

## WPW Syndrome Presented with Ventricular Tachycardia – A Case Report

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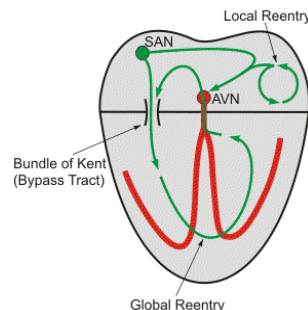
**About the Author:** Dr (Lt Col) Md Rabiul Alam, MBBS, MCPS, FCPS (Anaesthesiology) was at Armed Forces Medical College, Dhaka as Assistant Professor and now he is working at Combined Military Hospital, Chittagong, Bangladesh as Classified Anaesthesiologist. His areas of interests are casualty, medevac, intensive & palliative care.



**Abstract:** Intensivists come across moribund patients with preexcitation syndrome not very infrequent in intensive care units. WPW syndrome is the most common form and usually present with supraventricular tachyarrhythmia. This was a fatal case presented with ventricular tachycardia without any known illness and after revival it was detected as WPW syndrome.

**Key words:** WPW syndrome, Ventricular tachycardia (VT)

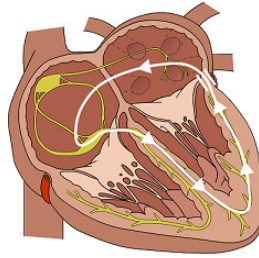
**Introduction:** Preexcitation usually refers to early depolarization of the ventricles by an abnormal pathway from atria. Rarely, more than one such pathway is present. The most common form of preexcitation is due to the presence of an accessory pathway (bundle of Kent) that connects one of the atria with one of the ventricles (Figure-1)<sup>1</sup>.



**Figure 1.** Bundle of Kent

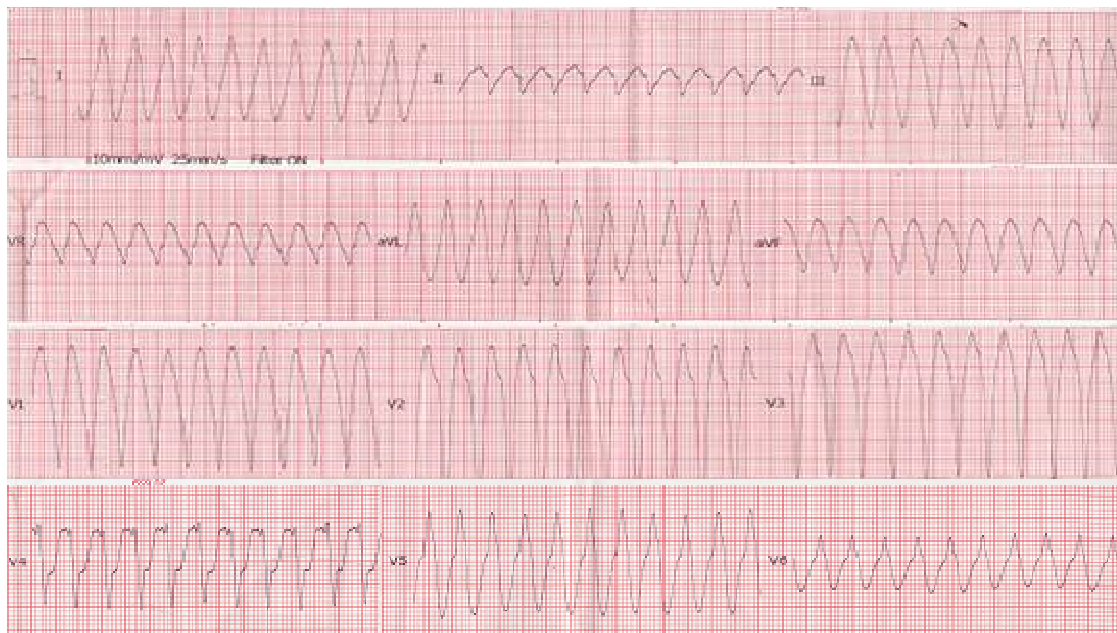
This abnormal connection allows electrical impulses to bypass the AV node, thus avoids AV nodal delay, reaches rapidly and depolarizes area of ventricles where the bypass

tract ends. The ability to conduct impulses along the bypass tract can be quite variable and may be only intermittent or rate-dependent. Bypass tracts can conduct in both direction, retrograde only (ventricle to atria) or, rarely, anterograde only (atrium to ventricle)<sup>2</sup> (Figure-2). The most prominent manifestation of ventricular preexcitation is Wolff-Parkinson-White (WPW) syndrome<sup>3</sup>.



