ANAESTHETIC MANAGEMENT OF PATIENT WITH MITRAL VALVE PROLAPSE AND WOLF PARKINSON WHITE SYNDROME FOR UROLOGICAL PROCEDURE

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ABSTRACT: Mitral valve prolapse (MVP) is a common valvular cardiac abnormality in the general population and is very rarely associated with Wolf Parkinson White (WPW) syndrome. These patients are prone for life threatening arrhythmias. Anaesthetics tend to change the electrophysiology of the atrio-ventricular conduction system and hence they tend to affect the behavior of the patient under anaesthesia. We present a case of mitral valve prolapse with WPW syndrome who underwent successful general anaesthesia for open nephrolithotomy.

KEY WORDS: MVP, WPW syndrome, dysrhythmias, general anaesthesia.

INTRODUCTION: Mitral valve prolapsed (MVP) is the most common valvular cardiac abnormality with prevalence rates ranging from 5-20%.^[1] There is an association between MVP and other congenital heart diseases such as ostium secundum atrial septal defect, hypertrophic cardiomyopathy, Marfan's syndrome, Ebstein's anomaly and Wolf Parkinson White (WPW) syndrome, although these are rare occurrences.^[2] An association between WPW syndrome and MVP was noticed for the first time in 1975.^[3] The classical pre-excitation syndrome was first described by Wolf-Parkinson and White in 1930 as an ECG, consisting of a "functional bundle branch block and short PR interval occurring in otherwise healthy, young people with paroxysms of tachycardia".^[4]

CASE HISTORY: A 41yr old male with known MVP and WPW syndrome was diagnosed with left sided renal calculus and was scheduled for open nephrolithotomy. Patient had repeated episodes of left loin and low back pain for the past 6 months. He had history of repeated episodes of palpitations, no dyspnoea on exertion and good effort tolerance. On examination pulse rate was 80/min, regular and blood pressure 116/78mmHg. Cardiovascular examination did not reveal any abnormality. ECG showed short PR interval with slurring upstroke of QRS complex (delta wave) suggestive of WPW syndrome. Echocardiography revealed thickened mitral leaflets with prolapsing anterior mitral leaflet and trivial mitral regurgitation. Rest of the preoperative investigations was within normal limits. He was offered catheter ablation for his WPW syndrome by the Cardiologist, which the patient refused. He was not on any medications

for the same. The proposed surgery was initially planned under combined general anesthesia with thoracic epidural for perioperative pain management. But the patient refused epidural, hence was done under general anaesthesia.

Patient was premedicated with alprazolam, pantoprazole and ondansetron. ECG, non invasive blood pressure and pulse oximetry monitoring were initiated. Anaesthesia was induced with propofol 2mg/Kg and fentanyl 2mcg/Kg and tracheal intubation with No.8.5 endotracheal tube was facilitated with vecuronium 0.1mg/Kg. Anaesthesia was maintained with oxygen, nitrous oxide and isoflurane 0.5-1% on circle system with controlled ventilation. Patient was positioned in right lateral posture and was stable throughout the intraoperative period with heart rate between 60-80/min, mean arterial pressure between 70-90 mmHg, end tidal carbon dioxide ranging 30-35mmHg and oxygen saturation 99-100%. Patient did not require anymore analgesics in the intraoperative period. At the end of the surgery (which lasted for 50mins), when patient had respiratory attempts, neuromuscular blockade was reversed with neostigmine 2.5mg and glycopyrrolate 0.5mg. Recovery and extubation was smooth and uneventful. During the first 24 postoperative hours the patient was monitored in intensive care unit. Postoperative analgesia was achieved with fentanyl 25mcg/hr iv infusion along with paracetamol 1gm iv infusion tid. Postoperative period was uneventful and the patient was discharged on 5th postoperative day.

