

PHOTO QUIZ

Worsened Dysrhythmia after Chemical Cardioversion with Digoxin; a Case of Malpractice

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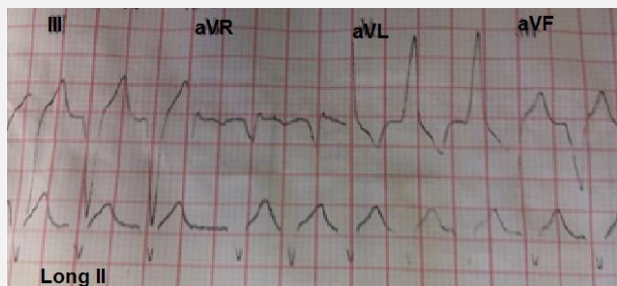


Figure 1 [↑](#)

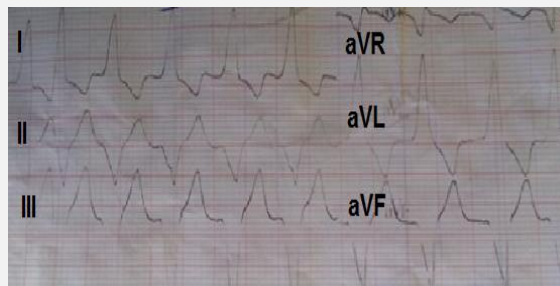


Figure 2 [↑](#)

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Case presentation:

The patient was a 23-year-old man referred to the emergency department (ED) with the chief complaint of palpitation. The patient experienced dizziness, cold sweating, and lightheadedness after getting up which started spontaneously. He had four episodes of the same problems in seven months ago that felt better after taking 10 mg propranolol. However, in the current episode his problem was not solved by the same medication. He had no history of smoking, substance abuse, medication use, congenital heart disease, syncope, previous surgery, chest trauma, or any other known medical problems. As well, he had no any positive history of the same problems in his family. The patients' on-arrival vital signs were as follow: systolic blood pressure (SBP): 90 mmHg, pulse rate (PR):

150/minute, respiratory rate (RR): 14/minute, oral temperature: 37°C, oxygen saturation 96% with nasal cannula and 100% oxygen, Glasgow coma scale (GCS) 15/15. He was not experienced any other concomitant problems such as ischemic chest discomfort, shortness of breathing, or sign of circulatory shock such as paleness, mottling, etc. On general physical examination the patients' lung and heart sounds, four limbs pulses, and capillary refill were normal. As well, focused neurological and abdominal examinations did not have any positive finding. The patient underwent close cardiac, vital sign monitoring and electrocardiography (ECG). [Figure 1](#) shows the on-arrival patients' ECG. Atrial fibrillation (AF) was diagnosed by the corresponding physician and digoxin (!?) prescribed that led to severe lethargy, weakness, sweating, and bradycardia. [Figure 2](#) shows the post medication ECG of patient.

What is your diagnosis?

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Diagnosis:

Wolff-Parkinson-White (WPW) syndrome + Atrial Fibrillation (AF)

Case fate:

The patient was admitted to CCU and after administration of Procainamide, his symptoms revealed (Figure 3) and he was sent for catheter ablation.

Discussion:

According to American Heart Association (AHA) 2010 guideline for cardiopulmonary resuscitation (CPR), if the patient who manifested by tachycardia is presented with severe signs and symptoms including ischemic chest discomfort, acute heart failure, acute altered mental status, sustain hypotension or other signs of shock, considered as unstable case and needs immediate cardioversion. However, in other symptomatic stable ones the subject presented with irregular wide complex tachycardia, then avoiding atrioventricular (AV) nodal blocking agent is crucial. These agents such as β -adrenergic blockers, calcium channel blockers, and Digitalis (summarized as BCD drugs) will not be effective, cannot block the conduction over the accessory pathway, and may even enhance conduction in hypotension or other dangerous outcomes such as ventricular fibrillation (VF) and cardiac arrest. Consequently, these drugs are contraindicated in such situations. Lidocaine is not useful for this purpose and may rarely exacerbate the conduction. Intravenous Procainamide is the treatment of choice and amiodarone may be a favorable second option in spite of some opposite literatures. Patients, who are hemodynamically unstable, clearly require electrical cardioversion following current instructions (1-3).

Wolff-Parkinson-White (WPW) syndrome is used for pre-excitation on ECG accompanied by paroxysmal tachycardia that may be conducted antegrade over the normal AV system or retrograde through the accessory pathway (AP). Antegrade type is characterized by normal and narrow QRS complexes, regular rhythm, ventricular rate of 150-250 beats/minutes, and sudden onset and abrupt termination. The classic electrocardiographic presentation of WPW syndrome consists of short PR interval, slurred and thickened initial upstroke of the QRS complex which is termed as delta wave, relatively normal-narrow terminal QRS deflexion which sometimes referred as the main QRS deflexion, slight widening of the QRS deflexion, and secondary ST

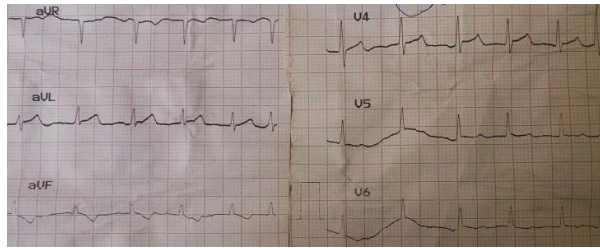


Figure 3 [↑](#)

segment and T wave changes (4, 5).

Although the incidence of dysrhythmias in WPW cases is rare, approximately 80% of dysrhythmias are AV reentrant tachycardia, 15-30% atrial fibrillation (AF), and 5% atrial flutter. AF is considered as an emergent situation when rapid antegrade conduction over an AP occurs in WPW syndrome. Clues in the ECG that may help suggesting WPW+AF include rhythm irregularity, a rapid ventricular response (often with R-R intervals approaching 300 beats/minutes), wide, and bizarre QRS complexes (6, 7).

Szumowski et al. found that age, gender, and the history of syncope are independent risk factors of AF in patients with WPW syndrome and also mentioned that antegrade conduction via AP has a major importance in the development of AF (8). Hamada et al. mentioned that there are two mechanisms of paroxysmal AF in patients with WPW syndrome: one mechanism is reversible and AP-dependent atrial vulnerability, and the other one is intrinsic and AP-independent atrial vulnerability (9).

During AF developing, the custom rate-limiting impression of the AV node will be bypassed, and the consecutive excessive ventricular rates (even 200-240 beats/minutes) may lead to disastrous consequences such as ventricular fibrillation (VF), syncope, and sudden death. Those who have survived from VF dysrhythmia, presented a rapid pre-excited ventricular response as it was demonstrated during the induction of AF in electrophysiological study (2). Sudden cardiac death and other possible dangerous outcomes in patients suffer from symptomatic arrhythmias can be preventable. If they are able to tolerate a rapid pre-excited ventricular response during AF (shortest pre-excited RR interval is less than 260 ms), accessory pathway catheter ablation or administration of antiarrhythmic drugs should be considered instead of such an invasive method to prevent conduction over the accessory pathway (2, 3).

It could be concluded that sometimes no cure is bet-



ter than wrong treatment. Without hurry, consultant with other colleagues and adhering to present guidelines in approach to dysrhythmias can be helpful and even lifesaving.

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Conflict of interest:

There was no conflict of interest.

Author's contribution:

All authors contributed in drafting/revising the manuscript, study concept or design, and interpretation of data.

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