Jurnal Anestesi Perioperatif
[**JAP**. 2020;8(3): 206–13]

LAPORAN KASUS

Laporan Kasus: Manajemen Pasien Sindrom Wolff-Parkinson-White (WPW) yang Menjalani Tindakan Pembedahan

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Abstract

Wolff-Parkinson-White (WPW) syndrome is a commonly undiagnosed cardiac rhythm anomaly in a previously healthy patient who may precipitate malignant arrhythmia under surgical stress. We report successful management of a reconstruction surgery patient who developed cardiac arrest under general anesthesia due to undiagnosed WPW syndrome and a malignant arrhythmia during subsequent emergency surgery. A male patient with no previous history of the co-existing disease, age 23 years old underwent 14 hours of leg reconstruction with a posterior back flap. At the end of the surgery, the patient developed malignant arrhythmia that worsens to pulseless ventricular tachycardia. High-quality resuscitation was conducted and resulted in the return of spontaneous circulation. The patient had to undergo emergency surgery the next day, and another episode of intraoperative malignant arrhythmia was treated with propafenone and diltiazem. The patient underwent ablation postoperatively and, on the 14th day, was discharged without any residual complications. In conclusion, WPW may appear asymptomatic in a healthy young patient. Good anesthesia management and monitoring, knowledge of selective antiarrhythmic drugs and high-quality resuscitation skills can provide an optimal outcome in an unpredicted intraoperative crisis.

Keywords: Arrhythmia, case report, sudden cardiac arrest, tachycardia, Wolff-Parkinson-White syndrome

Intervention in Undergoing Surgery with Undiagnosed Wolff-Parkinson-White Syndrome: Case Report

Abstrak

Sindrom Wolff-Parkinson-White (WPW) adalah kelainan irama jantung yang sering tidak terdiagnosis pada pasien usia muda dan sering tanpa gejala. Stres akibat pembedahan dapat memicu aritmia maligna pada kelainan ini. Kami melaporkan keberhasilan manajemen pada pasien bedah plastik rekonstruksi dalam anestesi umum yang mengalami henti jantung karena sindrom WPW yang tidak terdiagnosis sebelumnya dan terjadi aritmia maligna serupa pada operasi darurat berikutnya. Seorang laki-laki berusia 23 tahun tanpa riwayat penyakit penyerta sebelumnya, menjalani rekonstruksi kaki dengan flap posterior selama 14 jam. Pada akhir pembedahan, pasien mengalami aritimia maligna dan berkembang menjadi ventrikular takikardia tanpa nadi. Tindakan resusitasi jantung paru berkualitas tinggi dilakukan dan sirkulasi spontan kembali muncul. Pasien harus menjalani operasi darurat keesokan harinya dan mengalami episode aritmia maligna ulangan intraoperatif yang telah berhasil diidentifikasi pada pemeriksaan pascaoperasi pertama sebagai sindrom Wolff-Parkinson-White. Aritmia maligna tersebut diatasi dengan pemberian propafenon dan diltiazem. Pasien menjalani tindakan ablasi pascaoperasi dan pulang pada hari keempat belas perawatan tanpa gejala sisa. Simpulan, sindrom WPW mungkin tampak asimtomatik pada pasien muda yang sehat. Manajemen anestesi yang baik, pengetahuan mengenai profil berbagai obat antiaritmia serta pelaksanaan tindakan resusitasi berkualitas tinggi dapat memberikan hasil keluaran yang optimal bila terjadi krisis intraoperatif yang tidak diperkirakan sebelumnya.

Kata Kunci: Aritmia, henti jantung mendadak, laporan kasus, takikardia, sindrom Wolff-Parkinson-White

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p-ISSN 2337-7909; e-ISSN 2338-8463; http://doi.org/10.15851/jap.v8n3.2193

Introduction

The National Organization for Rare Disorders (NORD)[1] stated Wolff-Parkinson-White (WPW) syndrome is a congenital condition involving abnormal conductive cardiac tissues between the atria and ventricles that provides a pathway for a re-entrant tachycardia circuit with manifest tachyarrhythmia.[2] Besides, Bengali et al.[3] stated that the WPW pattern occurs in 0.13–0.25% of the general population, with the first presentation at 20–40 years old. Epidemiological data from the United States (US) shows 481,000–925,000 of the 370 million US population has a WPW pattern.

