Wolf-parkinson-white Syndrome Presented with Broad QRS Complex Tachycardia

Daulat Manurung, M. Yamin

ABSTRACT

Broad QRS complex tachycardia is tachycardia with widened QRS complex more than 12 s and caused by various mechanisms, either supraventricular or ventricular. It is important to differentiate between ventricular and supraventricular because it will determine treatment and prognosis of patients. We report a case which was referred to us and first diagnosed as ventricular tachycardia but happened to be atrial fibrillation with RBBB. On ECG examination we found irregular broad complex of tachycardia, RBBB, extreme right axis and heart rate 170-180 beat/minute. Intravenous bolus of 300 mg amiodarone was administered within 30 minutes and continued with 900 mg/24 hours. During administration of amiodarone, heart rhythm was converted to sinus rhythm with short PR interval (0.09 s), left axis deviation, and positive delta wave at lead V1. The final diagnosis of wolf-parkinson-white (WPW) syndrome was then confirmed.

Key words: QRS complex tachycardia, diagnosis, wolf-parkinson-white (WPW) syndrome.

INTRODUCTION

Broad QRS complex tachycardia is tachycardia with widened QRS complex more than 12 s and caused by various mechanism, either supraventricular or ventricular. It is important to differentiate between ventricular and supraventricular because it will determine treatment and prognosis of patient.

Twelve lead ECG examination is a main diagnosis tool including heart rate, rhythm, axis and morphology of QRS complex.

We report a case which was referred to us and first diagnosed as ventricular tachycardia but happened to be atrial fibrillation with RBBB. On ECG examination we found irregular broad complex of tachycardia, RBBB, extreme right axis and heart rate 170-180 beat/minute. Intravenous bolus of 300 mg amiodarone was administered within 30 minutes and continued with 900 mg/24 hours. During administration of amiodarone, heart rhythm was converted to sinus rhythm with short PR interval (0.09 s), left axis deviation, and positive delta wave at lead V1. The final diagnosis of wolf-parkinson-white (WPW) syndrome was then confirmed.

CASE ILLUSTRATION

A 65-year-old gentleman was referred to ER for further management of his ventricular tachycardia. He had no symptoms until 9 hours before admission when he felt a sudden onset of palpitation and accompanied by chest pain and sweating. He then visited a physician at a private hospital who diagnosed him of having ventricular tachycardia. He had never experienced such a bad palpitation before. He had no history of near-or syncopal attack and had no family history of sudden cardiac death. One week prior to his palpitation, he had itchy rashes on his left thigh and a doctor whom he saw made a diagnosis of herpes zoster and type 2 diabetes, which he was not aware of previously. He was then treated by the doctor.

He was known to have had hypertension in the past 5 years and he had not been compliant to the treatment. In the year 2000, he had acute myocardial infarction and warded at a government hospital. Unfortunately, he could not recall all the data and recommendation of treatment offered to him. This gentleman has been retired as a security guard since 3 years ago.

On admission, he was in distress because of palpitation. On physical examination, he looked pale. The temperature was 36.7 C and the blood pressure was 100/70 mmHg; the pulse was irregular with the rate of 180 beats per minute and respiration was normal. The remainder of examination was normal. The ECG showed an irregularly irregular, broad QRS complex tachycardia with right bundle branch block (RBBB) morphology, and
extreme right axis, and the rate of 170-180. (Figure 1) The results of routine laboratory tests, including a complete blood count and liver- and renal-function test, and levels of serum electrolytes and cardiac biomarkers were normal except random blood sugar of 315 mg/dL. The chest x-ray was within normal limit. The echocardiogram revealed structurally normal heart.

The working diagnosis made was atrial fibrillation with right bundle branch block (RBBB) and type 2 diabetes. Intravenous bolus of 300 mg amiodarone was administered within 30 minutes and continued with 900 mg/24 hours. Sliding scale protocol of regular insulin was applied for his diabetes. During administration of amiodarone, his rhythm was converted to sinus rhythm with short PR interval (0.09 sec), left axis deviation, and positive delta wave at lead V1. (Figure 2) The final diagnosis of wolf-parkinson-white (WPW) syndrome was then confirmed.

DISCUSSION

The differential diagnosis of irregular broad complex tachycardia (QRS duration >120 msec) includes atrial
fibrillation with bundle branch block or atrial flutter/atrial tachycardia with variable block and bundle branch block, atrial fibrillation or atrial flutter/tachycardia with variable block, and antegrade conduction over an accessory pathway. The accessory pathway has been existing since birth (congenital in origin) and consists of myocardial muscle structure and located anywhere along the atrioventricular groove. In this patient, the broad QRS tachycardia was very irregular; thus the most possible diagnoses were AF with RBBB or AF with preexcitation (preexcited AF) over an accessory pathway. From the ECG noted, a conduction rate of 200 msec (300 BPM) sometimes occurred (arrowhead of figure 1). This evidence excluded AF with RBBB since AV node was not capable of conducting impulse in such a very fast fashion. The only possibility for this conduction to occur is via an accessory pathway which has an all-or-none characteristics. The other clue was the presence of a normal QRS complex noted (arrowhead) among the broad QRS complexes; this indicates that at the time the impulse reached the accessory pathway, it has not yet recovered from refractory period of previous impulse. Thus, the impulse was then traveled down via normal conducting tissue (AV node and his bundle route) and gave normal QRS morphology. Atrial tachycardia or flutter with variable block and antegrade conduction over an accessory pathway is very unlikely since the irregularity was very prominent in this ECG.

The ECG during sinus rhythm confirmed the diagnosis of WPW syndrome which included short PR interval < 0.12 second, delta wave, and prolonged QRS complex >0.11 sec. Paroxysmal AF is a common arrhythmia in patients with WPW syndrome and is observed in up to one third of patients. It is observed that approximately 50% of patients with ECG feature of WPW are asymptomatic. However, patients with WPW syndrome are at risk should they develop ventricular fibrillation (VF) as the accessory pathway can conduct more rapidly than the AV node and very rapid ventricular rate can degenerate into ventricular fibrillation. In acute condition, patients are usually unstable due to very rapid ventricular rate and are required immediate electrical cardioversion. In more stable patients, procainamide, propafenone, ibutilide, or flecainide could be administered. Amiodarone and sotalol can also be used since they affect the conduction of both AV node and accessory pathway. AV nodal blocking agents such as calcium channel blocker, digoxin, and ß-blocker should be avoided in AF with pre-excitation since it slows down conduction through AV node and therefore, enhances the conduction over the accessory pathway. This will subject the patient into fatal ventricular fibrillation.

This patient is considered at high risk for sudden death since the shortest RR interval during AF < 250 ms (heart rate >240 beat per minute). Definitive treatment of choice is radiofrequency ablation with success rate of 98% and low complication rate (0.6%).

CONCLUSION

We report a case of wide QRS complex tachycardia first diagnosed as ventricular tachycardia. Reassessment of ECG revealed atrial fibrillation with RBBB. After treatment with amiodarone, the ECG pattern showed sinus rhythm, shorter PR interval, left axis deviation and delta wave at precordial lead V1. Final diagnosis of patient was wolf-parkinson-white syndrome.

REFERENCES