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Wolff Parkinson White Syndrome: Which Best Perioperative Strategy?

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Abstract

Background: First described in 1930, Wolff Parkinson White Syndrome is a congenital electrophysiological disorder that can results in life-threating arrhytmias.

Case Presentation: We report the case of a 22-year-old asymptomatic woman with a history of Ebstein's anomaly surgically treated, related to a WPW syndrome and a PFO, who underwent laparoscopic cholecystectomy under general anesthesia.

Conclusion: In Wolff-Parkinson-White patients, general anesthesia can be safe taking adequate monitoring and precautions.

Keywords: Wolff-Parkinson-White syndrome; General Anesthesia; Cholecystectomy; Arrhythmias, Cardiac.

Introduction

The electrocardiographic (ECG) pattern of a shortened PR interval, a delta wave (or pre-excitation), and a prolonged QRS complex was first described in 1930 by Louis Wolff, Sir John Parkinson and Paul Dudley White that reported eleven cases [1]. This Wolff Parkinson White (WPW) pattern becomes a syndrome when the patient presents symptoms (e.g., palpitation and dyspnoea). In 1967, during epicardia mapping in a WPW patient with an atrial septal defect, two atrioventricular connections were found to exist instead of one: this additional accessory pathway (AP) was linked to the syndrome [2]. WPW is often associated with other congenital heart lesions, such as Ebstein's anomaly and patent foramen ovale (PFO). Ebstein's anomaly is a congenital malformation characterized by malformed and displaced tricuspid valve leaflets, partly attached to the tricuspid annulus, and partly attached to the endocardium of the right ventricle, causing regurgitation and enlargement of the right heart [3]. Clinical presentation varies with severity of the lesion. This condition is of interest to the anaesthesiologist because of alterations in electrical conduction due to anaesthetic drugs and cardiovascular changes secondary to surgical stress [3]. Therefore, perioperative physicians must have a clear understanding of the syndrome to provide timely management [4].We report the case of a 22-year-old asymptomatic woman with a history of Ebstein's anomaly, treated surgically in childhood, related to a WPW syndrome and a PFO, who underwent laparoscopic cholecystectomy under balanced general anesthesia (intravenous and inhalation) without experiencing any arrhythmic complication.

Case Presentation

A 22-year-old woman, weighing 52 kg and 169 cm tall, underwent laparoscopic cholecystectomy in November 2023 for a history of recurrent colic in cholelithiasis. The woman denied chest pain and dyspnoea. The 12-lead ECG showed sinus rhythm with a right bundle-branch block. Both laboratory tests (complete blood count, coagulation, electrolytes and hepatorenal function) and systemic clinical examination were normal, with standard blood pressure values (105/70 mmHg), and a regular heart rate of 82/min. Her medical history showed a surgically treated Ebstein's anomaly in 2012, a PFO and asymptomatic WPW (unsuccessfully treated with a first ablative attempt). Her history included episodes of syncope,

for which she was a loop recorder carrier.

Strict monitoring, including Bispectral index and T.O.F., was applied in the operating room. A 12-lead ECG showed normal heart rate with a right bundle branch block associated with right axis deviation. Medications (esmolol, lidocaine, and procainamide) and defibrillator were kept on hand. Preoxygenation with 100% oxygen for three minutes was followed by induction with propofol 2mg/kg, fentanyl 3gamma/ kg and passage of a 6.5mm cuffed endotracheal tube was made easy after administration of rocuronium (0.6 mg/kg).

Anaesthesia was maintained with sevoflurane titrated to BIS levels (between 40-60), and ventilation was provided by intermittent positive pressure ventilation (i.e., end tidal volume 375 ml, respiratory rate 12/min, PEEP 5mmHg). Pneumoperitoneum was limited within 12 mmHg of CO2 insufflation, and end tidal CO2 was maintained within physiological limits (i.e., 35-40 mmHg).

