WPW Syndrome: Intricacies of Anesthetic Management in Cesarean Delivery

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Abstract

The Wolff-Parkinson-White (WPW) syndrome is one of the major causes of paroxysmal supraventricular tachycardia during pregnancy. Both incidence and symptoms of PSVT are exacerbated during pregnancy and may significantly affect maternal and fetal outcomes during caesarean delivery. We outline the anesthetic management of a patient with WPW syndrome undergoing caesarean delivery.

INTRODUCTION

The Wolff-Parkinson-White (WPW) syndrome is an inherited disorder occurring in 0.9-3% of the general population [1]. These patients have an anomalous conducting pathway, --- The bundle of Kent--- which allows supraventricular impulses to bypass the atrio-ventricular (AV) node, bundle of His and distal conducting system, and such, to pre-excite the ventricles [2]. Because of dual conduction between the atria and the ventricles, patients are likely to develop paroxysmal supraventricular tachycardia (PSVT) and atrial fibrillation (AF).

Physiologic changes in pregnancy can unmask or worsen preexisting arrhythmias [3]. Anesthetic agents and anti-arrhythmic agents also alter cardiac conduction. A pregnant patient with preexisting WPW syndrome requiring surgery is thus particularly predisposed to the development of paroxysmal supraventricular tachycardia (PSVT), atrial fibrillation (AF) and in turn to ventricular fibrillation (VF). Awareness, detection and management of these arrhythmias is thus the main focus of the anesthetic management of such patients [3-5].

We present the anesthetic aspects of the management of a diagnosed, symptomatic case of WPW syndrome who underwent an elective cesarean section.

CASE PRESENTATION

A 29-year old housewife of 37 week gestation, with a BMI of 23 (height 1.55m and a full term weight of 55-kg), presented, in her second pregnancy, with episodic palpitations and breathlessness for 2 years. The episodes lasted from twenty minutes to several hours. During one such episode, a diagnosis of SVT was made and terminated with Inj. Adenosine 6mg IV. Further cardiological evaluation revealed WPW syndrome with an antero-septal conducting pathway to be the cause of the SVT. The episodes became more frequent 3 months into her current pregnancy. She underwent a lower segment cesarean section under spinal anaesthesia for the delivery of her first child, 3 years ago, during which period the patient was asymptomatic and surgery was uneventful.

At admission for Cesarean Section she was asymptomatic with good exercise tolerance and on Metoprolol 50 mg PO for the last 6 months. The general and systemic examination revealed a pulse rate (PR) of 70 beats per minute, a blood pressure (BP) of 125/80 mm Hg. The electrocardiograph (ECG) showed normal sinus rhythm, a heart rate of 80/minute, shortened P-R interval (<0.12 seconds), a widened initial QRS complex with slurred upstroke and a normal terminal QRS deflection (Figure 1). Chest X-Ray and laboratory investigations including a haemogram, liver function tests, renal function tests, serum electrolytes and coagulation profiles were normal. 2D- Echocardiogram revealed normal valvular and ventricular functions with an ejection fraction of 66%. The patient was counseled and consented for surgery. Tab Metoprolol OD and tab ranitidine 150mg were continue on the night before, and the morning of surgery.

Anaesthesia technique

An 8-hour NPO status was confirmed. Surgery under epidural anaesthesia was planned. Intravenous access was secured and the patient preloaded with Ringer lactate 500 ml at the rate of 10 ml/min. Availability of Anti-arrhythmic agents inj. Adenosine, inj. Esmolol, inj. Procainamide, inj. Amiodarone, Lignocaine 2% and a defibrillator was confirmed. Pulse oximetry (SpO2), ECG monitoring (lead II) and noninvasive blood pressure (NIBP) were monitored. ECG showed normal sinus rhythm, a short PR interval, delta wave and widened QRS complex with the heart rate of 78/min and BP of 110/80 mm of Hg.
Epidural Anaesthesia was obtained with an 18G epidural catheter, inserted to 8 cm in the L3-L4 intervertebral space. After a test dose of 3 cc Lignocaine (2%), Bupivacaine (0.5%) was administered epidurally in incremental doses over a period of 20 minutes to achieve a sensory level of T6. The patient was positioned supine with a wedge under the right hip. Oxygen was administered via Hudson’s mask at 6 liters/min. The vitals at this stage were NIBP-118/72, PR-78 /min, SpO2-100%. At 15 minutes, an episode of hypotension and concomitant bradycardia occurred, with NIBP 65/40 mmHg and PR-48/min. The level of sensory block at this stage was T6. It was treated with a bolus of phenylephrine 50mic, atropine 1 mg and 200 ml hetastarch (0.4%). 5 minutes later the NIBP was 104/70 and PR 64 bpm. After delivery of a healthy 2.5 kg male child, oxytocin was administered (5 units in 100 ml saline over 10 minutes). The immediate postoperative vitals were within normal limits. After the operation, lasting 60 minutes, the patient was observed in the ICU for a period of 24 hours. Postoperative analgesia was maintained with 50 mics fentanyl 6 hourly and tramadol 100mg IV, on patient demand (given twice over a period of 48 hours). The patient was discharged a week later.