Since WPW pattern is an electrocardiography (ECG) diagnosis and ECG is not routinely done by the general public, especially at a young age, the findings of WPW will mostly be accidental or during a catastrophic medical complication, usually during surgery. Even more, most of them are asymptomatic, although some may develop palpitations, angina, loss of consciousness, and sudden death in 0.4% of patients. The WPW patient may also develop life-threatening tachycardia and cardiac arrest during surgery, especially if it was not diagnosed in the preoperative period.[1–3] We present the management of a plastic reconstruction patient with an undiagnosed WPW syndrome.

Case Report

A 23-year-old, 54 kg male patient was presented with a popliteal defect and diffuse contracture raw surface on his right leg due to a motor vehicle injury two months earlier. He was scheduled for elective contracture release and resurfacing with musculocutaneous latissimus dorsi free flap as a surrogate for deformity. The patient had previously two general anaesthesia for wound necrotomy and had no remarkable disease history upon anamnesis. He was active and was working a day job without any so-called limitations, but the activities he claimed were limited after the accident due to his contractures.

Laboratory examination showed hemoglobin of 13.4 g/dL and a normal coagulation profile with no electrolyte disturbance. An ECG was provided in the preoperative period, although this was uncommon practice for a healthy young patient in our facility, never the less the ECG was within normal limits (Figure 1). The patient was classified as an American Society of Anaesthesiologists (ASA) class 1. He was then asked to perform preoperative fasting as the guideline stated and had an intravenous line already placed on his hand for preoperative antibiotic and pain medicine route.

Upon arrival in the operating theatre, the patient was alert and comfortable. He was not premedicated and was taken directly to the operative room, where standard monitoring was attached. His heart rate was 60 beats per minute, blood pressure at 110/70 mmHg, saturation 99% on room air, and ECG showed sinus rhythm.

The patient was pre oxygenated with 100% oxygen and induced intravenously with 100 micrograms of fentanyl, 100 mg of propofol, and 25 mg atracurium. He was then intubated with a 7.5 enforced endotracheal tube and connected to the anaesthesia machine. Anaesthesia was maintained using isoflurane 1–1.5% in a 50% oxygen and air mixture, and the ventilator was set in a volume control fashioned with 400 cc tidal volume with a rate of 12 times per minute.

Due to the surgical area's access, namely the posterior part, the patient was then positioned in the left lateral decubitus position. During the positioning period, the patient's heart rate and blood pressure did not significantly change. Along with surgery, his heart rate was 58–66 beats per minute, blood pressure was 90/60mmHg to 134/84 mmHg, saturation at 99–100%, and ECG was sinus rhythm without ST-segment depression or elevation.

On the 12th hour of surgery and after 600 cc of bleeding, the patient started to develop unifocal ventricular extrasystoles around 12 to 16 times per minute with a pulse at 43–48 beats per minute. Atropine 0.5 mg bolus was

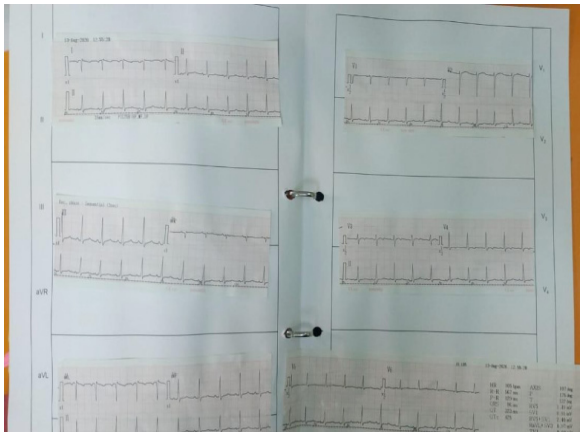

Figure 1 Preoperative ECG

given, and rhythm returned to sinus, although specific arrhythmia was noted on the ECG. We noted that the patient's temperature had continuously declined since the 6th hour surgery to 35.8–36.1°C despite the use of warming devices and warmed maintenance isotonic solutions.

Due to the recurring arrhythmia, attempts to increase oxygen delivery were performed by upping the oxygen concentration to 80%, an addition of 180 cc packed red cell and rewarming techniques proceeded. Even though blood pressure was relatively stable during the entire episode, we noticed that the ventricular extrasystole episode was recurring more often.

