

CLINICAL VIGNETTE

Irregularly Irregular Wide Complex Tachycardia (sometimes narrow)

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Question:

A 38-year-old male with a long history of palpitations complains of dizziness and racing heart. An EKG is obtained (see Figure 1).

What is the diagnosis?

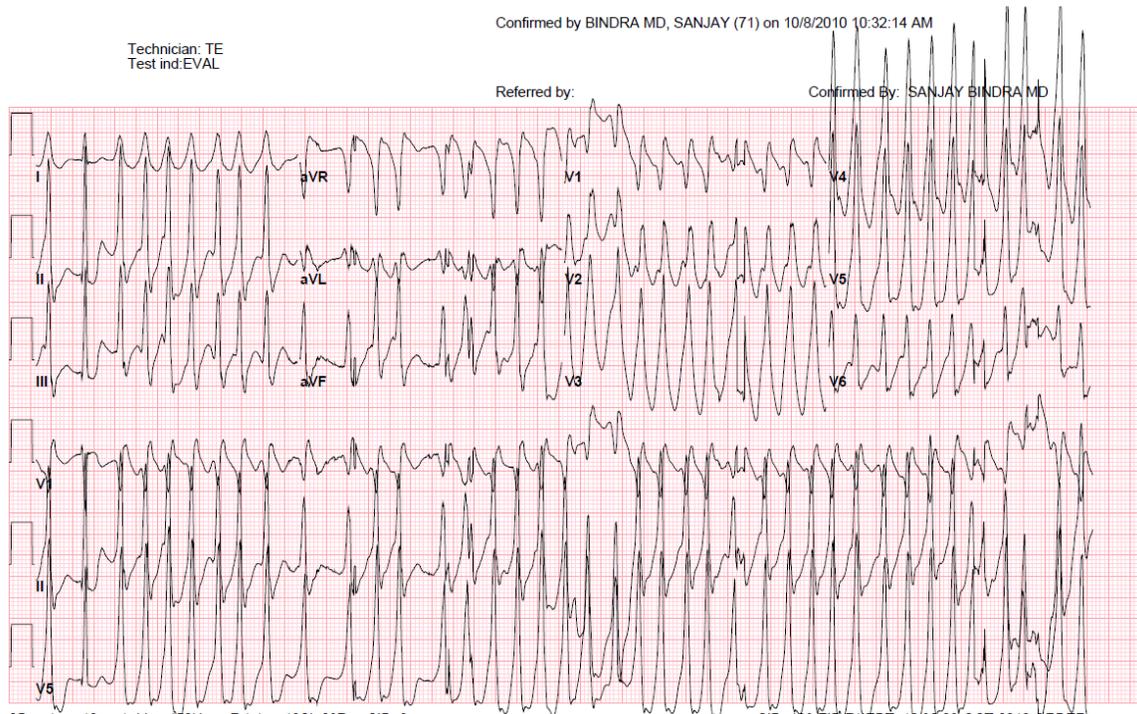


Figure 1.

Answer:

Atrial fibrillation with WPW (Wolff-Parkinson-White) with rapid ventricular response.

Figure 1, EKG reveals Irregularly irregular, wide-QRS complex tachycardia with R-R intervals varying from 0.20 to 0.48 second, representing a rate range of 125 to 300/minute.

Three types of different QRS complexes are seen:

1. One narrow, normal QRS complex marked with, normal conduction through the AV node.
2. Few broad bizarre QRS complexes marked with, fusion complexes formed due to conduction over both AV nodal and bypass tract.
3. Rest of the QRS complexes are wide and of similar shape, due to antegrade conduction through the bypass tract, thereby producing delta wave.

Treatment:

Since the patient was haemodynamically unstable, he was managed with synchronized cardioversion at 200 Joules. The arrhythmia terminated, and subsequent 12-lead electrocardiogram revealed features suggestive of WPW type of pre-excitation syndrome (see Figure 2):