Analgesia was completed with acetaminophen 1g, and prophylaxis against postoperative nausea and vomiting (PONV) was successfully managed with dexamethasone 4mg and ondansetron 4mg. At the end of surgery, muscle relaxation was reversed with Sugammadex 2mg/kg, no extubation reflexes were observed, and given hemodynamic stability, the patient, after adequate monitoring in the recovery room, was safely transferred to the postoperative ward.

Discussion

Although most patients remain asymptomatic throughout life, these individuals are always prone to develop atrial fibrillation or other arrhythmias due to the presence of APs. The WPW pattern is present up to 0.25% of the population [5]. About 1% of those having a WPW pattern have the WPW syndrome [6]. The risk of sudden death, due to malignant arrhythmia is estimated at 0.4%/ year in patients with the WPW syndrome [7].

Diagnosis is done with history and ECG. The AP is a tissue remnant left over from embryologic formation of the heart with different conduction speeds (in most cases is the “Kent” pathway) as opposed to the single normal atrioventricular node (AVN) [8]. The AVN uses a calcium-dependent slow inward current, while the AP uses a sodium-dependent fast inward current for electrical impulse transmission [3]. Anaesthetic drugs tend to change the physiology of atrioventricular (AV) conduction [9]. An important safety feature of the AVN is the “decremental conduction” that serves as a protective mechanism reducing the speed of conduction in the AVN as the heart rate increases, feature not displayed by the AP that can conduct extremely rapidly at a ratio of 1:1 for the atria to the ventricles [4]. This determines that depolarization impulses from the atria can reach the ventricles via both pathways resulting in three features of abnormal electrical conduction: a short PR interval (less than 0.12 seconds), Pre-excitation or “delta” wave (determined by the fusion between the early- via the AP- and the late – via the AVN- ventricular depolarization), and a widened QRS-wave (result of pre-excitation) [4]. Traditionally, WPW is classified in type A and B. The first, on ECG, resembles a right bundle branch block with right hypertrophy frequently associated to the Ebstein’s anomaly (as seen in our patient), whereas the latter, resembles a left bundle branch block with left ventricular hypertrophy [10].

It must be considered that not always these electric characteristics are present: a delta wave may not be apparent according to the duration of the refractory period of the AP [11] as well as some APs may only conduct in a retrograde manner participating in re-entrant arrhythmias with a normal ECG showing a sinus rhythm without a WPW pattern (i.e. “concealed pathways”) [12]. 20% of WPW syndromic patients present with atrial fibrillation or flutter while the remaining majority present an atrioventricular re-entrant tachycardia (AVRT) that can be either orthodromic (OAVRT) or antidromic (AAVRT) [13]. The first (76% of arrhythmias) displays a narrow QRS complex (unless a pre-existing alteration or an aberrant conduction is present) with no pre-excitation because of an atrioventricular conduction via the AVN with a back up to the atria via retrograde conduction through the AP [14]. The latter instead is also a re-entrant tachycardia, but depolarization implies travels in the opposite direction amounting to a 4% of the arrhythmias in the syndrome with a wide QRS indistinguishable from ventricular tachycardia [15]. Atrial fibrillation and flutter are not based on atrioventricular circular electric loops; instead, impulses are conducted via the AP, with a ventricular rate that will be determined by the refractory period of the AP, representing a true life-threatening situation [13].

Radiofrequency ablation remains the best method for prevention therapy. It presents a success rate of 90% demonstrating the best long-term method for final therapy [16].Among those who fail the first ablation the second one present a nearly 100% complete resolution of symptoms [16]. Surgical ablation is reserved for malignant pathways that fail radiofrequency or for those not eligible for ablation (e.g., AP in proximity to normal conducting pathways) [17]. Alternatively, Class Ia (e.g., procainamide), class Ic (e.g., flecainide, propafenone) and Class III (e.g., amiodarone, sotalol, ibutilide) are anti-arrhythmic medications that all slow AP conduction [18]. If there is a history of flutter or atrial fibrillation class II (beta-blockers including metoprolol and esmolol) and class IV (calcium channel blockers such as verapamil) can be added to class Ia and Ic to slow AVN conduction [4].Ic drugs are contraindicated in congestive heart failure and in coronary artery disease [19]. In pregnancy both sotalol and flecainide are safe [20] and all medications, used for long-term management, must be taken at the usual doses on the day of surgery [4].

