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Case Series

Perioperative management of Wolff–Parkinson-White syndrome – A case series

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ABSTRACT

Wolff-Parkinson-White (WPW) syndrome is a type of pre-excitation syndrome**,** where part of cardiac ventricles are activated too early by virtue of an accessory pathway, that directly attaches atria and ventricles, thereby allowing electrical activity to bypass AV node, thus leading to episodes of tachycardia which can be either asymptomatic or may present with dyspnoea on exertion or with palpitation. As it's a rare condition, we report 3 cases of WPW Syndrome which adds more evidence to the current literature. The anaesthetic management can be challenging and has to be customized to the nature of the surgery and the patient to avoid life-threatening tachyarrhythmias.

Key Messages: Perioperative management of WPW syndrome is challenging and requires good pre-operative planning and adequate preparation for intraoperative and post-operative period.

1. Introduction

WPW syndrome seems to be an electrophysiological heart condition that was first described in 1930 by Louis Wolff, Sir John Parkinson, and Paul Dudley White from 11 individuals who suffered from tachycardia bouts linked to sinus rhythm with a short PR interval on ECG.[1] The occurrence of WPW pattern upon surface ECG is estimated to be 0.13 to 0.25% among overall population, with a prevalence of up to 0.55 percent among first degree familial relatives reckoning a familial component.[2,3] The prevalence of WPW syndrome is even lower than WPW pattern, with values varying between the studies. Most people experience no symptoms, while others may experience chest pain, palpitations, syncopal episodes, light headedness, or even sudden cardiac death.[1]

2. Case 1

A 47 years old lady was diagnosed with Cystadenoma ovary right side with bilateral hydroureteronephrosis, Acute on Chronic kidney disease, and Systemic Hypertension**.** New York Heart Association (NYHA) grade 1, and Metabolic equivalents of 4, Canadian Cardiovascular Society Class 1.

Clinically patient did not give any history of palpitation and syncope. On examination: moderately built and well-nourished with BMI of 23 kg/m^2, PR of 98/minute, and Grade 1 hypertension on a beta-blocker. Airway examination revealed Mallampati score of class I, adequate mouth opening, thyromental distance of 7 cm, and sternomental distance of 14 cm. Patient was anaemic with Hb of 10.1 g%, with Renal Function Tests (RFT) revealing elevated creatinine level of 1.9 mg/dl. Pre-operative 12 lead ECG revealing WPW syndrome type B is illustrated in Figure 1. ECHO revealed a 62% ejection fraction and grade 1 LV diastolic dysfunction. To eliminate out covid 19, a HRCT chest as well as an RT PCR throat swab were done. The patient was given 40 mg of pantoprazole and 0.25 mg of

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alprazolam, on the night before surgery, and was fasted for 8 hours. The patient was scheduled for staging laparotomy with bilateral Double "J" Stent (DJS) under epidural and general anaesthesia.

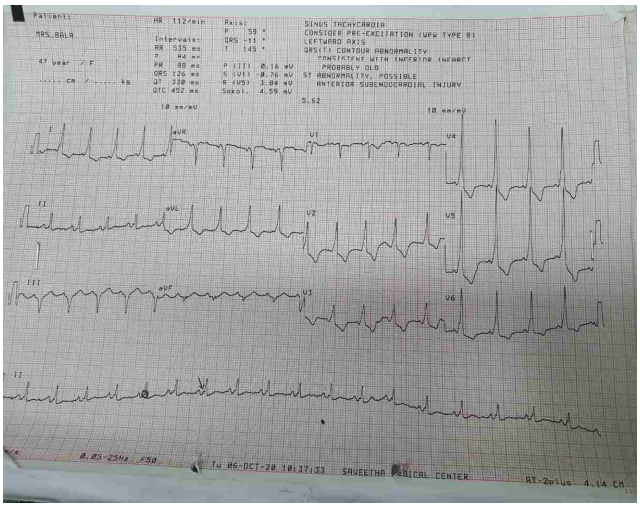

Fig. 1: Pre-operative ECG revealing WPW syndrome type B

Adenosine, xylocard, amiodarone, esmolol, and a defibrillator were kept ready in the OT. Two 16 gauge cannulas were used to secure venous access. Patient's left radial artery was cannulated for monitoring intraoperative blood pressure. In the sitting position, an 18G tuohy needle was inserted at the L1-2 level for epidural. After the epidural was placed, the patient was given 100 mcg fentanyl, 100 mg propofol, and 8 mg cisatracurium. One milligram of metoprolol was given intravenously to blunt the intubation response. Patient was intubated with a 7.0 mm size endotracheal tube using Macintosh laryngoscope. The plane of anaesthesia was maintained with isoflurane at a Minimum Alveolar Concentration (MAC) of 1.5 and 50% each of N_2O and O_2.

