Intraoperative interfascicular ventricular tachycardia: A rare occurrence

INTRODUCTION

The occurrence of intraoperative wide complex arrhythmias (WCT) can not only lead to haemodynamic compromise but their confounding diagnosis may lead to delay in the start of exact antiarrhythmic therapy. According to recent Cardiopulmonary Resuscitation guidelines,[1] any unidentified WCT with unstable haemodynamics should be cardioverted while a stable WCT may be evaluated by experts for correct diagnosis and exact treatment. In the operation theatre, however, the onus of an early and correct diagnosis of the ongoing WCT into supraventricular or ventricular tachycardia (SVT or VT) may be entirely on the anaesthesiologist.

CASE REPORT

A 66-year-old, 70 kg male patient with a BMI of 33 presented for laparoscopic cholecystectomy. He was a chronic smoker with chronic obstructive pulmonary disease which required salbutamol MDI. He was a hypertensive on treatment with oral ramipril and losartan. He had one episode of acute coronary syndrome 18 months back which required percutaneous intervention with stenting of right coronary artery. Subsequently, the patient was advised to take aspirin and clopidogrel orally which had been stopped 5 days prior to the surgery. History pertaining to other organ systems was unremarkable. On preoperative evaluation he was NYHA class II with decreased effort tolerance (<4 METs). His ECG showed normal sinus rhythm with left anterior fascicular block [Figure 1] while his resting echocardiography showed regional wall motion abnormalities in the right coronary artery territory with an ejection fraction of 40%.

In the operation theatre ECG, non-invasive blood pressure and SpO2 monitor were attached. After intravenous access and radial artery cannulation, anaesthesia was induced with 150 mcg of fentanyl and 100 mg of propofol. 35 mg of atracurium was given to attain muscle relaxation to facilitate tracheal intubation. There was a fall in blood pressure to 70/40 mmHg after induction which was managed with 6 mg of ephedrine. The haemodynamics recovered to normal limits after endotracheal intubation. However, immediately after creation of pneumoperitoneum, the patient developed regular WCT with a ventricular rate of 150/min and a mean blood pressure of 80-90 mmHg. The abnormal heart rhythm immediately reverted back to normal on deflation of pneumoperitoneum. An open cholecystectomy was planned in consultation with the surgeons. Following skin incision the same pattern of WCT recurred. The arrhythmia reverted spontaneously within 3 to 4 min of stopping the surgery. The surgery was abandoned and the patient immediately shifted to ICU without reversal of anaesthesia. In the ICU, the patient had multiple episodes of WCT with severe hypotension which required synchronised DC cardioversion with 100-150 joules biphasic energy. An infusion of dopamine was also started at the rate of 10 mcg/kg/min. Cardiologist was urgently summoned who diagnosed the WCT as interfascicular VT on 12 lead ECG [Figure 2a, b] and thus a loading dose of 150 mg of amiodarone was administered over 10 min intravenously followed by infusion at 1 mg/min for 6 hours and 0.5 mg/min for subsequent 18 hours. Despite the treatment

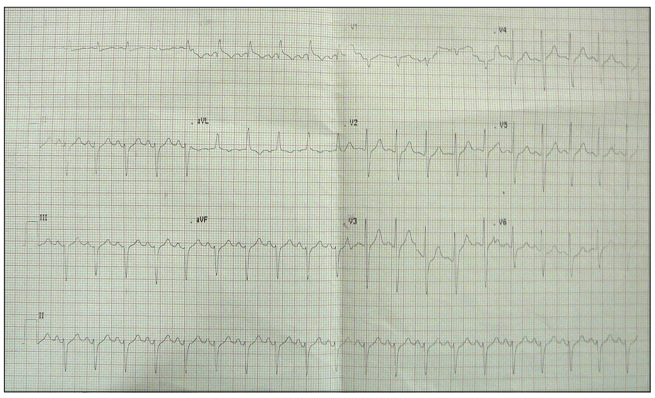

Figure 1: Preoperative twelve lead ECG of the patient

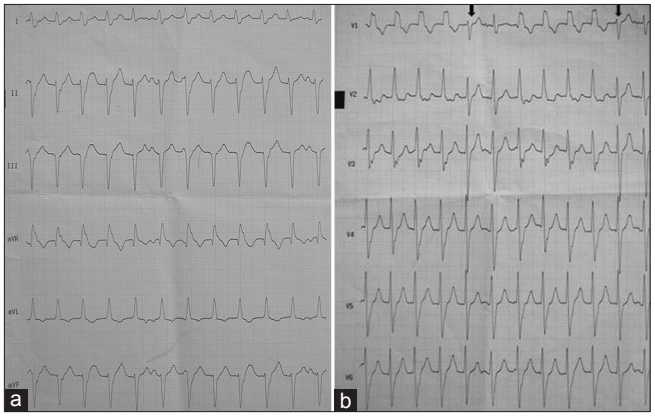

Figure 2: (a and b) Twelve lead ECG taken in the ICU showing interfascicular ventricular tachycardia. Bold arrows show capture beats

the patient had multiple episodes of WCT which required cardioversion again with 150 joules of biphasic energy. All his biochemical parameters like serum electrolytes, renal and liver function tests were normal, and no increase in cardiac enzymes occurred thus ruling out possible myocardial ischemia. An echocardiography could not be done at as facilities of in-bed echocardiography were not available with us. The patient was weaned off inotropes over a period of 6 hours, the tracheal tube was subsequently removed, and he could be discharged from the hospital after 3 days.

