial pressure is 6-12mmHg. Pulmonary edema is likely to develop when left atrial pressure exceeds 30 mmHg. Increased left atrial pressure predisposes to atrial fibrillation because the associated enlargement of the left atrium increases the distance of the travel of cardiac impulse and thus increasing the likelihood of reentry. There is intense constriction of pulmonary arterioles with resulting pulmonary hypertension and right ventricular hypertrophy.

Rheumatic process causes the valve leaflets to thicken, calcify and funnel shaped. Mitral commissures and chordate tendinae fuse and shorten and thus the valve cusps become rigid as a result the leaflets exhibit doming or bowing shape(3) during diastole in ECHO.

1. Slow rise of end diastolic transvalvular pressure gradient to maintain cardiac output.
2. Left atrial dilatation promote reentry pathways and hence arrhythmias like atrial fibrillation.
3. Stasis of blood flow in the left atrium promotes the formation of thrombi and thus predispose the threat of embolisms.
4. Chronic rise in left atrial pressure is followed by backpressure on pulmonary vasculature and produce irreversible changes and thus ending in pulmonary hypertension.
5. Reduced lung compliance result in dyspnoea due to increase in work of breathing.
6. Pulmonary hypertension result in right ventricular hypertrophy and eventually right ventricular failure.
7. Marked dilatation of the right ventricle can result in tricuspid and pulmonary valve regurgitation.
8. Embolic events due to dislodgement of left atrial clot .Cerebral embolism is common.
9. Haemoptysis result from rupture of pulmonary bronchial venous communications.
10. Enlarged left atrium compress the left recurrent laryngeal nerve and thus produce hoarseness of voice.
11. The left ventricle is chronically underloaded in patient with mitral stenosis.At the same time the leftatrium,right atrium and right ventricle are frequently dilated and dysfunctional.

GENERAL EVALUATION OF PATIENTS:

Preoperative evaluation should be primarily concerned with determining the identity and severity of the lesion, residual ventricular function and the presence of any secondary effects on pulmonary, renal, and hepatic function. History of Dyspnoea, Orthopnoea, Paroxysmal Nocturnal Dyspnoea, exercise tolerance, fatigability, pedal edema, chest pain, palpitations, syncopal attacks and neurological deficits secondary to embolic phenomena should be asked for and documented.

Murmurs occur as a consequence of the accelerated blood flow through narrowed openings in stenotic and regurgitant lesions. Although systolic murmurs may be related to increased blood flow velocity, the ACC/AHA guidelines report that all diastolic and continuous murmurs reflect pathology.

The New York Heart Association functional classification of heart disease is useful for grading the severity of the disease and estimating the prognosis.(4)

NYHA Class	Symptoms
I	Cardiac disease, but no symptoms and no limitation in ordinary physical activity, e.g. no shortness of breath when walking, climbing stairs etc.
II	Mild symptoms (mild shortness of breath and/or angina) and slight limitation during ordinary activity.
III	Marked limitation in activity due to symptoms, even during less-than-ordinary activity, e.g. walking short distances (20–100 m). Comfortable only at rest.
IV	Severe limitations. Experiences symptoms even while at rest. Mostly bedbound patients

MEDICAL HISTORY:

Patients with valvular heart disease would be on medications like diuretics, vasodilators,ACEinhibitors, Betablockers, Antiarrythmics and anticoagulants. A detailed medical history has to be taken from the patient for appropriate anaesthetic management. Surgical or interventional procedures like comissurotomy,balloon valvotomy or valve replacement should be well documented.

CLINICAL ASPECTS OF MITRAL STENOSIS:

Mitral stenosis almost always occur as a delayed complication of rheumatic fever. The stenotic process is estimated to begin after a minimum period of 2 years of the RHD and results in progressive fusion and calcification of the valve leaflets.The normal mitral valve area is 4-6 cm^2. Symptoms generally develop after 20 to 30 years

when mitral valve area is reduced to less than 1.5 cm^2.

SEVERITY	MITRAL VALVE AREA Cm2	END DIASTOLIC PRESSURE GRADIENT [In sinus rhythm]in mmHg	PAP IN mmHg	SYMPTOMS
MILD	>1.5	<5	Normal< 30	Usually absent
MODERATE	1.0-1.5	5-10	30-50	NHYA Class 2
SEVERE	<1.0	>10	>50	NHYA Class 3 to 4

Mitral valve area <0.8 cm^2 is often referred as critical mitral stenosis.In severe mitral stenosis the trans valvular pressure gradient rises to 20mmHg and thus even minimal exertion can produce dyspnoea. Although cardiac output is nearly normal at rest, it fails to increase the blood flow during exertion due to decreased left ventricular preload. It becomes a fixed cardiac output state both at rest and exertion.

ANAESTHETIC CHALLENGES AND GOALS:

The plan of anaesthesia varies from patient to patient based on the severity of the valvular heart disease. Anaesthetic management is challenging and crucial. A multidisciplinary approach with a team of Obstetricians, Cardiologists, Anaesthetists and emergency physicians are mandatory for the management of a parturient with a cardiac disease .

In the antenatal period patients are advised to avoid excess weight gain, avoid excess physical activity and advised to take low sodium diet and seek appropriate medical advice and management whenever needed.

Cardiac decompensation occurs mostly in 18-24 weeks, during labour and immediate postpartum(5).

Our anaesthetic goals are focused to maintain sinus rhythm if present preoperatively, to avoid tachycardia, hypotension, hypoxia and hypercarbia and to maintain euvolemia .

