



INTRODUCTION

WPW syndrome is a condition in which an accessory tract (Bundle of Kent) bypasses the normal cardiac conduction system, resulting in aberrant electrical conduction. This accessory pathway permits cardiac electrical activity to circumvent the atrioventricular node conduction delay and arrive at the ventricle early, resulting in premature ventricular depolarization. This preexcitation also bypasses the fast-conducting His-Purkinje pathway, resulting in ventricular depolarization that occurs early but spreads slowly.

Patients with WPW may develop paroxysmal atrial fibrillation, which is frequently overlooked by physicians in the Emergency Department. Reversing this with manoeuvres by administering with AV-nodal blocking agents may result in fatal arrhythmias.

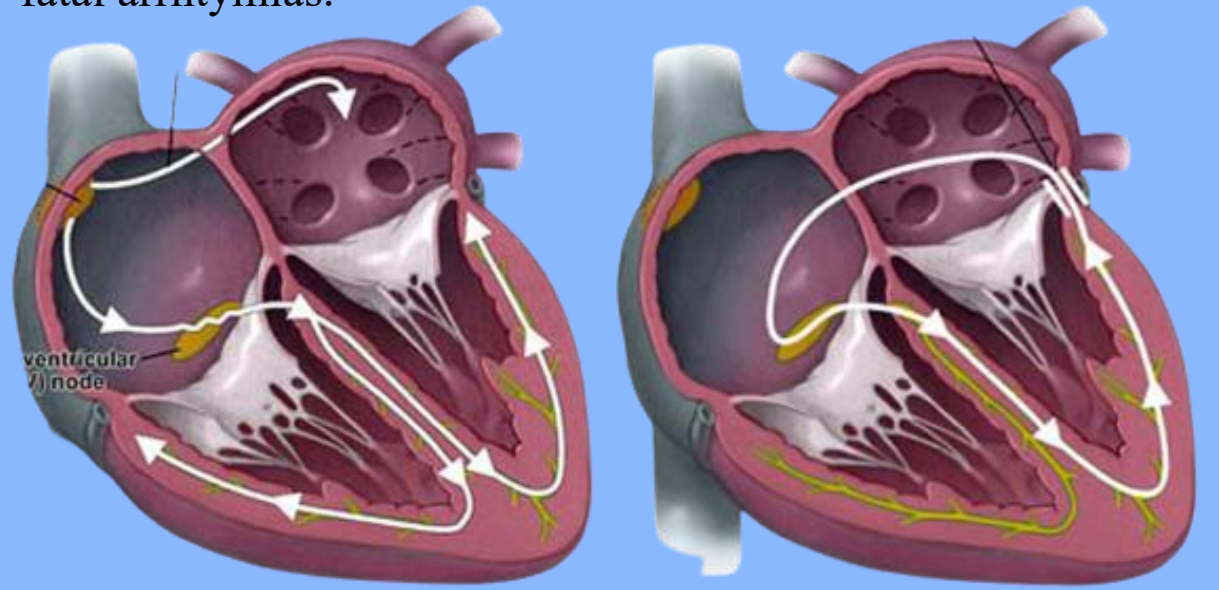


Figure 1: Conduction via an accessory tract bypassing normal AV node resulting in premature ventricular contraction (shortened PR interval & delta wave). This preexcitation also bypasses the His-Purkinje pathway and results in early but slowly propagated ventricular depolarization (wide QRS complex)

HISTORICAL BACKGROUND

- 1930 : Louis Wolff, Sir John Parkinson, and Dr. Paul Dudley White published a case study of 11 individuals with paroxysmal tachycardia & an underlying ECG of sinus rhythm with short PR and bundle branch block/wide QRS.
- 1943 : Preexcitation electrocardiographic characteristics were first linked to anatomic evidence of abnormal conducting tissue or bypass pathways.

CASE REPORT

A 27-year old gentleman with no underlying heart diseases, presented to the ED with palpitation, lightheadness, and chest discomfort. Upon arrival, he was alert and not in respiratory distress. Vital signs demonstrated blood pressure 108/75 mmHg, heart rate 220 beats/min, and saturation 98% under room air. Radial pulses were exceedingly rapid and fairly regular.