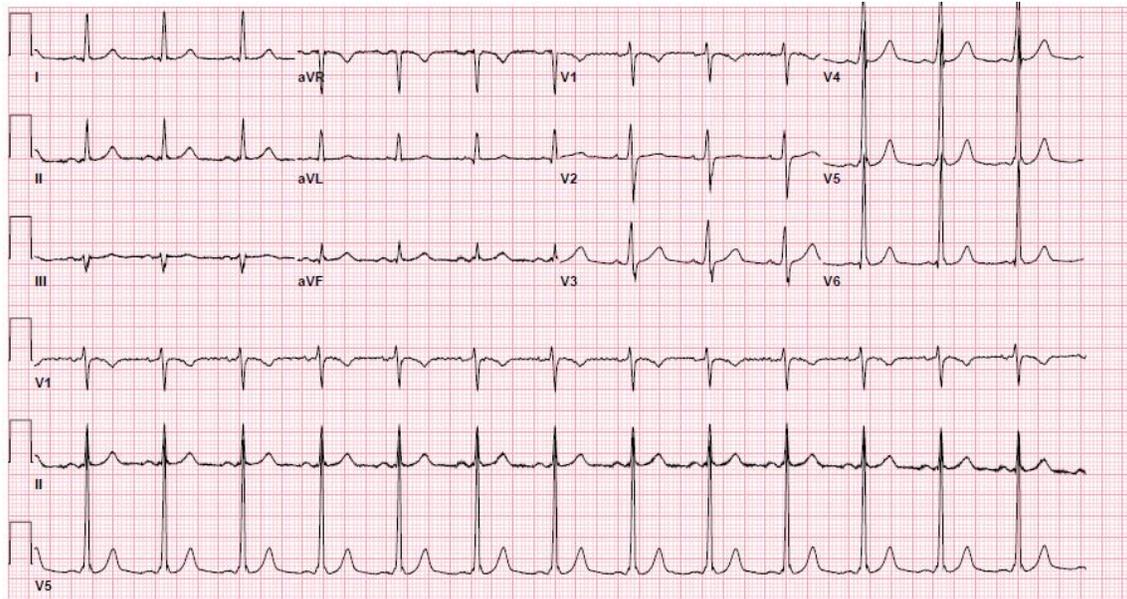


Figure 2: EKG post cardioversion (subtle delta waves in V4,V5)

Figure 2

1. A slurred, initial upstroke of the QRS complex, which is termed as the delta wave. Delta wave seen in V4, V5.
2. A relatively normal, narrow ensuing terminal QRS
3. PR interval (not short since left lateral pathway).

Discussion:

Atrial Fibrillation with accessory pathway (WPW Syndrome).

In 1930, Wolff, Parkinson and White reported 11 cases of young, healthy patients with normal hearts who presented with widened QRS, abnormally short P-R intervals, and paroxysms of tachycardia, including supraventricular tachycardia, paroxysmal atrial fibrillation and atrial flutter¹.

Atrial fibrillation in patients with WPW Syndrome is potentially lethal arrhythmia due to its potential to deteriorate into ventricular fibrillation (Vfib)².

In normal patients without an accessory pathway, the heart is protected from exceptionally high ventricular rates by the relatively long refractory period of the AV node. This generally limits the maximum ventricular rate.

In patients with WPW, short anterograde refractory period of the accessory pathway, may allow faster transmission of impulses from the atrium and correspondingly higher ventricular rates can be reached. The rapid ventricular rate may not allow for adequate diastolic filling of the ventricle and this in turn can predispose to hypotension. In addition, sympathetic discharge secondary to

hypotension can lead to an even shorter refractory period of the accessory pathway and subsequently increase the ventricular rate further. If the ventricular rate becomes too high, this can predispose to Vfib¹.

Afib usually does not conduct at a rate of more than 180 bpm through the normal AV node. On the other hand, conduction through an accessory pathway often results in more rapid ventricular rates. This usually appears as a bizarre, wide complex, irregular tachycardia on ECG with rates often in the 250 bpm range or higher as was noted in this patient.

Treatment of Atrial fibrillation associated with WPW is necessarily different than for a patient without an accessory pathway.

In WPW associated Atrial fibrillation, the goal is to prolong the anterograde refractory period of the accessory pathway relative to the AV node. This slows the rate of conduction through the accessory pathway, thus the ventricular rate slows down.

In patients with non WPW associated Afib, the goal is to increase the refractory period of the AV node (slow conduction).

Standard rate control by drugs that prolong the refractory period of the AV node (e.g., calcium channel blockers, beta-blockers, digoxin, and even adenosine) conversely result in a higher rate of transmission through the accessory pathway and paradoxically increase the ventricular rate. This could have dangerous consequences possibly causing the arrhythmia to deteriorate into Ventricular fibrillation. Thus, such drugs are contraindicated in WPW associated Afib.

Antiarrhythmic drugs: Amiodarone though recommended may accelerate ventricular rate or fibrillation and hence should be avoided as well^{3,4}. Lidocaine administration generally has no significant effect or produces acceleration of ventricular response during AF, hence is unlikely to have beneficial effects and may be deleterious⁵.

If the patient is hemodynamically *stable*, pharmacologic conversion with ibutilide, a class III antiarrhythmic drug available for intravenous administration⁶ or procainamide⁷ may be attempted.

Ibutilide (1mg iv over 10 minutes) with monitoring of QT intervals for 6 hours post infusion. There is a 8% risk of torsades with ibutilide infusion.

Procainamide at 30 mg/min with a maximum dose of 17 mg/kg iv infusion. Ibutilide is preferred because procainamide takes a long time to achieve therapeutic levels (about 60 minutes) and is associated with hypotension during rapid infusion.

If the patient is *unstable*, with evidence of hypoperfusion, primary synchronized cardioversion should be the first-line of treatment (this patient was successfully cardioverted).

In this case, the patient was deemed unstable and treated with synchronized cardioversion. His post cardioversion ECG is shown below exhibiting the delta wave of WPW syndrome. He subsequently underwent EP guided radiofrequency catheter ablation of the accessory pathways (left lateral and left posterior).

Conclusion:

Pre-excited atrial fibrillation is a well recognized cause of sudden cardiac death, for which there is a potential “cure” in the form of radiofrequency ablation of the pathway.

In patients presenting with hemodynamic instability secondary to atrial fibrillation with wpw, prompt synchronized cardioversion is the favored strategy. For hemodynamically stable patients pharmacotherapy with agents like ibutilide or procainamide may be utilized.

REFERENCES

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Submitted on March 31, 2011