At the end of surgery, the patient was positioned back to the supine position, and preparation of emergence was done. Awake extubation was planned, but upon suctioning the airway for extubation preparation, the patient suddenly developed a pulseless ventricular tachycardia, and end-tidal CO2

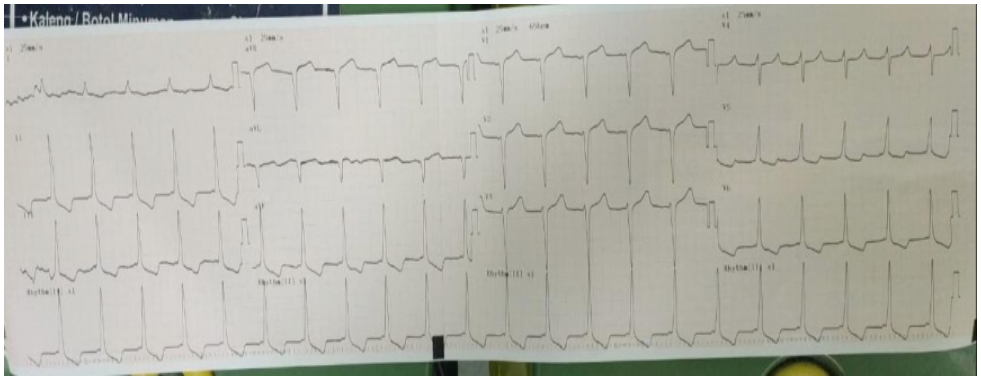

Figure 2 ECG on Return of Spontaneous Circulation

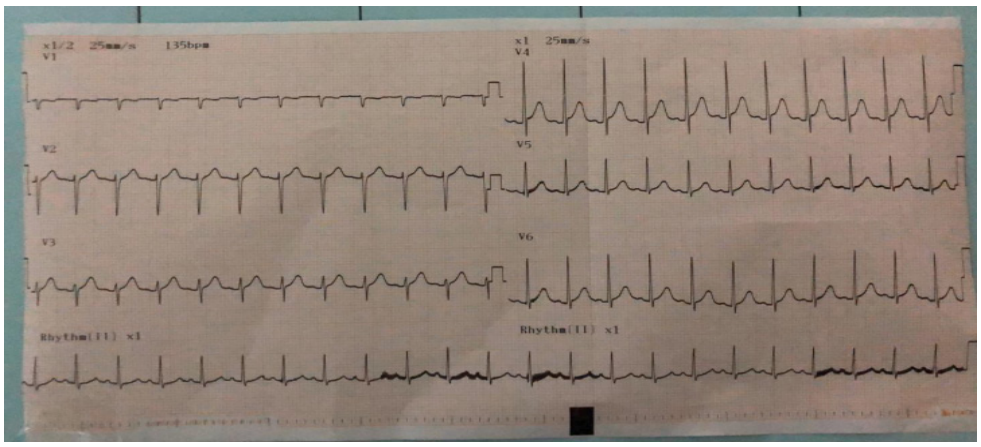

Figure 3 ECG Postoperation

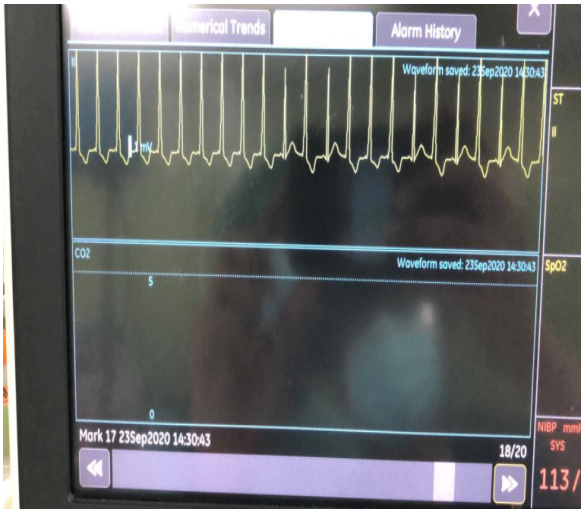

Figure 4 ECG During General Anesthesia: AVRT orthodromic

Figure 5 ECG During General Anesthesia: sinus tachycardia with intermittent preexcitation

was zero. Pulse check confirmed a cardiac arrest, and a blue code status was announced on the floor.

Immediate high-quality cardiopulmonary resuscitation using a guided feedback machine and 360 joules of defibrillation was conducted. A bolus of 1 mg of epinephrine was given during this time. Return of spontaneous circulation (ROSC) was achieved in five minutes, and patient rhythm was sinus tachycardia (Figure 2).