Electrocardiogram demonstrated an irregular, broad QRS complexes with varying morphologies, and rapid ventricular responses. This was initially misinterpreted as supraventricular tachycardia with aberrancy by the attending doctor.

Attempts at carotid massage and Valsalva's manoeuvre were unsuccessful. The decision to administer intravenous Adenosine was nearly made, however the diagnosis was revised following senior consultation. A 120 Joule synchronised cardioversion was performed, and the rhythm was reverted to sinus. Narrow PR interval, Delta waves, and broad QRS complex which are indicative of WPW syndrome, were revealed on repeated ECGs.

Patient was admitted to the cardiology intensive care unit for close monitoring, and oral Flecainide was initiated. He was scheduled for an electrophysiological study and radiofrequency ablation.

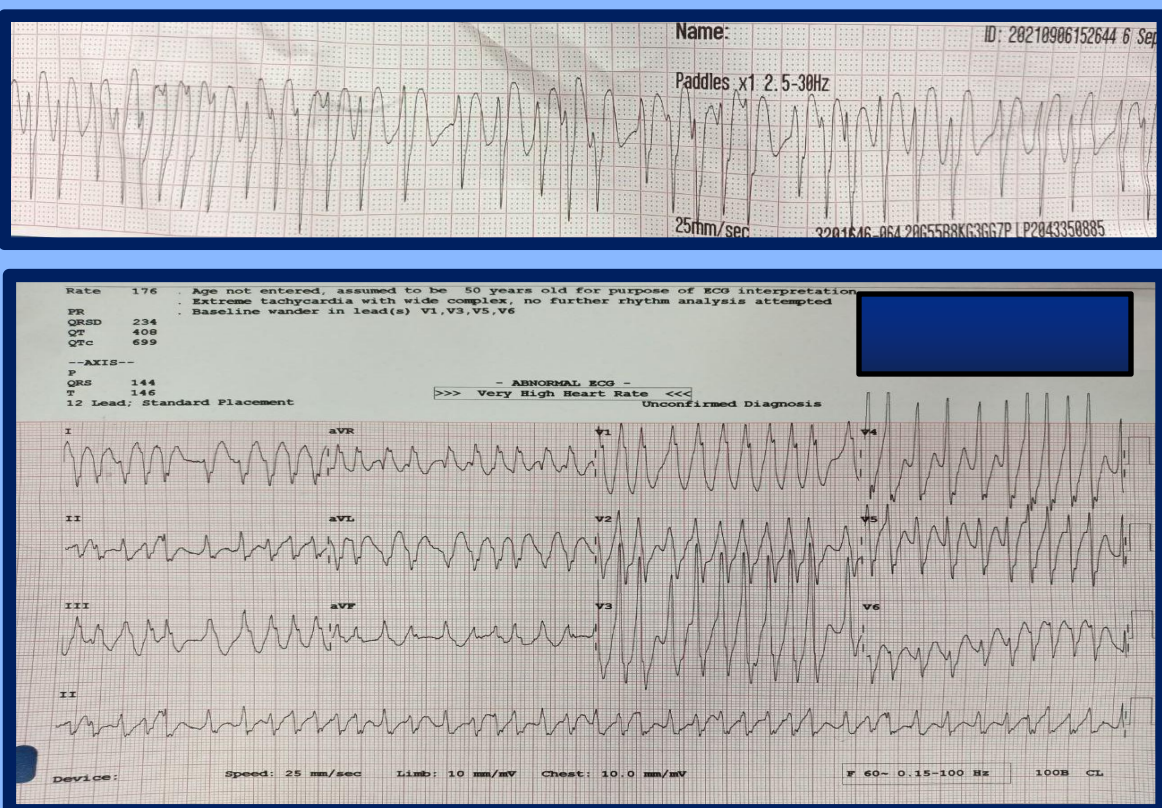


Figure 2a & 2b : Cardiac monitor & ECG reveals AF with fast ventricular response with varying QRS morphologies