Before incision, the epidural was activated with a bolus of 8 ml of 0.25% levobupivacaine, followed by 5 ml/hour infusion. Intraoperative ECG is illustrated as in Figure 2. The patient was extubated with adequate reversal using a TOF monitor. Intraoperatively, sinus rhythm was maintained. Prophylaxis against PONV was taken intraoperatively, and the patient was sent to ICU for observation. The patient was shifted to the ward the next day.

3. Case 2

A 10-year old male child of weight 25 kg came with breathlessness and fatigue on and off for the past 5 years, NYHA class II. History of cyanosis present. On Examination, patient was moderately built, pulse rate of 96/minute, blood pressure measured non-invasively was 100/60 mm hg. Cardiovascular system examination showed

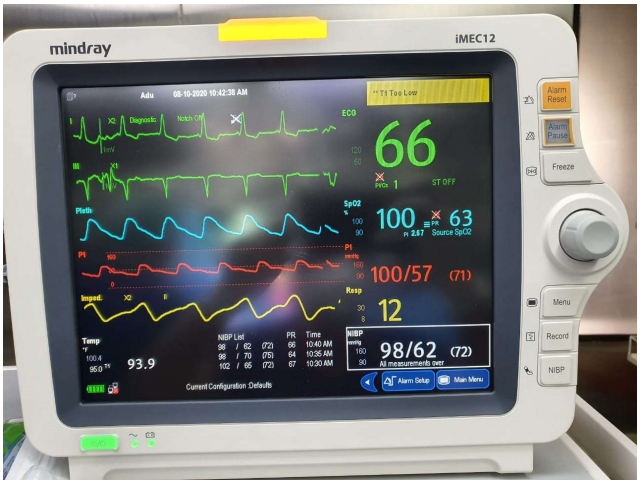

Fig. 2: Intra operative ECG

a systolic murmur of 3/6 intensity heard better over the tricuspid area. Airway – Mallampati I, thyromental and interincisor distance were 6.5 cm and 3.5 cm respectively.

Electrocardiogram showed a rate of 100/minute, one ventricular ectopic beat present in rhythm strip, P wave tall and wide in V1, 1st degree heart block, bizarre QRS complex, RBBB, secondary ST-T changes as illustrated in Figure 3. Chest x-ray showed cardiomegaly.

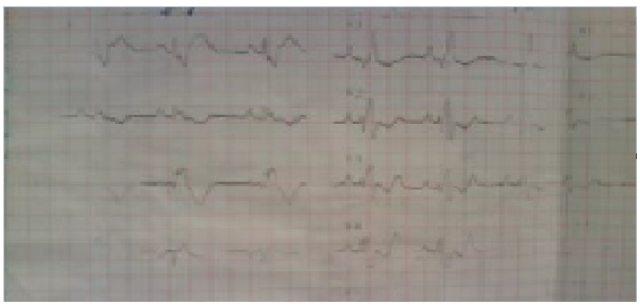

Fig. 3: ECG

Cardiologist evaluation was done and was confirmed as Ebstein's anomaly. 2D ECHO showed anterior Tricuspid Valve (TV) leaflet large and redundant, septal leaflet tethered to annulus –17 mm, moderate tricuspid regurgitation, ASD[OS]. Child was taken up for the repair of Ebstein's anomaly under ASA III. General anaesthesia was planned.

Pre-op spo2 – 91%

Pre-op ABG – PH-7.39, Paco2- 32,

PaO2 –52, Sao2 – 86.9%, Hco3 – 19.4

Child was premedicated with tablet diazepam 5mg the night before surgery. Intravenous access secured, intravenous glycopyrrolate of 0.1 mg given as anti-sialagogue, sedated with midazolam 1.5 mg IV. Induction done with fentanyl 80 mcg, thiopentone 125 mg and

vecuronium 2 mg, intubation was done with 6.0 mm sized cuffed portex endotracheal tube and confirmed with end tidal carbon dioxide. Patient maintained with O2:N2O –50%:50% and 1.5% isoflurane, Left radial artery cannulation and right femoral vein catheterization was done. Intraoperative ECG as shown in Figure 4.

