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Atrial fibrillation in pregnancy: An Anaesthetic predicament

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***Abstract*---**Developing countries with higher prevalence of RHD encounter cases of AF in pregnancy. Szekely et al reported new onset of AF in 8% of pregnant patients with RHD.[1] Dilatation of LA, elevated LA pressure and myocardial stretch due to mitral valve disease results in slow conduction velocities, increased dispersion of refractoriness and automaticity leading to AF. Occurrence is common in third trimester due to Increased HR, cardiac output, heart size, plasma volume, wall tension and decreased SVR. AF intensifies hemodynamic changes and predisposes to heart failure.[2]

***Keywords*---**Atrial fibrillation, Rheumatic heart disease, Pregnancy, Mitral valve repair, LSCS.

Introduction

Presentation of case

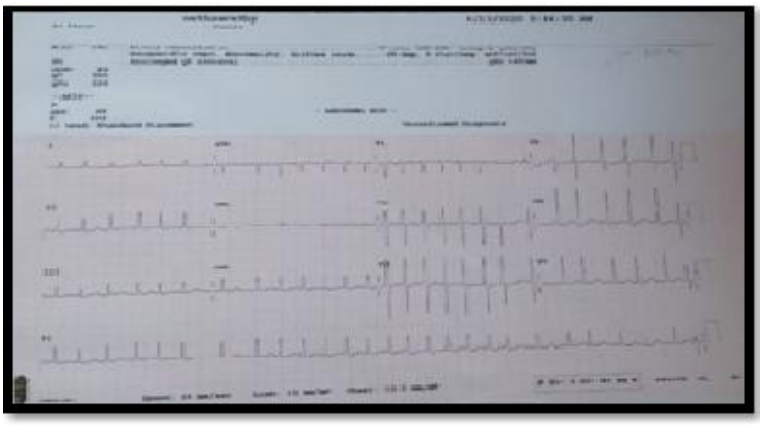
23-year-G2A1lady, 38weeksgestation, presented with generalized weakness and increasing shortness of breath. She was a known case of rheumatic mitral stenosis, post mitral valve repair with tricuspid valvuloplasty 8years ago, initiated on oral Warfarin 5mg OD, IV penicillin prophylaxis post repair, but was

noncompliant. In third trimester of present pregnancy, she was unable do her household activities and dyspnoea progressed from NYHA2 to 4. On examination she was propped up with two pillows, had facial puffiness, bilateral pitting pedal edema grade 2.

Clinical diagnosis: Atrial fibrillation in pregnancy
Differential diagnosis: Atrial flutter, atrial tachycardia, Paroxysmal supraventricular tachycardia, WPW syndrome.
Pathological discussion: She received oral Metoprolol 25mg BD, Digoxin 0.25mg OD, MGSO4 2mg IV, oxygen support in ICU. Inj Heparin 5000 s/c TID administered as she had CHADS-VASc score 2. LSCS was planned for obstetric reasons, informed written high risk consent taken after explaining risk of intra operative arrhythmia and pulmonary edema, maternal and fetal mortality and morbidity. (Preoperative findings as mentioned in table 1). LSCS under GA with RSI with IPPV was planned as deranged coagulation with LV systolic dysfunction ruled out epidural anaesthesia as an option.

Table 1
Preoperative findings

PREOPERATIVE FINDINGS	
VITALS	PR:92/min, irregularly irregular, BP: 90/60mmhg, SPO2 93% on room air.
SYSTEMIC EXAMINATION	CVS: loud P2, loud S1 in mitral area, systolic murmur grade 3 best heard in mitral area. RS: bilateral basal crepitations.
BLOOD INVESTIGATIONS	Deranged coagulation, hypomagnesemia
ECG	Atrial flutter reverted to atrial fibrillation with stat IV. Digoxin 0.25mg and IV metoprolol 2.5mg.
2D ECHO	RHD S/P mitral valve repair and TV plasty, severe MR Dilated LA(5.5cm), AML doming with PML mobility restricted, MVOA 2CM2, systolic dysfunction, EF 45%, PA pressures 40mmhg, no visible clots.



