

PREEXCITATION SYNDROME PRESENTED WITH VENTRICULAR TACHYCARDIA - A CASE REPORT

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Abstract

An adult unconscious patient was brought to intensive care unit (ICU) with impalpable peripheral pulse, non-recordable blood pressure (BP), gross pallor, cyanosis, sweating and gasping respiration along with very rapid and feeble carotid pulse. It revealed ventricular tachycardia on monitor and was revived successfully by immediate direct current (DC) cardioversion along with other resuscitative measures. There was no contributory past history. Subsequent electrocardiogram (ECG) on sinus rhythm was diagnosed as Wolff-Parkinson-White (WPW) syndrome, the most prominent manifestation of preexcitation syndrome, in which the most common tachyarrhythmia is atrio-ventricular reciprocating tachycardia (AVRT). It is classified as orthodromic (more common) or antidromic (less common). Antidromic AVRT is difficult to distinguish from ventricular tachycardia on ECG. Atrial flutter and fibrillation are less common but potentially more serious because they can result in rapid ventricular response rates and, in rare instances, ventricular fibrillation. However, any sustained symptomatic tachyarrhythmia warrants urgent resuscitative electrical and pharmacological maneuver and interventions to restore life, regarding which the health care providers should always remain familiar and updated by Continuing Medical Education (CME).

Key words: Wolff-Parkinson-White syndrome, tachyarrhythmia, preexcitation syndrome.

Introduction

Preexcitation usually refers to early depolarization of the ventricles by an abnormal pathway from atria. Rarely,

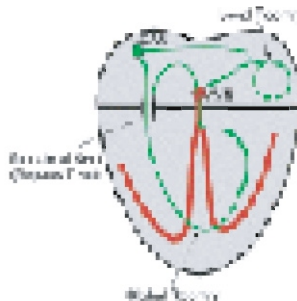


Fig-1: Bundle of Kent

more than one such pathway is present. The most common form of preexcitation is due to the presence of an accessory pathway (bundle of Kent) that connects one of the atria with one of the ventricles (Fig-1)¹.

This abnormal connection allows electrical impulses to bypass the atrio-ventricular (AV) node, thus avoids AV nodal delay, reaches rapidly and depolarizes area of ventricles where the bypass tract ends. The ability to conduct impulses along the bypass tract can be quite

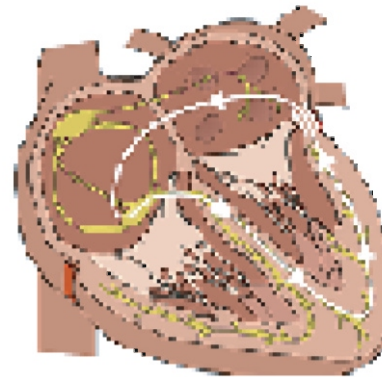


Fig-2: Abnormal pathway in WPW syndrome.

variable and may be only intermittent or rate-dependent. Bypass tracts (Fig-2) can conduct in both direction, retrograde only (ventricle to atrium) or, rarely, only (atrium to ventricle)². The most prominent manifestation of ventricular preexcitation is WPW syndrome³.

Case Report

A 39-year-old male soldier was evacuated from Bangladesh Military Academy (BMA) and directly received in ICU of Combined Military Hospital (CMH) Chittagong Cantonment on 15 March 2009 with history of sudden onset of chest compression, palpitation, shortness of breaths and sweating followed by unconsciousness. On quick assessment, he was found cyanosed with gross pallor and gasping respiration. His peripheral pulses were impalpable and blood pressure (BP) was not recordable. Carotid pulse was very rapid and feeble. On monitor (multi-parameter), his ECG was detected as ventricular tachycardia (Fig-3) and SpO₂ was not accessible.

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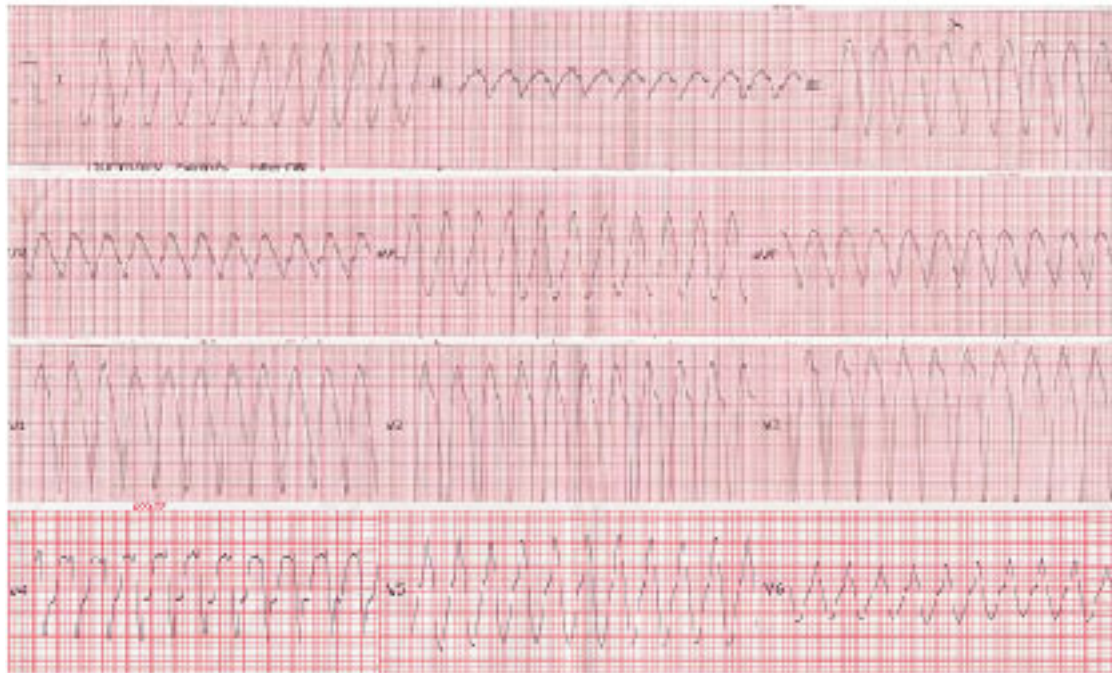