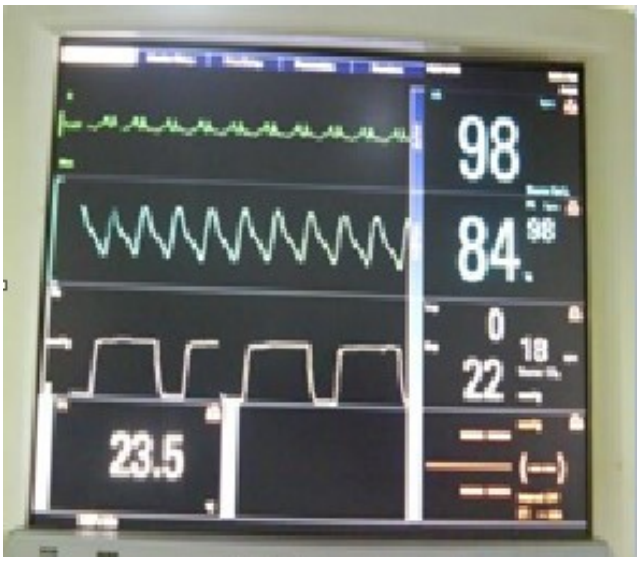

Fig. 4: Intraoperative ECG

Heparin at a dose of 300 IU/kg was given. Under cardiopulmonary bypass, tricuspid valve repair and atrial septal defect closure were done. Aortic cross clamp time-45 minutes, on cardiopulmonary bypass (CPB) for 1.5 hours. Patient weaned from cardiopulmonary bypass with inotropic support of dopamine of 5 mcg/kg/min and nitroglycerine 1 mcg/kg/min and heparin reversed with protamine at 1:1 dose. Intraoperative arrhythmias encountered were junctional rhythm, right bundle branch block, mobitz type 1 block, pre-excitation rhythm. Supraventricular arrhythmias treated with amiodarone infusion 1 mg/min. Pacemaker was kept on standby.

Post CPB ABG- PH-7.35, PaCO2-33, PO2-463, SaO2-99.9%, HCO3-18.

Activated clotting time was 102 seconds. Patient shifted to intensive care unit for elective ventilation. Child was extubated the next day morning uneventfully and weaned off from dopamine the 2nd day.

4. Case 3

A 27-year-old lady, semi-skilled labourer by profession, with no known co morbidities presented with complaints of mass in front of the neck, diagnosed as multinodular goiter (MNG), histopathologically follicular adenoma, was posted for subtotal thyroidectomy. She gave history of frequent episodes of light headedness and palpitation while working, which got better on taking rest. She was found to be moderately built and nourished, with a Body Mass Index of 25 kg/m^2, general and systemic examinations were found to be normal. Airway examination revealed Mallampati score of class II, adequate mouth opening, thyromental distance of 7 cm and sternomental distance of 14 cm. Her 12 lead ECG revealed WPW Syndrome with shortened PR interval and slurring of upstroke of QRS complex. 2D echo revealed no valvular abnormalities, normal diastolic and systolic functions. Patient was fasted for 8 hours prior to surgery, and premedicated with pantoprazole and alprazolam. Drugs kept ready in OT were adenosine, xylocard, amiodarone, esmolol and defibrillator on standby. Patient was shifted to the operating room. Apart from the standard ASA monitoring, arterial blood gas analysis, invasive blood pressure recording and input output fluid charting were done. Left radial artery was cannulated.

Superficial Cervical Plexus block was given bilaterally with 20 ml of 0.2% ropivacaine, mixed with 4 mg dexamethasone. 2 mcg/kg of fentanyl and 1.5mg/kg of lignocaine were given intravenously to attenuate the intubation response. The dose of propofol was titrated, 6 mg of vecuronium was given. Airway secured with 7.0 mm size endotracheal tube using Macintosh Laryngoscope. Was maintained on isoflurane at a MAC of 1.5% with O_2 and N_2O at 50% each.

One episode of PSVT was treated with 6 mg of adenosine and 2 mg of verapamil. Intraoperative ECG as illustrated in Figure 5 and Figure 6 show delta wave and paroxysmal supraventricular tachycardia.

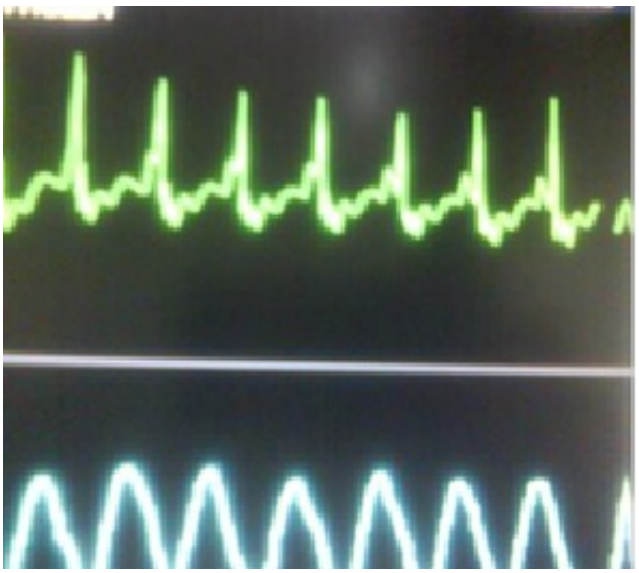

Fig. 5:

Patient was extubated with adequate reversal using TOF monitor. Prophylaxis against PONV was given intraoperatively and patient was sent to ICU for monitoring of vitals. Patient was shifted to the ward next day.

