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Wolff-Parkinson-White syndrome: anaesthetic care for meningioma excision

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Abstract

In Wolff-Parkinson-White (WPW) syndrome, the presence of an accessory pathway between the atrium and ventricle predisposes the patient to paroxysmal supraventricular tachyarrhythmias, which may progress to ventricular fibrillation and sudden cardiac death. Several drugs that are used perioperatively may alter the cardiac conduction velocity and refractory period. This fact, interacting with factors such as increased sympathetic tone (*e.g.*, anxiety, pain, or seizure) or haemorrhage, leads to tachycardia, where shortened R-R interval predisposes the heart to re-entrant tachyarrhythmias. We reported and highlighted the perioperative issues while anaesthetising a 15-year-old boy with WPW syndrome for craniotomy and excision of parietal meningioma.

Keywords: craniotomy, general anaesthesia, intracranial surgery, meningioma, Wolff-Parkinson-White syndrome

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Introduction

Wolff-Parkinson-White (WPW) syndrome is a subtype of a ventricular pre-excitation syndrome caused by the presence of an anomalous accessory pathway (bundle of Kent) between the atrium and ventricle. The most significant anaesthetic risk in these patients is the development of haemodynamically unstable tachyarrhythmias, which may progress to ventricular fibrillation and sudden cardiac death. Currently, there is no validated scoring system that predicts the risk of tachyarrhythmias under anaesthesia. Its anaesthetic management is challenging, more so in neurosurgery, where specific neuroanaesthesia goals may be at times in conflict with haemodynamic considerations. Here, we present the successful anaesthetic management of a patient with WPW syndrome for the excision of a large parietal meningioma.

Case presentation

A 15-year-old boy was admitted to the cardiology ward for a workup of WPW syndrome after presenting with chest discomfort, syncopal attack, and breathlessness that subsided on their own. His presenting electrocardiogram (ECG) showed the classical type A WPW pattern: shortened PR interval and delta waves with widened QRS complex without right ventricular hypertrophy, while his 24-hour Holter showed several episodes of non-sustained supraventricular tachycardia, for which no intervention was required. In the cardiology ward, he developed one episode of a generalised tonic-clonic seizure. Following that, an urgent contrasted computed tomography of the brain (Fig. 1) was performed, revealing a large left parietal meningioma (10.2 x 8.8 x 8.5cm) with adjacent skull hyperostosis. He was posted for elective left craniotomy and excision of parietal meningioma. During preanaesthetic consultation, he was asymptomatic and fit-free, with a baseline heart rate of 74–83 beats/min. His blood investigations were normal. The echocardiogram did not show evidence of Ebstein anomaly or valvular pathology. After the cardiologist consults, he was not started on antiarrhythmic drugs, and we were cautioned to avoid tachycardia intraoperatively. The patient and his guardian were adequately counselled on the high cardiac risk and reassured. The patient was kept fasted, and an intravenous (IV) fluid was started to keep him euvolemic. No premedication was given to avoid obtundation of his neurological status.

We took all the necessary precautions to avoid tachycardia and arranged vital drugs to treat complications together with stringent monitoring, which were very important for a favourable outcome in this patient. In the operating room, an arterial line was inserted while the patient was still awake, and a self-adhesive

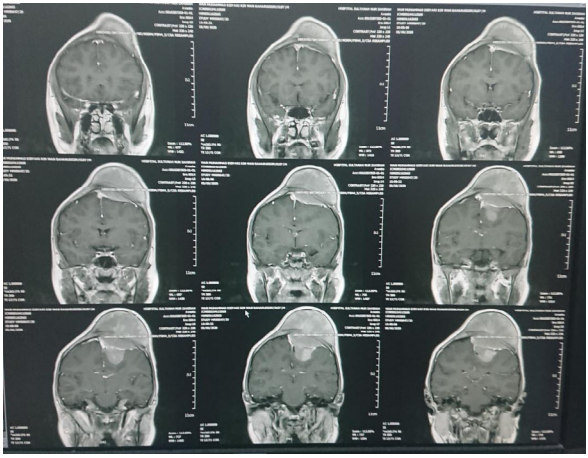

Fig. 1. A contrast-enhanced computed tomography of the brain (sagittal view) shows a large, left parietal meningioma measuring 10.2 x 8.8 x 8.5 cm with adjacent skull hyperostosis.