DISCUSSION

Cardiac arrhythmias are noted with greater frequency during pregnancy. Gleicher et al, in a report of three pregnant patients, suggested pregnancy predisposed asymptomatic patients with pre-excitation to tachyarrhythmia’s. A retrospective study of 60 patients with documented SVTs, showed that pregnancy is associated with both an increased risk, and an exacerbation of symptoms of SVTs [6]. The high Plasma catecholamine concentrations, increased adrenergic receptor sensitivity and high end diastolic volumes associated with pregnancy, all heighten the predisposition to arrhythmias [7]. Pre-operative history plays an important role in identifying patients of WPW syndrome, since, even on ECG, WPW features may not be readily apparent [8].

The pregnant state overlaid with WPW syndrome is thus a high risk situation for the occurrence of arrhythmias. Other factors that trigger arrhythmias e.g. heightened sympathetic activity caused by pain, anxiety, the stress of intubation and/or hypovolaemia should therefore be avoided. Antiarrhythmic drugs are best avoided as most cross the placenta. Epidural anaesthesia avoids many factors that trigger arrhythmias e.g. administration of multiple drugs and intubation and was hence preferred to General Anaesthesia here. Moreover, epidural anaesthesia allows tighter segmental block control, increases hemodynamic stability and provides a route for post-operative analgesia, and may be preferred over spinal anesthesia for these reasons [9]. High sympathetic blockade and resulting increased parasympathetic activity, associated with either neuraxial procedure, can lead to preferential conduction via the accessory pathway. The blockade level therefore, bears close monitoring [8]. Regional anaesthesia can lead to decreased atrial filling and therefore increased arrythmicity [3]. The fluid preload and left tilt to avoid aorto-caval compression, were used in this case to avoid reduced atrial fill. Despite proper positioning and preloading our patient underwent an episode of severe bradycardia and hypotension shortly after induction. It was treated with a bolus dose of Phenylephrine 50 mcg, and Atropine 1mg with a 200 ml colloid bolus. The use of anti-muscarinic agents in the treatment of bradycardia has been reported to result in SVTs because of their sympathomimetic activity [10]. Conversely, Atropine can terminate ventricular pre-excitation and normalizes AV conduction as evidenced by the disappearance of the delta
waves on ECG. Atropine administration should therefore be individualized [4]. Phenylephrine may be used to treat hypotension during regional anaesthesia. It increases vagal tone by indirectly stimulating baroreceptor reflexes and therefore reduces the incidence of SVTs and successfully terminated existing SVT in a patient of WPWS [3].

Although no arrhythmias occurred in the perioperative period, our patient in a previous episode, had suffered an Atrioventricular reentrant tachycardia with a heart rate of 200, invisible P-waves, a narrow QRS complex with normal T-waves, indicating orthodromic conduction. This was terminated with Adenosine administered as a bolus. (Figures 2a,2b) Non-pharmacological treatments (carotid massage, Valsalva maneuver) should always be tried before drugs, as they are efficacious, may aid in diagnosis and avoid the trans-placental effect of drugs [11]. Because of its short half-life and efficacy, adenosine is ideal in the treatment of PSVTs in pregnancy. However, its administration may lead to a transient fetal bradycardia [3].

Non-selective beta blockers have the theoretical disadvantage of causing vasodilatation and uterine relaxation hence selective beta-blockers are preferred. Verapamil and diltiazem, share the disadvantages of the non-selective beta blockers [11]. As oxytocin may cause SVT, its use should be limited to 5 units IV or as an infusion [3].

Atrial fibrillation with rapid conduction over the aberrant pathway, may lead to ventricular fibrillation hence requires urgent treatment with cardioversion using 150 to 200 joules in hemodynamically unstable patients. In stable patients drugs enhancing the refractoriness of the abnormal pathway e.g. procaainamide and propranolol may be used. Digoxin and verapamil are contraindicated [12].

With a success rate approaching 95% and complication rates as low as 4%, radio-frequency catheter ablation of the accessory pathway remains the mainstay treatment of symptomatic WPW syndrome [8]. Since this procedure is commonly performed under fluoroscopic guidance, due to concerns of ill-effects of fetal radiation exposure, this therapy may be deferred till after termination of pregnancy. It is also recommended for symptomatic patients of WPW syndrome, including pregnant patients, not responding to medical therapy. Imaging techniques to limit maternal and fetal radiation exposure like intracardiac echocardiography and electro anatomic mapping systems gain more importance in this setting [13].

**CONCLUSION**

Both pregnancy and the WPWS aggravate each other’s tendency to produce arrhythmias. Adequate preparation with fluid preloading and positioning, securing appropriate drugs and defibrillator, avoidance of tachyarrhythmia, and their prompt treatment on occurrence is required for the safety of both the mother and the fetus. Atropine and/or phenylephrine are useful for intraoperative hypotension. Since most antiarrhythmic drugs cross the placenta, non-pharmacological treatment of these should be initially attempted.

**REFERENCES**