The patient was then transferred to the ICU, still intubated and deeply sedated. ROSC protocol, including targeted temperature management, was carried out in the ICU. Postoperative blood examination showed haemoglobin of 10.4 g/dL, lactate of 9.1, and blood pH of 7.18, reflecting a severe metabolic acidosis. Post ROSC ECG showed an abnormal delta wave, and the cardiologist made a diagnosis of WPW.

ECG showed left ventricular hypertrophy with an ejection fraction of 60%. Arrhythmia continued in the ICU, and propafenone 3 x 150 mg orally was given via nasogastric tube and achieved sinus rhythm within 24 hours (Figure 3). At 36 hours, hemodynamic parameters were stable, and the patient was then extubated. The patient was fully conscious, and a mini neurological examination showed no signs of motoric or sensory disturbance.

On 56 hours after surgery, the nurse noticed that the patient seemed paler and, upon quick examination on his leg showed an oozing blood on his bandages. The patient suffered continuous bleeding from the right leg and haemoglobin dropped to 6 gram/dL. Even after leg compressions and wrapping were done to minimize the bleeding, his hemodynamic was unstable.

The surgeon decided to conduct an emergency surgery bleeding control. The cardiac and intensive planned a further workup for possible ablation on the patient in the morning but due to the life- threatening event, surgery was deemed the only choice at that time. The patient was taken to the emergency operating theatre and standard monitoring was applied. The patient's heart rate was 132–138 times per minute with blood pressure at 90/60mmHg, ECG was sinus tachycardia, and saturation 99% using 5 liters of oxygen by a simple mask.

The patient was induced using 2 mg of midazolam, propofol 60 mg, fentanyl 100 microgram and 30 mg rocuronium. Volatile anaesthesia using sevoflurane was selected initially but was switched to TIVA due to extrasystoles. Anaesthesia was maintained with fentanyl and midazolam boluses with

60% oxygen and air (Figure 4).

During the surgery, multiple sinus tachycardia episodes with intermittent pre-excitation and AVRT occurred (Figure 5). Since intravenous propafenone was not available, a 300 mg dose via nasogastric tube and a bolus of diltiazem 0.25 mg/kg was given, and rhythm was stabilized. The patient was extubated and treated for two days in the ICU and released for further treatment in the ward.

Upon re-anamnesis, the patient mentioned having some episodes on palpitation in the past but was ignored because of its short onset and had no other complaints. An elective ablation was planned for the future.

Discussion

Arrhythmia is commonly found in the perioperative period. Determining whether or not a particular arrhythmia has the potential to cause perioperative morbidity and mortality has long been the anesthesiologist's goal. Arrhythmia can result from an abnormal initiation of impulse or electrical activity in a single cardiac cell or myocyte but more realistically from a close cluster of myocytes. The electrical activity origin is commonly found in the so-called pacemaker cells of the sinoatrial (SA) node or atrioventricular node (AV), but this activity may also occur in non-pacemaker myocytes. The mechanism found is similar to the physiological automaticity of pacemaker cells (SA and AV node), and is thus named 'abnormal' or 'enhanced automaticity'.

An alternative form of abnormal impulse initiation is due to the vibrations of membrane potential or early or delayed 'after-depolarizations'. In such cases, the resulting arrhythmias take the name of 'triggered activity'. Arrhythmias from enhanced automaticity and triggered activity are defined as 'non-re-entrant'. Arrhythmias can also be found when myocardial regions activated later in propagation re-excite regions with already recovered excitability. These are results from abnormal propagation of the excitation wavefront and/or of tissue refractoriness. This mechanism, named 're-entry', is based on the syncytial nature of myocardial tissue and is thus radically different from focal impulse initiation.[4]

Some people are born with an extra piece of heart muscle tissue that connects directly between the atria and the ventricles, bypassing the AV node altogether. This abnormal piece of muscle is referred to as a bypass tract or an accessory pathway. The extra piece of tissue can serve as a passageway for the electric signals between the atria and ventricles and allow the electric activity in the ventricles to occur immediately after the electric activity in the atria without having to wait for the electric impulse to travel through the AV node. In this situation, the ECG may not have much of a flat PR interval but instead may have the P wave (from the atrial activity) right up against the R wave (from the ventricular activity), with the R wave beginning with an upslope referred to as a delta wave (Figure 2). This delta wave results from the electric activity traveling over the accessory pathway and bypassing the AV node.[8]

Kaplan et al.[5] stated that WPW syndrome is a form of arrhythmias that involved one or more accessory pathways bypassing the atria and ventricles. Such disorders could trigger malignant arrhythmias such as atrioventricular reentry tachycardias (AVRT), pre-excited atrial fibrillation, or even ventricular fibrillation. Kulig and Koplan6 elaborated WPW syndrome as the combination of accessory pathway activation seen on an ECG (delta waves) and tachycardia episodes.