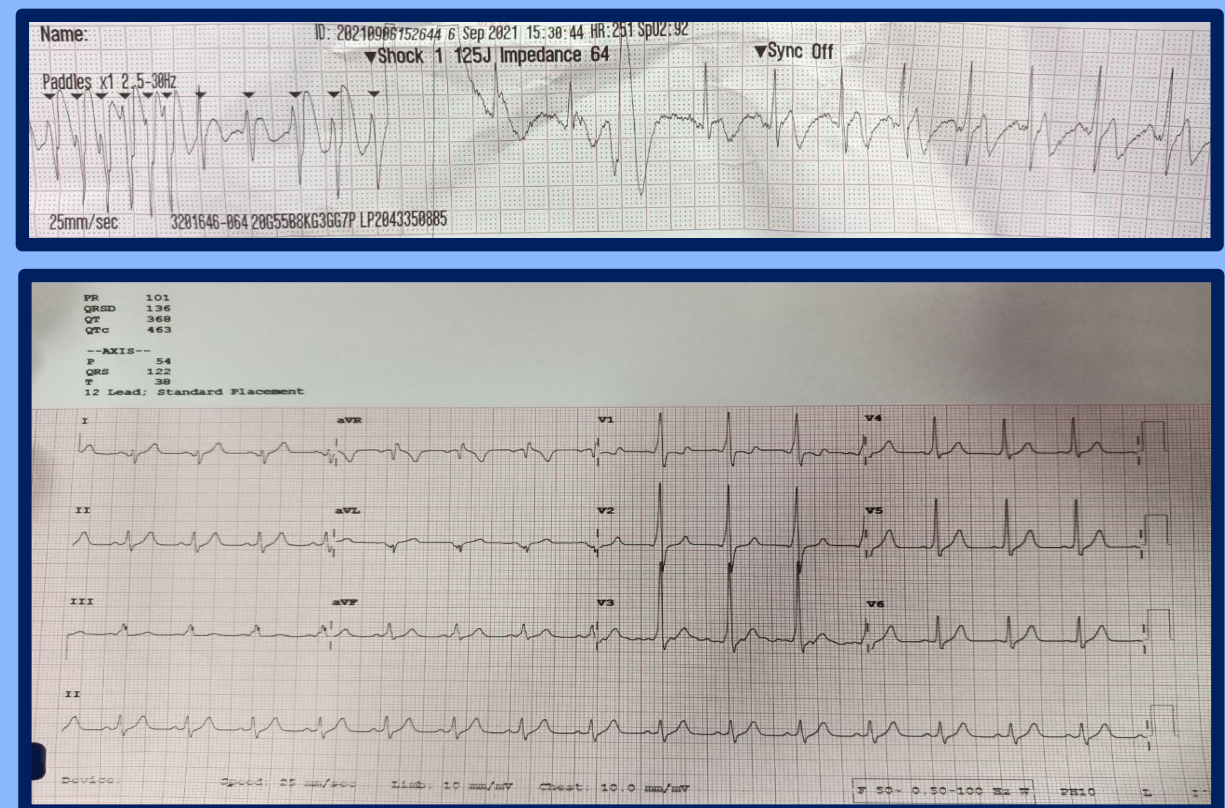


Figure 3a & 3b : Post-cardioversion-cardiac monitor & ECG reveals sinus rhythm with shortened PR, delta wave & wide QRS

DISCUSSION / CONCLUSION

One relatively rare ECG that you must recognize- or you may be fooled into initiating wrong treatment, which can potentially be fatal. Vagal stimulation, induced by carotid sinus massage and Valsalva's manoeuvre causing atrial desynchronization, dispersion of atrial refractoriness, and intraatrial reentry. Several reports revealed orthodromic tachycardia in WPW patients reverted to atrial fibrillation after performing these manoeuvres. The effects of vagal stimulation on patients with atrial fibrillation in WPW is uncertain.

The cornerstone management is intravenous procainamide and synchronized cardioversion is used for unstable patient. Radiofrequency ablation of the accessory pathway is another option to reduce the risk of life-threatening arrhythmias.

DISCLOSURES

ACKNOWLEDGEMENT

We would like to thank the Director General of Health, Malaysia for publication of this poster.

CONFLICT OF INTEREST FOR ALL AUTHORS

None declared.

AVOID AV-NODAL BLOCKING AGENTS

- A Amiodarone, Adenosine
- B Beta-blockers
- C Calcium Channel Blockers
- D Digoxin

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