defibrillator pad was applied at the anterior-lateral placement prior to induction. A crash cart with relevant antiarrhythmic drugs (adenosine, esmolol, lignocaine and amiodarone) was also available in the operating room. Cardiac-stable induction was performed, with target-controlled infusion (TCI) remifentanil infusion [Minto model, effect site-targeted concentration (Cet) up to 6 ng/ml], titrated dose of IV propofol (total 120 mg), and IV rocuronium 50 mg. Care was taken not to hyperventilate the patient, and intubation was performed at the deep plane of anaesthesia. Intubation proceeded without any untoward events. The patient was put on a ventilator, and sevoflurane in an oxygen and air mixture was started. This was followed by obtaining additional IV access, a central venous line, and Foley's catheter insertion. Prior to head pinning, scalp block was performed at bilateral supratrochlear, supraorbital, zygomaticotemporal, and auriculotemporal nerves using landmark technique, with 3 ml of 0.25% bupivacaine (adrenaline free) per site. TCI remifentanil was then increased to Cet 6 ng/ml, and the Mayfield clamp was applied by the surgeon. Before starting the surgery, IV esmolol 10 mg was given to attenuate the sympathetic surge.

Intraoperatively, there were two episodes of sinus tachycardia (maximal heart rate of 110 beats/min): at scalp dissection and the period of rapid blood loss during meningioma excision (Fig. 2). During the first episode, which was probably due to intense pain stimuli, analgesia was managed by increasing TCI remifentanil to Cet of 6 ng/ml, and supplemental bupivacaine was infiltrated by the surgeon directly at the surgical site. In the second episode, which was probably due to ongoing blood loss, fluid resuscitation was initiated with crystalloids, packed red blood cells, and blood components. In both instances, we managed to return the patient's

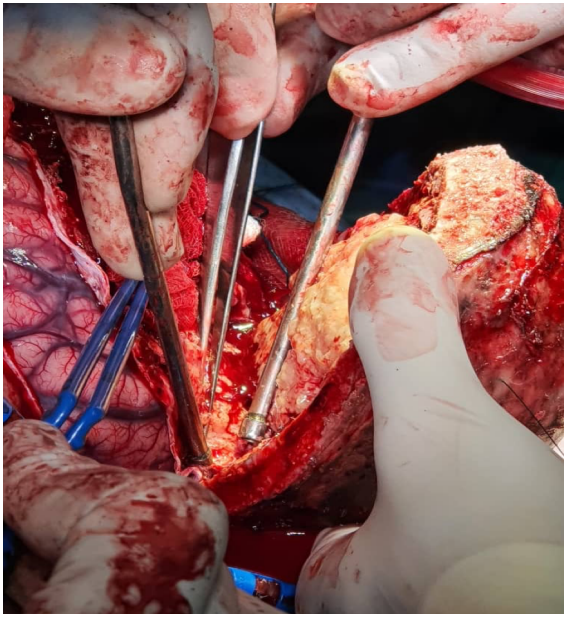

Fig. 2. The second episode of tachycardia occurred during excision of the meningioma, where there is a possibility of ongoing and underestimated blood loss.

heart rate to his baseline without any episodes of malignant supraventricular tachycardia and significant haemodynamic instability. The meningioma was successfully removed after 3 hours. After surgery, IV sugammadex 2 mg/kg was given, and the patient was extubated. Extubation was controlled and smooth. Postoperatively, his pain was managed with a titrated IV dose of fentanyl and morphine. He remained stable in the recovery and was transferred to the neuro intensive care unit (ICU) for monitoring. He was reviewed by the cardiologist in the neuro ICU and was given an electrophysiology study (EPS) appointment.