**Figure 2:** Abnormal Pathway in WPW Syndrome

**Case Report:** A 39-year-old soldier was evacuated from on duty and directly received in ICU with history of sudden onset 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 BP was not recordable. Carotid pulse was very rapid and feeble. On monitor, his ECG was detected as ventricular tachycardia (Figure-3) and SpO<sub>2</sub> was not accessible.

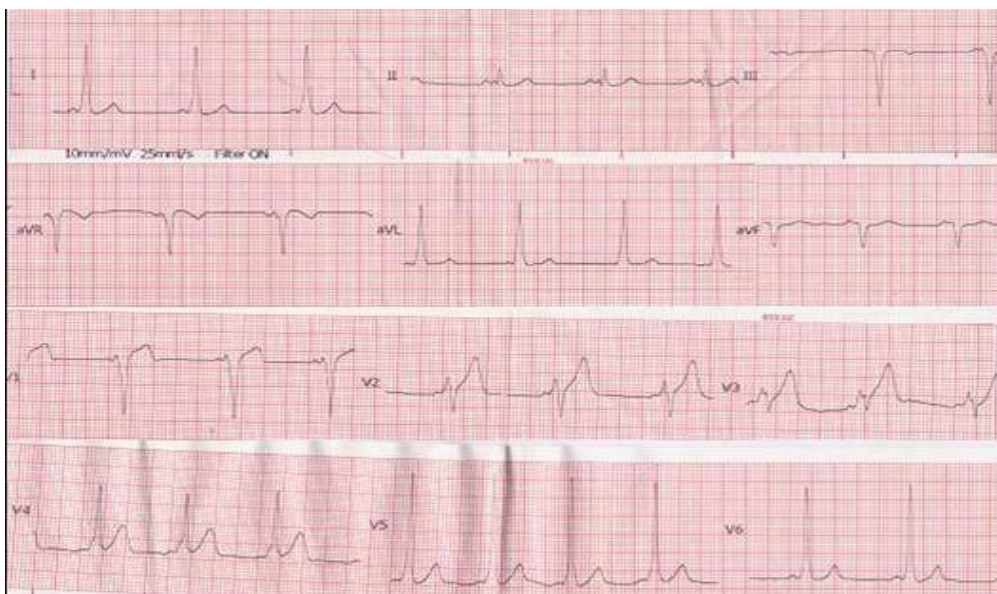


**Figure 3:** Preexcitation in the form of Ventricular Tachycardia

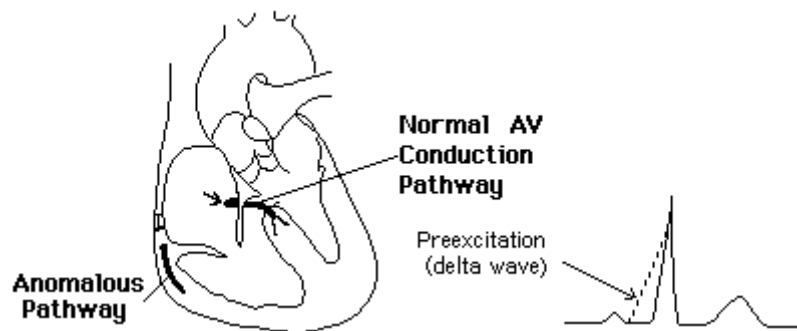
Immediately the airway and effective bag-mask ventilation with 100% O<sub>2</sub> were ensured. An external DC cardioversion with 100 joules was performed instantly along with

50 mg i.v. 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 bpm and BP was 120/70 mmHg. 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 (Figure-4). He was then managed by oral amiodarone, nitroglycerin, low-dose aspirin, H<sub>2</sub>-blocker, sedatives and an antibiotic. His two-week hospital stay was uneventful and he is now planned 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 population<sup>4</sup>. Symptomatic tachy-arrhythmias associated with WPW syndrome typically begin during early adulthood; and pregnancy is associated with the initial manifestation of the syndrome in some women<sup>5</sup>. 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-year<sup>6</sup>. Paroxysmal palpitations with or without dizziness, syncope, dyspnoea, or angina pectoris are common in presence of the tachyarrhythmias. 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' (Figure-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)<sup>7</sup>.



**Figure 4:** Just after Cardioversion it appeared as WPW Syndrome



**Figure 5:** Delta Wave in ECG

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 cardioversion<sup>8</sup>. 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 avoided<sup>9</sup>. The definitive treatment of choice for symptomatic patients is radiofrequency catheter ablation of the accessory pathway<sup>10</sup>.

**Conclusion:** Preexcitation akin various morbid tachyarrhythmias on monitor. Quick and correct recognition of cardiac dysrhythmias 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 bed-sides clinics.

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