Preoperative ECG

Figure 1. Preoperative ECG

Anaesthesia machine and equipements checked, GA and emergency drugs loaded, defibrillator kept ready. Patient propped up in OT, received O2 by face mask at 12 L/min. Wedge placed to prevent aorto caval compression. Standard ASA monitors attached. On table TTE showed dilated LV, severe MR and LV systolic dysfunction. 18G IV cannula secured, RL started as maintenance. Baseline vitals: irregular HR 112/min, BP 106/60mmHg, respiratory rate 32/min, Spo2 100% with 12l/min O2. Right radial artery cannulated. 7FR triple lumen 16cm central venous catheter inserted. Pediatrician informed about planned use of opiod, GA was induced with IV Fenatanyl 2mcg/kg, Etomidate 0.2mg/kg, titrated Sevoflurane and RSI with Succinylcholine 2mg/kg. Post intubation vitals were stable. Delivered Healthy baby with APGAR 8 at 1min, within 5 minutes of abdominal incision. Oxytocin 5U slow IV, 10units added as infusion over 1 hour. Anaesthesia maintained with oxygen 50%, air, Sevoflurane titrated and Atracurium 0.1mg/kg boluses. Morphine titrated upto 0.1mg/kg for analgesia. Patient developed AF with fast ventricular rate postdelivery, Inj.lignocaine 2mg/kg IV, Inj. Amiodarone 150mg stat and infusion administered. TTE showed poorly contracting LV during fast ventricular rate (figure 2). Rest of the surgery was uneventful. Cardioversion was deferred due to dilated LA. After spontaneous respiratory efforts patients reversed and extubated with HR 114/m, BP 104/54mmhg, spo2 96% and shifted to ICU for observation. She received supportive care. Postoperative TTE: dilated LV, severe MR and LV systolic dysfunction. On POD 4 she was started on oral Warfarin 3mg shifted to Ward, discharged on POD5 with antibiotics, Warfarin 5mg OD, Digoxin 0.25mg OD, Frusemide 40mg OD and Metoprolol XL 12.5 mg BD. Need for mitral valve replacement was explained.

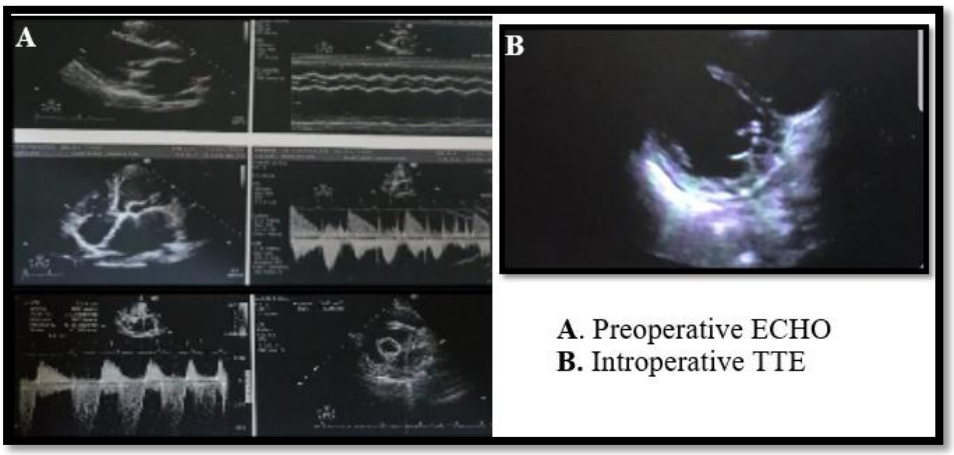


Figure 2: Preoperative ECHO and Intraoperative TTE

Discussion and management

Diagnosis of AF is done by clinical examination and ECG. Treatment of AF in pregnancy improves maternal and fetal outcomes but results in embryopathy in first trimester and potentially affects fetal growth, fetal arrhythmias in other trimesters. Management of AF is divided into management of acute onset AF, control of ventricular rate maintenance of NSR and prevention of