Discussion

Most patients with WPW syndrome may remain asymptomatic throughout life, but these patients are at risk of paroxysmal supraventricular tachycardia, atrial fibrillation, and sudden cardiac death during the perioperative period. Due to the rarity of this condition, the incidence of patients with WPW syndrome presenting for surgery is unknown, but ECG screening in the general population showed that asymptomatic WPW occurs in 0.7–1.7 patients per 1,000 population, where a further 1–1.8% are symptomatic.[1,2] For symptomatic patients, the most common presen-

tations include palpitations, chest pain, and dyspnoea. Our patient presented with chest discomfort, syncopal attack, and breathlessness, which were self-limiting but warranted a workout because of recurrent symptoms in a previously fit young man.

The normal atrioventricular (AV) node utilizes a calcium-dependent slow inward current, while the accessory pathway in WPW syndrome patients utilizes a sodium-dependent fast inward current for electrical impulse transmission. The lack of physiological delay in transmission of the sinus impulse via the abnormal accessory path*, i.e.*, the bundle of Kent, results in a short PR interval. Ventricular excitation, formed by the two impulses, results in a fusion beat seen as a "delta wave" with prolonged QRS complex in a typical ECG of WPW syndrome. WPW syndrome is traditionally classified into type A and type B. On ECG, type A resembles a right bundle branch block with right ventricular hypertrophy and posterior myocardial infarction, whereas type B resembles a left bundle branch block with left ventricle hypertrophy.[3] Our patient's ECG showed a type A pattern but without right ventricular hypertrophy. The cardiac anomaly most frequently associated with this problem is Ebstein's anomaly, but not in our patient, as his echocardiogram was normal.

Most anaesthetic drugs and techniques tend to change the physiology of AV conduction. The principle guiding perioperative anaesthetic management in this patient was to utilize agents with the least effect on myocardial contractility, conduction velocity, and refractory period with minimal circulatory depression.[4] Specific to the neurosurgical population, the selected agents must provide a rapid central nervous system recovery. In addition to the correct use of anaesthetic agents, a proper plan of the anaesthetic technique, teamwork, and vigilant monitoring are key to surgical success.

The effect of most anaesthetic drugs on conduction speed and the refractory period has been studied, principally derived from anaesthetic management during EPS.[4-6] Anaesthetic agents proven to be devoid of effect on cardiac conduction are fentanyl, remifentanil, alfentanil, propofol, etomidate, cis-atracurium, and rocuronium.[7] In fact, propofol has been shown to normalize WPW conduction in one case series.[8] For inhalational agents, sevoflurane, isoflurane, and nitrous oxide have been proven to be safe. Anaesthetic agents that are proarrhythmic (halothane), have intrinsic sympathomimetic properties (ketamine, desflurane, meperidine), or cause tachycardia either due to histamine release (causing reflex tachycardia, such as thiopentone and atracurium) or vagolysis (such as pancuronium) should be avoided. Specific to neurosurgery, such as in this case, fentanyl, propofol, and rocuronium were used during induction, and the anaesthesia maintained with remifentanil and sevoflurane in oxygen and air. Desflurane, with its potentially

detrimental effect on cerebral haemodynamics, was avoided in our patient. For the reversal of neuromuscular blockade, sugammadex, a selective reversal binding agent devoid of autonomic properties, was used.[9] Neostigmine, which increases parasympathetic activation and hence delays myocardial conduction speed and prolongs the refractory period, was avoided. Furthermore, anticholinergics such as atropine and glycopyrrolate, which are coadministered with neostigmine, were avoided due to their vagolytic properties.

Induction is a high-risk procedure in WPW syndrome, as 20% of tachyarrhythmias occur during induction of anaesthesia, and 10% develop ventricular fibrillation. For this patient, the intubation was performed smoothly and gently by the anaesthetist within a short intubation time. Monitoring in this patient was achieved by serial 12-lead ECG, defibrillator cardiac monitor, invasive arterial waveform analysis with pulse pressure variation as well as systolic pressure variation reading and bispectral index monitor.