DISCUSSION: The vast majority of patients with MVP are asymptomatic. Some patients may complain of palpitations, atypical chest pain, syncope or fatigue. ^[5] Cardiac dysrhythmias commonly associated with MVP are nonspecific and may include both supraventricular (sinus tachycardia, atrial fibrillation, atrial flutter, junctional tachycardia) and ventricular (premature ventricular contractions, ventricular tachycardia) dysrhythmias. ^[6] Association of MVP with WPW syndrome is uncommon. WPW syndrome is a type of pre-excitation syndrome and is caused by the accessory pathway, mainly bundle of Kent joining atria and ventricle bypassing the normal atrio-ventricular pathway. Short PR interval and delta wave is the expression of pre-excitation caused by accessory pathway. Patients with WPW syndrome are known for having life threatening arrhythmias. Atrial fibrillation which can result in ventricular fibrillation and re-entrant tachycardia causing paroxysmal supraventricular tachycardia or ventricular tachycardia are known to occur. Anaesthetic drugs tend to change the electrophysiology of the atrio-ventricular conduction system and hence they tend to affect the behavior of the patient under anaesthesia.

The goals of anaesthetic management in patients with MVP and WPW syndrome are to avoid sudden decreases in heart rate, avoid increase in sympathetic activity, hypovolaemia and minimize drug-induced myocardial depression. Drugs (digoxin, verapamil) that enhance conduction of cardiac impulses through accessory pathways should also be used carefully. These patients may be on beta blockers and antidysrhythmic drugs, which should be continued through the perioperative period. Optimization of electrolytes preoperatively seems prudent to minimize the risk of intraoperative cardiac dysrhythmias. Premedication should produce anxiolysis without causing tachycardia. Scopolamine or glycopyrrolate are preferred to atropine if administration of anticholinergic drugs is deemed appropriate.

There is no clinical evidence that contraindicates the use of regional anaesthesia in patients with MVP. In patients with WPW syndrome, regional anaesthesia is advantageous over general anaesthesia as it avoids multiple drugs and noxious stimulus of laryngoscopy and intubation. Central neuraxial blocks have been found to be advantageous and have been

successfully used to manage various lower abdominal and perineal surgeries. [7, 8, 9] The chances of arrhythmias are increased in general anaesthesia because of pain and lighter plane of anaesthesia.

Induction of anaesthesia can be achieved with propofol, etomidate, thiopental or opioids. Ketamine would be an unlikely option as it increases sympathetic response that could accentuate mitral regurgitation and increases chances of mitral regurgitation. Propofol has no effect on the refractory period of accessory pathways. Etomidate causes minimal myocardial depression and alterations in sympathetic functions. There are no clinical data to support the use of one muscle relaxant over another in the presence of MVP. However drug-induced hemodynamic alterations (vagolysis, histamine release) deserve consideration when selecting specific drugs. Vecuronium and rocuronium being more cardio stable are preferred agents. Pancuronium is undesirable as it causes tachycardia. Atracurium causes histamine release and has less autonomic safety. [10] Volatile anaesthetics combined with nitrous oxide and/or opioids may be useful for attenuation of sympathetic activity. Sevoflurane and isoflurane have no effect on conduction through accessory pathway and are preferred to halothane. [11]

If dysrhythmias do develop under anaesthesia, it is important to exclude the more common causes (blood gas abnormalities, electrolyte disturbances, depth of anaesthesia) before attributing them to MVP or WPW syndrome. [12] If paroxysmal supraventricular tachycardia occurs and the patient is hemodynamically stable then vagal maneuvers, lignocaine, adenosine can be tried. If this is ineffective then procainamide can be an alternative. If still not responding and patient is in atrial fibrillation or hemodynamically unstable direct current cardioversion should be considered. Digitalis and verapamil are strictly contraindicated as they suppress normal pathway and increase conduction through accessory pathway. [6]

In conclusion thorough understanding of pathophysiology of MVP and WPW syndrome, meticulous planning and preparation for management of dysrhythmias is the key to a successful outcome.

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