Historically, WPW was first described in 1930 by Louis Wolff, Sir John Parkinson, and Paul Dudley White. Along with the ominous delta wave, WPW patients have a shorter time between the conduction of an impulse from the atrium to the ventricle or referred as having a short PR interval. What is described above is called a pre-excitation or WPW pattern.[4,6]

Brugada et al.[4] stated that the presence of a pattern does not necessarily mean that the patient will experience WPW syndrome. The WPW pattern will be seen in about 0.2% of the general population. Of those patients with

the WPW pattern, a minority will experience tachycardia and be defined as having WPW syndrome, such as found in our patient. As found on our case, our patient also did not find any episodes of disturbing palpitations prior to surgery, and thus this case was harder to diagnose.

WPW can be associated with congenital heart disease involving endocardial cushions, such as Ebstein anomaly, a heart defect in which the tricuspid valve is abnormally formed and placed lower than normal in the right ventricle and atrioventricular septal defect. However, in most patients, WPW is not related to any other heart abnormality. Our patient also did not have any structural heart deformity on his subsequent echocardiography and all chambers were relatively normal.

Symptoms of WPW syndrome are usually abrupt and may include palpitations, chest discomfort, and occasionally fainting. On very rare occasions (less than 0.1% of the time), a patient with WPW can experience sudden cardiac death that results from the development of a chaotic irregular beating in the atrium, causing atrial fibrillation with rapid conduction down an accessory pathway leading to an extremely rapid pulse that can lead to cardiac arrest. Fortunately, this is a rare event in patients with WPW, and there are certain factors a physician can often identify ahead of time to risk-stratify patients with WPW.

WPW syndrome is usually diagnosed in the preoperative period with the suggested symptoms of arrhythmia and abnormal delta wave findings of ECG. As Brugada et al.4 and Bengali et al.[3] stated, asymptomatic WPW can occur in 10–30% of cases and is also relevant to this patient. Preoperative ECG showed no delta wave, which is due to intermittent preexcitation.

KC et al.[7] reported a similar case, but the patient was diagnosed preoperatively due to light palpitations, an abnormal ECG and a 24-hour Holter monitoring was done. Due to the difficulty of finding it on a routine ECG, another examination was warranted. A Holter monitor, which is a portable 24 hours heart electrical activity monitoring device, an exercise stress test, or an electrophysiology study, should be performed to diagnose WPW preoperatively.[4,8] These examinations could be considered for young patients with supraventricular tachycardia (SVT) suggestive symptoms,[9] such as history of recurrent palpitations, lightheadedness, dyspnoea, angina, or fatigue in the preoperative period.

On this patient, a normal ECG during the preoperative period plus the patient's asymptomatic state led to an additional preoperative examination, although available in our hospital, was not carried out on this patient.

Further anamnesis in the post-op period concluded differently after the patient stated that he actually had several episodes of "chest fluttering" and "uncomfortable chest" in the past that lasted for a "short time" and claimed that it resolved after rest. The patient did not relate this symptom to the palpitations or limitation of activity questions in the standard pre-anesthetic questionnaire and history taken by our staff. Palmieri & Stern[10] stated that 40–60% of patients do not disclose their symptoms to their doctor. The reason for this behavior may be the preservation of autonomy, avoidance of therapy, or needing therapy and assert one's sense of self. Graham & Brookey[9] found that simply some patients do not understand the question asked by their doctor and are ashamed to tell their doctors that they do not understand. This patient confessed to stating his complaints due to fear of delay in his surgery since he was already on a long waiting list due to the Covid19 pandemic that affects our hospital's ability for elective surgery. It is crucial to understand our patients better and that anamnesis and effective communication should be taught at medical schools, and evaluated for their quality during practice.

Intermittent WPW is considered low risk in causing cardiac arrest, and risk stratifying either by non-invasive or invasive strategy is needed.[4,8] However, wide complex QRS tachycardia was induced during added stress of long surgery that also required microvascular anastomosis tissue transplantation. This

catastrophic episode indicated the future need for an electrophysiology study and catheter ablation as secondary prevention since the patient risk for sudden cardiac arrest is now classified as high risk.