Fig-3: Pre-excitation in the form of ventricular tachycardia.

Immediately the airway and effective bag-mask ventilation with 100% oxygen (O₂) were ensured. An external direct current (DC) cardioversion with 100 joules was performed instantly along with 50 mg intra-venous pethidine and his ECG was resumed on sinus rhythm subsequently. Then as prophylaxis a bolus of 80 mg lignocaine was given intravenously. The patient regained his consciousness after few minutes and his pulse was

found 70 beat per minute and BP was 120/70 mm Hg. He had no contributory past history, a non-smoker, high-average built and is father of two kids.

After getting successive strips of ECG, it was diagnosed as a case of WPW syndrome (Fig-4). He was then managed by oral amiodarone, nitroglycerin, low-dose aspirin, H₂-blocker, sedatives and an antibiotic. His two-



Fig-4: WPW syndrome.

week hospital stay was uneventful and the patient was released from hospital with advice to get further evaluation and definitive treatment by radiofrequency catheter ablation of the abnormal electrical pathway.

Discussion

Preexcitation occurs in approximately 0.3% of general population⁴. Symptomatic tachyarrhythmia associated with WPW syndrome typically begin during early adulthood and pregnancy is associated with the initial

In this case, according to the moribund state of the patient, it was presumed that this ventricular arrhythmia might be following an acute myocardial infarction. So, immediately cardioversion was done and the successful outcome was obtained. Flecainide, propafenone or amiodarone are the prophylactic anti-arrhythmic drug therapy, only indicated in symptomatic patients. The agents those shorten the refractory period like digoxin and verapamil should be avoided⁹. The definitive treatment of choice for symptomatic patients is radiofrequency catheter ablation of the accessory pathway¹⁰.

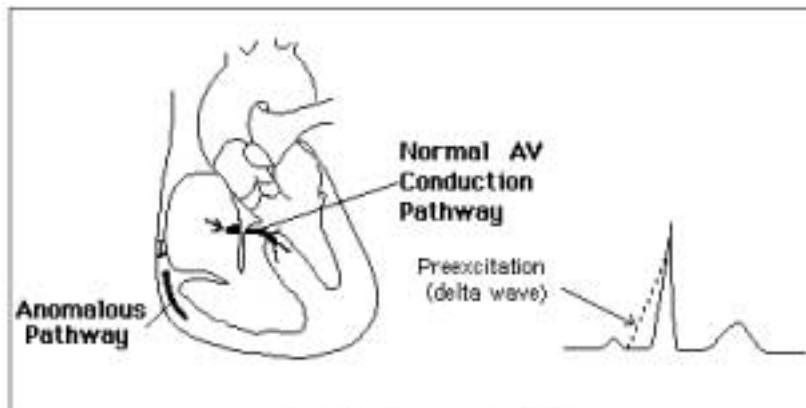


Fig-5: Delta wave in ECG.

manifestation of the syndrome in some women⁵. The first manifestation may appear during perioperative period. In some patients the first manifestation of WPW syndrome is sudden death presumably due to ventricular fibrillation. The estimated incidence of sudden death in patients with WPW syndrome is 0.15% per patient-year⁶. Paroxysmal palpitations with or without dizziness, syncope, dyspnoea, or angina pectoris are common in presence of the tachyarrhythmia. Premature activation of ventricular tissue via the accessory pathway produces a short PR interval in ECG and a 'slurring' of QRS complex, called 'delta wave' (Fig-5). The ECG appearance of this tachycardia may be indistinguishable from that of AV nodal reentry tachycardia (AVNRT) and can mimic bundle branch block, right ventricular hypertrophy, ischaemia, myocardial infarction, and ventricular tachycardia (during atrial fibrillation)⁷.

Carotid sinus pressure or intravenous adenosine can terminate the tachycardia. If atrial fibrillation occurs, it may produce a dangerously rapid ventricular rate and may cause collapse, syncope and even death. It should be treated as an emergency, usually with DC cardioversion⁸.

Conclusion

Preexcitation akin various morbid tachyarrhythmias reflect on monitor. Quick and correct recognition of cardiac dysrhythmia is the hallmark of managing the critically ill patients in intensive care settings. Health care providers, specially paramedics should be thoroughly conversant, familiar and updated to the management of moribund patients by continuing medical education and bedside clinics.

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