Segmental blockade with a lumbar epidural anaesthesia(6) is recommended when the clinical findings are of mild severity. An incrementally dosed lumbar epidural will provide the least amount of hemodynamic alteration. Vasopressors preferred is phenylephrine since it lack beta adrenergic agonist activity. Beta blockers can be used to avoid tachycardia and help in providing increased diastolic time for ventricular filling. Titrated intravenous fluid should be given to avoid pulmonary edema. Vasodilatation that occur in neuraxial anaesthesia can lead to peripheral venous blood pooling and inadequate volume delivery to the left ventricle. This can precipitate haemodynamic collapse. Hence maintanence of euvolemia with titrated IV fluids and vasopressors is very important to prevent such untoward events.

General anesthesia may provide very stable hemodynamics if the sympathetic stimulation associated with laryngoscopy and intubation are attenuated either by use of anaesthetic agents or blockade. During the intraoperative period adequate depth of anaesthesia and analgesia is required to avoid tachycardia and hypertension. General anesthesia provides the advantages of definitive airway control and the ability to use transesophageal echocardiographic monitoring throughout the procedure. In the case of Caesarean section, however, this technique could result in prolonged neonatal respiratory depression(7).

INVESTIGATIONS AND MANAGEMENT:

The diagnosis of MS should be based on the history, physical examination, chest X-ray, and ECG. The diagnostic tool of choice in the evaluation of a patient with MS is 2-D and Doppler echocardiography(8).The morphological appearance of the mitral valve apparatus should be assessed by 2-D echocardiography, including leaflet mobility, leaflet thickness, leaflet calcification, subvalvular fusion, and appearance of the commissures(9).

The mean transmitral gradient can be accurately and reproducibly measured from the continuous wave Doppler signal across the mitral valve with the modified Bernoulli equation.

Medical Therapy

1. General principles(10):

Because MS is primarily caused by rheumatic fever, prophylaxis against rheumatic fever is recommended. Appropriate endocarditis prophylaxis is also recommended. Agents with negative chronotropic properties, such as -blockers or calcium channel blockers, may be of benefit in patients with sinus rhythm who have exertional symptoms if the symptoms occur with high heart rates. Salt restriction and intermittent administration of a diuretic are useful if there is evidence of pulmonary vascular congestion. Digitalis does not benefit patients with MS in sinus rhythm unless there is left and/or right ventricular dysfunction.

2. Atrial fibrillation:

Atrial fibrillation develops in 30% to 40% of patients with symptomatic MS. Significant hemodynamic consequences may result from acute development of atrial fibrillation, with loss of atrial contribution to LV filling, and from the rapid ventricular rate. Treatment of an acute episode of rapid atrial fibrillation consists of anticoagulation with heparin and control of the heart rate response. Intravenous digoxin, calcium channel blockers, or -blockers should be used to control ventricular response. If there is hemodynamic instability, electrical cardioversion should be undertaken urgently, with intravenous heparin before, during, and after the procedure.

Anticoagulation in Atrial fibrillation: Patients with severe MS, LA diameter >55mm,Prior embolic events, atrial fibrillation are indications of anticoagulation in Mitral stenosis.

3. Indications for Surgical or Percutaneous Valvotomy:

In the asymptomatic patient who has documented mild MS (valve area >1.5 cm2 and mean gradient <5 mm Hg), no further evaluation is needed on the initial workup. These patients usually remain stable for many years. If MS is more significant, further evaluation should be considered if the mitral valve morphology appears to be suitable for mitral valvotomy with pliable, noncalcified valves with little or no subvalvular fusion and no calcification in the commissures. Patients with moderate pulmonary hypertension at rest (pulmonary artery systolic pressure >50 mm Hg) and pliable mitral valve leaflets may be considered for percutaneous mitral valvotomy even if they deny symptoms. Objective limitation of exercise tolerance with a rise in transmitral gradient >15 mm Hg and pulmonary artery systolic pressure >60 mm Hg may be an indication to consider percutaneous valvotomy if mitral valve morphology is suitable(11).

The immediate results of percutaneous mitral valvotomy are similar to those of mitral commissurotomy. The mean valve area usually doubles (from 1.0 cm2 to 2.0 cm2), with a 50% to 60% reduction in transmitral gradient. Overall, 80% to 95% of patients may have a successful procedure, which is defined as a mitral valve area >1.5 cm2 and a decrease in left atrial pressure to ≤18 mm Hg in the absence of complications.

More than 90% of patients free of events remain in NYHA functional Class I or II after percutaneous mitral valvotomy(9).Relative contraindications to percutaneous balloon valvotomy include the presence of a left atrial thrombus and significant (3+ to 4+) MR.If there is significant calcification, fibrosis, and subvalvular fusion of the mitral valve apparatus, commissurotomy or percutaneous balloon valvotomy is less likely to be successful, and MVR will be necessary. For the patients with NYHA functional Class III symptoms due to severe MS or combined MS/MR, MVR results in excellent symptomatic improvement. Severe PHT with pulmonary artery systolic pressure of 60mm-80mmHg are the candidates for MVR.

CONCLUSION:

The Anaesthetic plan and management should be based on the

severity of the disease and care must be taken to adhere to our anaesthetic goals as mentioned in our discussion. A multidisciplinary approach and management should start from antenatal period and should extend to entire perioperative period for the successful maternal and foetal outcomes.

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