Skull pinning is an intense and stimulating part of neurosurgery. Several methods have been proposed for its management, such as deepening the anaesthetic agents, opioids such as fentanyl or remifentanil, beta blockers such as esmolol or labetalol, scalp block, or avoidance of skull pinning where possible. In patients with WPW syndrome, sympathetic activation increases the conduction speed through the accessory pathway, putting them at risk of tachyarrhythmia. The scalp is richly vascularized. As a result, the risk of cardiotoxicity and local anaesthetic systemic toxicity (LAST) is higher. Levobupivacaine, which has a higher (safer) cardiovascular collapse:central nervous system ratio, yet has a reasonable duration of action, should be the local anaesthetic of choice, but it was unavailable for our patient and only an epinephrine-free preparation was used. Adrenaline is a potent vasoconstrictor with the potential advantages of prolonging block duration, reducing the risk of LAST, and reducing bleeding. However, its use for scalp block is associated with a biphasic response: an earlier, brief episode of hypotension caused by beta-2 adrenoceptor-induced systemic vasodilation, followed by hypertension and tachycardia due to beta-1 and alpha-1 adrenoceptor activation, causing increased dromotropy, inotropy, and vasoconstriction—both of which were detrimental in our case.[10]

Treatment of perioperative hypotension is crucial. Phenylephrine (an alpha-1 agonist) should be the vasopressor of choice. Phenylephrine, with its resultant reflex bradycardia, has been shown to abolish pre-excitation in one case series. Furthermore, phenylephrine does not affect intracerebral vasomotor tone since the alpha-1 receptor is absent in the brain vasculature.[11] Euvolemia should be instituted to minimize vasopressor use. In this case, fast response on catching up of the blood losses with fluid and blood products avoided any episodes of precip-

itated intraoperative tachyarrhythmia. In urgent situations with supraventricular tachycardia, especially with atrial fibrillation and atrial flutter, direct current shock is the treatment of choice, for which the defibrillator pad was readily attached preinduction. Patients developing atrial fibrillation with haemodynamic stability should be treated pharmacologically. In contrast, haemodynamically unstable patients should be treated by cardioversion with 150–200 J. Digoxin and verapamil should be avoided, as these antiarrhythmics have been shown to enhance anterograde conduction through the accessory pathway.

Hyperventilation, with its resultant hypocapnia, lowers the conduction threshold and should be avoided. Furthermore, it is not favourable in the neurosurgical patient as it impairs cerebral perfusion. In this patient, extubation response can be detrimental. Few reports in the literature show that tracheal extubation under deep anaesthesia may reduce the incidence and complications. However, it often causes airway obstruction and risks an unprotected airway. Particularly in the neurosurgical patient, assessment of neurological recovery mandates extubation in an awake patient. Few case reports have implemented laryngeal mask airway insertion after tracheal extubation, which may minimize the stress response while providing a patent airway during emergence from anaesthesia (Bailey procedure). However, in our patient, only after transferring him to the ICU bed, supplementation of adequate multimodal analgesia, gentle suctioning of oral airways, and keeping the remifentanil at a lower dose, did we cut off the gas before he regained full consciousness until extubation.

Conclusion

Successful anaesthetic management of patients with WPW syndrome depends on factors such as cardiac stable anaesthesia, avoidance of tachycardia and sympathetic surge, and ability to manage haemodynamically significant tachyarrhythmia. Scalp block using adrenaline-free local anaesthetics is an attractive method of analgesia.

Declarations

Informed consent for publication
This case report was published with the written consent of the patient and his mother.

Competing interests
Dr. Vanitha Sivanaser serves as Section Editor of Malaysian Journal of Anaesthesiology. Dr. Sivanaser was not involved in any part of the publication process prior to manuscript acceptance; peer review for this journal is double blind. The remaining authors state no conflict of interest.

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