During surgery for WPW patients, anaesthesia aims to blunt sympathetic triggers such as laryngoscopy, pain, light anaesthesia plane, hypovolemia and fever. Deviseti and Pujari[11] stated that some anaesthesiologists prefer regional anaesthesia since it reduces polypharmacy and avoids laryngoscopy but, in this patient, since surgery was performed in both posterior trunk and legs, regional anaesthesia is impossible. The length and extent of surgery on this patient may have triggered higher stress response compared to his previous history of short uneventful surgeries. On the second surgery, the patient was in hypovolemic shock and further triggered sympathetic response and subsequent re-entry episodes.

One of the key successes of resuscitation in this report was the effective high-quality compression and defibrillation given during the cardiac arrest period. Guided CPR devices and $ETCO_2$ have a better outcome and ROSC, especially in witnessed cardiac arrest. Resuscitation on the surgical table, although catastrophic but outcomes show that witnessed cardiac arrest has better ROSC case.[12,13] In our hospital, basic life support training and phantom cases increase the anaesthesia team alertness and preparedness for specific unpredicted episodes, such as in this case. All equipment is also standardized, and maintenance is also crucial in maintaining a responsive resuscitation team.

WPW syndrome could be overt as various tachyarrhythmias. Most of them induced orthodromic AVRT, which traveled the re-entrant impulse anterogradely through the AV node, from atria to ventricle, and through the accessory pathway (AP) in a contra manner or retrogradely. Orthodromic AVRT manifests as narrow complex tachycardia in ECG, while a wide complex tachycardia in ECG can be manifest if the re-entrant impulse conducts anterogradely using AP and re-entered by AV node retrogradely, being called antidromic AVRT. The AVRT might deteriorate and inducing atrial fibrillation (AF), and the impulse travels through the AP and resulting in rapid ventricular response and fatal condition. Thus, the drug of choice should be in accordance with the tachyarrhythmia's mechanism and the drug's pharmacological properties on the precipitating factors such as extrasystole or AF, and arrhythmia perpetuation's mechanism, for example, cardiac chambers', anterograde and retrograde pathways' refractory periods.

Brugada et al.[2] stated that vagal maneuver and intravenous adenosine are the first line strategies in managing AVRT. However, adenosine is no longer available in our country; therefore, the drug of choice for antidromic AVRT as seen in our patient is intravenous procainamide or propafenone, a class IA and IC antiarrhythmic. Both of them inhibit the fast inward sodium channel during phase 0 of the action potential. Procainamide slightly depresses it while propafenone markedly reduces it and has little effect on repolarization. They also prolong the chambers and AP's refractory period, anterogradely and retrogradely, in a consistent manner; thus, they will halt the reciprocating reentry impulse. However, they were unavailable in our hospital, so diltiazem or verapamil becomes our option in this case.

Diltiazem and verapamil, as part of dihydropyridine calcium channel blockers, share similar pharmacological properties. They mainly cease the AVRT on the AV node by prolonging Atrio-Hisian (AH) interval, prolonging AV nodal effective refractory periods, maintaining AV nodal conduction in 1:1 ratio, and shortening atrial pacing cycle length. They do not affect atrial, ventricular, and AP's refractory periods. They can effectively stop the perpetuating re-entry but not directly prolonging the AP; thus, they are preferred in orthodromic rather than antidromic AVRT.

The use of amiodarone as the tachycardia pathway suggest may not be beneficial since it strengthens the re-entry pathway of WPW.8 In orthodromic AVRT, it has class IIb recommendation, while not recommended

in antidromic one. Amiodarone is a class III antiarrhythmia with nonselective activity and prolongs all parts of the cardiac refractory period. Most of the time, it effectively prevents the extrasystoles that provoke tachyarrhythmias, and it shows inconsistency in inhibiting the re-entrant pathway. It also can trigger ventricular arrhythmias in pre-excited AF and antidromic AVRT.

Cardioversion is always an option if there is no appropriate antiarrhythmic or pre-excited atrial fibrillation occurs. The patient was given oral propafenone titrated up to 300 mg tid as a maintenance dose and scheduled for elective AP ablation.

Conclusion

Intermittent WPW is a rare condition. It may appear as an asymptomatic condition; however, young patients with SVT suggestive symptoms may be required to screen for intermittent AP in the preoperative period, especially for those who will undergo moderate to high-risk, long-duration surgery. Good anaesthesia management and monitoring, knowledge of appropriate antiarrhythmic agents and rapid, high-quality resuscitation provisions in the operating room can provide good outcome if an unsuspected intraoperative crisis occur.

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