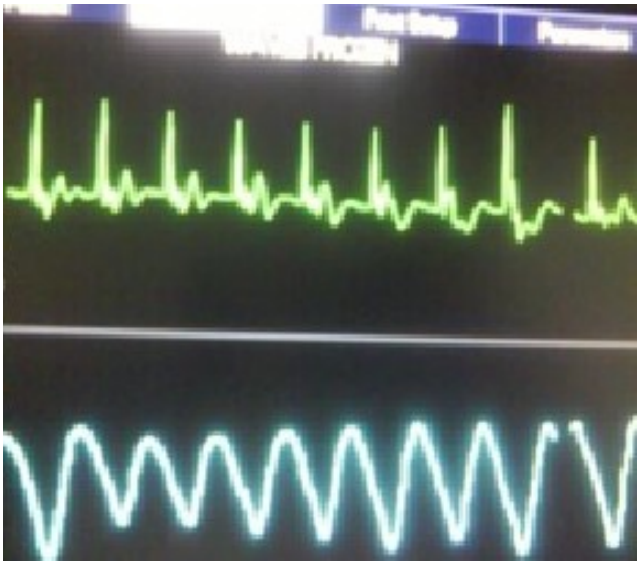

Fig. 6:

5. Discussion

Patients with WPW have an atrioventricular accessory pathway known as the "Bundle of Kent"; bypassing the AV node resulting in a shorter PR interval(less than 0.12 seconds).[4] The QRS complex is formed by the fusion of preexcitation and later ventricular activation due to transmission through the AV node and infranodal conduction system to the ventricles. It starts earlier than expected, causing the initial part of ventricular activation to be slowed, resulting in upstroking of the QRS complex forming Delta waves. The quantity of the myocardium depolarized increases as conduction through the Bundle of Kent accelerates, resulting in a pronounced Delta wave and a wider QRS complex.

The tissue in the accessory pathway is congenital. Atrioventricular accessory routes if related to congenital heart illness are more likely to be located on the right side than on the left.[4] Ebstein's abnormality is the congenital lesion most strongly linked to WPW syndrome, with 10 to 20% of individuals having one or more accessory pathways.[5] There has also been a link between the left-sided accessory route and Mitral valve prolapse.[6]

WPW syndrome is diagnosed by surface ECG, characterized by a short PR interval, delta waves, and a widened QRS complex that help in distinguishing from other pre-excitation syndromes such as Lown-Ganong-Levine syndrome and Mahaim-type.[4] WPW syndrome is further divided into the following categories: Type A: right bundle branch block with right ventricular hypertrophy and myocardial infarction in the posterior chamber. Type B is similar to the left bundle branch block with hypertrophy of the left ventricle. Electrophysiology testing is utilized not only to confirm the diagnosis but also to risk-stratify them. During the same session, it is frequently combined with mapping and transcatheter ablation of the bundle of kent.

The type of anaesthesia and the anaesthetic agents used affect the physiology of AV conduction and may result in cardiac arrhythmia. Propofol is preferred for general anaesthesia since it does not impact the accessory pathway's refractory period.[7] Drugs causing tachycardia like atropine, ketamine should be avoided. Isoflurane and sevoflurane maintain optimal hemodynamic stability while having no effect on AV node conduction. Fentanyl is used to reduce the intubation response. Vecuronium and rocuronium provide higher cardiovascular stability than atracurium and pancuronium, hence they are preferred.[7]

5.1. Arrythmia Associated with WPW Syndrome

Atrioventricular re-entrant (or reciprocating) tachycardia (AVRT) is divided into two types: orthodromic and antidromic. Orthodromic AVRT is characterized by the existence of narrow QRS complexes, as opposed to large QRS complexes in Antidromic AVRT. In PSVT, vagal manoeuvres are first attempted. In hemodynamically stable patients, class 1 anti-arrhythmic medications such as lignocaine, adenosine (6 to 12 mg), or beta-blockers such as esmolol (50-300 mcg/kg/min IV) are administered.[7]

Atrial fibrillation (AF) accounts for 10 to 30% of cases with WPW syndrome, with AVRT preceding one-third of cases of Atrial fibrillation.[8,9] Patients with stable hemodynamics should be treated pharmacologically. In contrast, patients with unstable hemodynamics should be treated with cardioversion of 50-100 J.[7] Atrial flutter when it is transmitted through the accessory pathway can result in Ventricular fibrillation(VF).

6. Conclusion

Owing to limited literature available about the perioperative management of WPW syndrome, this case series gives insights about the successful management of patients with WPW syndrome posted for cardiac and non-cardiac surgeries.

7. Source of Funding

None.

8. Conflict of Interest

The authors declare no conflict of interest.

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