DISCUSSION

The appearance of any kind of sustained arrhythmia under anaesthesia requires early diagnosis and accurate management of the arrhythmia, haemodynamic sustenance, and exclusion of ongoing myocardial ischemia simultaneously. In our patient, the WCT was not associated with any haemodynamic instability initially and thus for us the main concern was to accurately identify the arrhythmia and initiate therapy for it. WCT can either be monomorphic VT or SVT with aberrant conduction. In less than 5% of the patients they can be because of accessory conduction pathways leading to pre-excited VT (as seen in Wolff-Parkinson-White syndrome), drug intoxication, dyselectrolytemia, etc.[2] The QRS morphology at the time of tachycardia had a right bundle branch block with left anterior hemiblock pattern. Any WCT with ECG morphology similar to bundle branch block or fascicular block is more suggestive of SVT with aberrancy. However, the ECG also shows RS complexes in precordial leads which are more than 100 ms, A-V dissociation with P-waves not in concordance with QRS complexes and capture beats with almost normal QRS morphology in between the abnormal QRS complexes. These findings are highly suggestive of monomorphic VT.[3] Monomorphic VT with morphology similar to either bundle branches block is because of re-entry phenomenon affecting the His Purkinje system also known as bundle branch re entrant tachycardia. Bundle branch re-entry is the cause of nearly 6% of VT in patients with ischemic heart disease.[4] The more common form of bundle branch tachycardia which involves the left and right bundle branches has antegrade conduction over the right bundle and retrograde conduction over the left bundle and thus has left bundle branch block morphology of the QRS complexes. The tachyrrhythmia in our patient was due to re-entry involving both the fascicles of the left bundle with antegrade conduction over the anterior fascicle and retrograde conduction up the posterior fascicle. Since the ventricles in this kind of re-entry are activated through the anterior fascicle of the left bundle system, the QRS morphology has a right bundle branch block with left anterior hemiblock pattern as was seen in our patient. Interfascicular blocks are one of the most uncommon types of bundle branch re-entry phenomenon tachycardia and have been described very rarely in literature[5] and that too never under anaesthesia. These are also different from another commonly described entity, the fascicular or intrafascicular blocks.[5]

According to 2010 American Heart Association Guidelines,[1] the treatment of any WCT leading to haemodynamic instability is synchronised DC cardioversion while a stable undiagnosed WCT may first be treated empirically with intravenous adenosine. Empirical use of adenosine will convert a SVT to sinus rhythm but it may rarely convert an underlying monomorphic VT to ventricular fibrillation although adenosine rarely causes complications in patients with WCT because of short half life. The immediate diagnosis of any WCT and subsequent respective treatment is always better than giving empirical treatment. Over the years some algorithms have been developed which make the diagnosis of WCT more scientific and straightforward.[6,7] There are some simpler signs for identifying VT e.g. QRS duration more than140 ms in RBBB and more than 160 ms in LBBB suggests VT (Classical Wellen's criteria)[8] and the 'rabbit-ear sign' (VT more likely if left peak is taller). VTs in a structurally damaged heart due to coronary artery disease are due to a combination of multiple factors like ventricular premature beats, variations in the autonomic nervous system, or transient myocardial ischemia.[9] Thus, sympathetic stimulation due to pneumoperitoneum in the first instance and incisional pain during the second instance could have been the causative factor of the interfascicular block intraoperatively while the recurrent episodes of interfascicular block that were seen in the ICU may well have been a result of myocardial hypoxia due to subsequent hypotension. These subsequent episodes may have been avoided if the correct diagnosis had been reached intraoperatively and respective therapy in the form of amiodarone started earlier.

CONCLUSION

To conclude, this case report describes the occurrence of a very rare form of monomorphic VT due to interfascicular block. The treatment modality for such a WCT is amiodarone. The difficulties in diagnosing such a tachycardia intraoperatively have also been discussed. However, until proven otherwise any WCT should be managed as if it were VT because FIRST DO NO HARM.

Vimi Rewari, Rashmi Ramachandran, Neeraj Parakh[1], PM Singh, Sreekumar E Jayanandan

Departments of Anaesthesia and [1]Cardiology, All India Institute of Medical Sciences, Delhi, India

Address for correspondence:
Dr. Rashmi Ramachandran,
31-G, Sector-8, Jasola Vihar, Delhi - 110 025, India.
Email: rashmiramachandran1@gmail.com

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