thromboembolism, rate control being the initial goal. Digoxin, beta blockers and calcium channel blockers are used for rate control, increasing diastolic filling time. CCB are not recommended in AF with preexcitation syndrome as they paradoxically accelerate ventricular rate. Digoxin is safe due to non-teratogenicity but altered serum levels can occur in third trimester[3]. Cardio selective beta-blockers are preferred due to lower incidence of beta 2 mediated peripheral vasodilatation, uterine relaxation and fetal hypoglycemia. Pharmacological cardioversion with IV Flecanide and Ibutilide indicated for persistant AF with no structural heart disease. Electrical cardioversion is safe during all stages of pregnancy and recommended in AF risking maternal and fetal life, hemodynamically unstable AF, pre-excited AF. Transient fetal dysarrythmia has to be monitored. DC requires prior anticoagulation for 3weeks if AF lasts more than 48hrs or duration was unknown and to be continued 4weeks post cardioversion. The risk of embolization in nonanticoagulated patients is 5% whereas 1% in anticoagulated. Electrical or pharmacological cardioversion can be done after 24hours if the heart rate is controlled and hemodynamically stable.

Anticoagulation is mandatory except in lone AF or low thromboembolic risk and chosen according to stage of pregnancy. Heparin is preferred in first and last trimester as it does not cross placenta. Compared to UFH, LMWH carries better safety profile and does not require frequent APTT monitoring4.In pregnancy higher dose of anticoagulant with more frequent administration is required to maintain therapeutic level due to increased plasma volume, glomerular filtration and placenta degradation. Vitamin K antagonists are considered in second trimester to 1month before expected delivery. Resumption of anticoagulants to be postponed 24hours post LSCS to reduce bleeding.

The clinical and echocardiographic predictors of TE are recent history of TE, increased atrial size, LAA thrombus or sludge, severe spontaneous ECHO contrast, LAA emptying velocity less than 20, LVS dysfunction and complex aortic atheroma.TTE has low sensitivity in detecting LA thrombus compared to TEE. LA size predicts probability of successful cardioversion. Framingham study demonstrated 39% increased risk AF with 5mm increase in LA dimension. Psaty found 4times risk of developing AF with LA size more than 5cm. Diastolic dysfunction increases risk of AF and heart failure5.

Induction with Etomidate preserves hemodynamic stability by maintaining sympathetic outflow and autonomic reflex. Opioid induction can cause the fetal respiratory depression and aspiration in parturient. Stress response has to be minimized intra operatively due to resultant pulmonary edema. Application of PPV can decrease preload causing hypotension and cardiovascular collapse. CSE has an advantage of immediate block, enabling to start urgent surgery and post op analgesia, in our case it was not possible due to deranged coagulation.

Onset of AF intra operatively can be due to Acid base and electrolyte abnormality, hypertension, hypothermia, tachycardia, hypovolemia, hypoxia, hypecarbia, central venous catheters malposition, vagolytic effect of anaesthetic agents[6]. Correction and identification of precipitating factors lead to spontaneous conversion to sinus rhythm Sensitization of myocardium to catecholamines by Halothane can cause AF but others volatile anesthetics have antifibrillatory

effect[7,8]. AF results in loss of atrial component of diastole, rapid ventricular rate, thromboembolism and patient discomfort due to palpitations. Loss of atrial contraction decreases cardiac output and drop in BP upto 50%. LV dysfunction depends more on atrial systole although atrial kick has a small impact on cardiac output when LVEDP is high[9], Ventricular rate has to be controlled to increase the diastolic filling time, although FVR helps in compensating loss of effective atrial contraction. The return of atrial electrical activity is faster with AF of short duration.

Intra operatively FVR with AF is managed with intravenous verapamil and esmolol due to negative chronotropic action but esmolol allows easy manipulation of plasma level due to short plasma level. Amiodarone, a class 3 antiarrhythmic with an advantage of causing chemical cardioversion by prolonging action potential and atrial refractory period. But when given with GA can cause cardiac depression, heart failure, QT prolongation, bradyarrhythmias and hypotension that respond to volume expansion and titration[10].Our patient received amiodarone bolus dose and infusion and did not require any vasopressor therapy intra or post operatively.

Final diagnosis
Atrial fibrillation with fast ventricular rate in pregnancy.

Conclusion

AF during pregnancy warrants immediate attention. Management of AF requires use of antiarrhythmic agents, anticoagulants and cardioversion. Pre-operative optimization, knowledge of precipitating factors, prompt intervention, with intensive monitoring, and careful hemodynamic management by anaesthesiologist improves outcome. The role TTE in assessing prognosis is of value.

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