Presence of WPW alone does not warrant invasive monitoring or pre-emptive placement of cardioversion pads.

When arrhythmia occurs intraoperatively, the urgency of intervention is contingent on the hemodynamic stability of the patient. If hemodynamically stable, drug treatment should be attempted, whereas if drugs are not possible or if hemodynamically unstable, direct synchronized cardioversion (50 to 200 biphasic joules) is required [21]. If synchronization is not possible, direct defibrillation should be attempted (200 Joules biphasic) since tachycardia at such high rates May quickly lead to ventricular fibrillation [21].

In stable OAVRT, adenosine is the best first line agent (in doses of 6mg, 12mg, and 12mg again every 1 to 2 minutes) when vagal manoeuvres are ineffective [22]. It inhibits adenyl cyclase, increasing potassium out flux determining an increased refractory period at the AVN. It should be used with caution in those patients prone to bradycardia [23]. As second line agent for OAVRT, verapamil can be administered at the dosage of 5mg every 2 to 3 minutes (for a maximum of 15mg) with caution in hypotensive patients considering the risk to decrease cardiac output [24]. Next choice includes intravenous procainamide at 10mg/kg over 10 minutes up to 15mg/kg within 30 minutes or intravenous beta-blockers [25]. The latter blocks sodium channels prolonging refractoriness in the AP as well as the His Purkinje system, but not in the AVN [26]. Intravenous amiodarone can be considered in those situations where no other medical treatments are amenable since it prolongs refractoriness of all myocardia (both AP and normal pathways) [27]. In stable AAVRT, intravenous procainamide is the best option followed by amiodarone as a load of 150mg followed by a continuous infusion of 1mg/min over 6 hours then 0.5mg/min over 18hours [28].

Regardless, in the presence of tachycardias of unclear mechanism, procainamide, by slowing AP, diverts conduction along normal pathways and is the best option when available.

It is critical that the perioperative setting provider obtain a thorough cardiac history, as palpitations, syncope, angina, and dizziness may be the only clues to a latent WPW syndrome [7]. The incidence of cardiac arrhythmias during general and regional anaesthesia suggests rates as high as 61% [29]. Perioperative nausea, hypothermia, sympathetic blockade, laparoscopic insufflation, laryngoscopy, and hyperventilation may accentuate the propagation of arrhythmias [30]. Although propofol, benzodiazepines, fentanyl, sevoflurane, or desflurane may alter electrical conductivity and refractoriness, none has demonstrated clinical relevance [31]. From a practical point of view, balanced anaesthesia seems optimal, considering that sevoflurane has no effect on AVN conduction, providing optimal hemodynamic stability along with fentanyl and rocuronium [32]. What should be avoided is a light plane of anaesthesia (as it causes episodes of stress), excessive sympathetic stimulation (e.g., through laryngoscopy and reversed myorelaxant agents) [33], and predominant vagal tone (causing AVN slowing by directing conduction along the AP) [8]. Thus, the perioperative goal of anaesthesia is a medium balance between sympathetic augmentation and vagal tone.

Conclusions

Appropriate management of arrhythmias is critical and must be performed in a timely manner [4]. WPW tachycardias are unpredictable and life threatening and represent a challenging scenario for the anaesthesiologist [4]. This is because anaesthetic drugs alter the physiological AV conduction [34]. Therefore, clinicians must be watchful and mindful of the salient features of WPW, considering the epidemiology, pathophysiology, and treatment modalities [4]. Proper management in the perioperative setting includes adequate prevention of pain, anxiety, and stress response (secondary to intubation, extubation, hypovolemia, and a lighter anaesthesia plan) [3]. In conclusion, our case is intended to emphasize, once again, how adequate medical knowledge coupled with opportune precautions that avoid a sympathetic rise are the key to a successful outcome in patients with WPW syndrome undergoing surgery [3].

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