

# Complete Atrioventricular Block-Induced Torsade de Pointes, Manifested by Epilepsy

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Complete atrioventricular (AV) block is frequently regarded as a cause of informed syncopal attacks, even though the escape rhythm is maintained. Torsade de pointes (TdP) may be a significant complication of AV block associated with QT prolongation. Here, we report the case of a 42-year-old female who was referred to our hospital due to recurrent seizure-like attacks while taking anti-convulsant drugs at a psychiatric hospital. TdP with a long QT interval (corrected QT = 0.591 seconds) was observed on an electrocardiogram (ECG) taken in the emergency department. The patient's drug history revealed olanzapine as the suspicious agent. Even after the medication was stopped, however, the QT interval remained within an abnormal range and multiple episodes of TdP and related seizure-like symptoms were found via ECG monitoring. A permanent pacemaker was thus implanted, and the ventricular rate was set at over 80 beats/min. There was no recurrence of tachyarrhythmia or other symptoms. (**Korean J Intern Med 2011;26:99-102**)

**Keywords:** Complete atrioventricular block; Pacemaker; Torsade de pointes

## INTRODUCTION

Torsade de pointes (TdP) is a malignant ventricular arrhythmia that is associated with syncope and sudden death, and may be a significant complication of atrioventricular (AV) block associated with QT prolongation [1]. The term torsade de pointes literally means a "twisting of points" and refers to a characteristic pattern of ventricular tachycardia. TdP is associated with long QT intervals and is generally unresponsive to the usual anti-arrhythmic drugs [2,3]. Our patient complained of presyncope and syncope, and it appeared that the major ailment was epilepsy. Serial electrocardiograms suggested conduction system abnormalities, and evaluation of these results did not reveal any additional causes. The patient's dysrhythmia abated with moderate rate cardiac pacing. Herein we report a case of complete AV-block-related TdP that

appeared to be attributable to a primary cardiac-conducting system disease rather than to a drug, thus leading us to confuse it with epilepsy.

## CASE REPORT

A 42-year-old woman with a history of aortic mechanical-valve replacement, due to rheumatic-valve disease, was referred to the emergency department of our hospital after she complained of recurrent seizures. Approximately 13 years ago, the patient underwent open-heart surgery and there had been no history of syncope or seizure since that point. The patient had been hospitalized in a psychiatric hospital for one month because of a history of psychosis, and was treated with the medication olanzapine. Several different anti-seizure treatments had been tried, but none of them led to any significant improvement.

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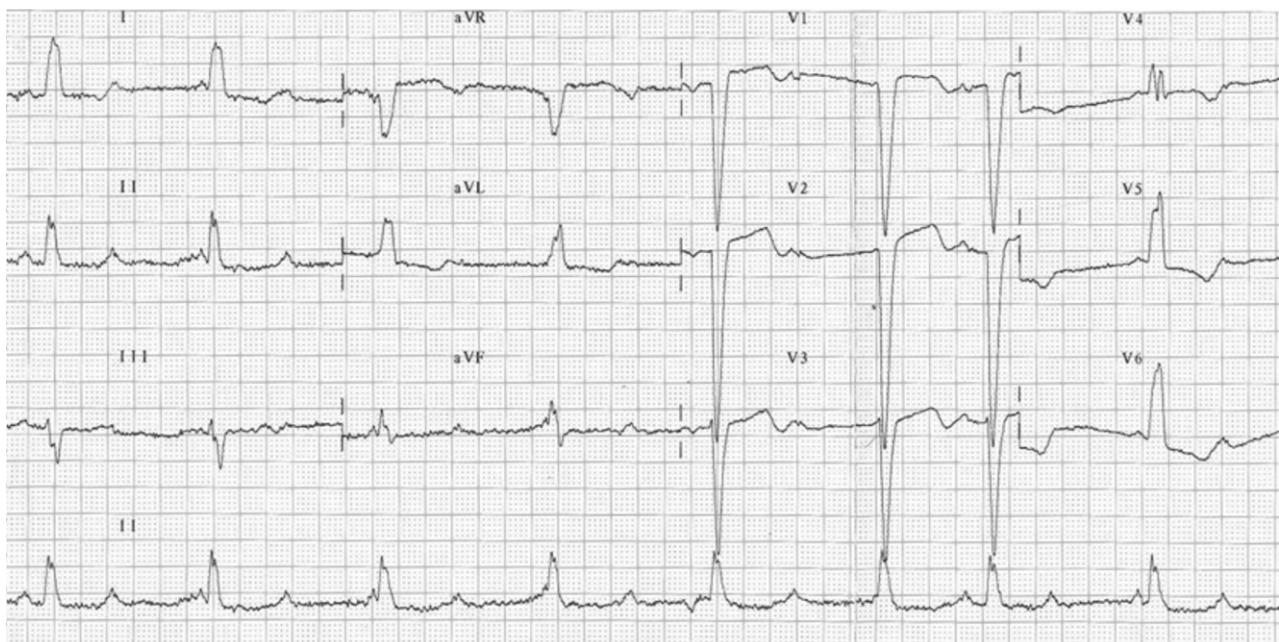
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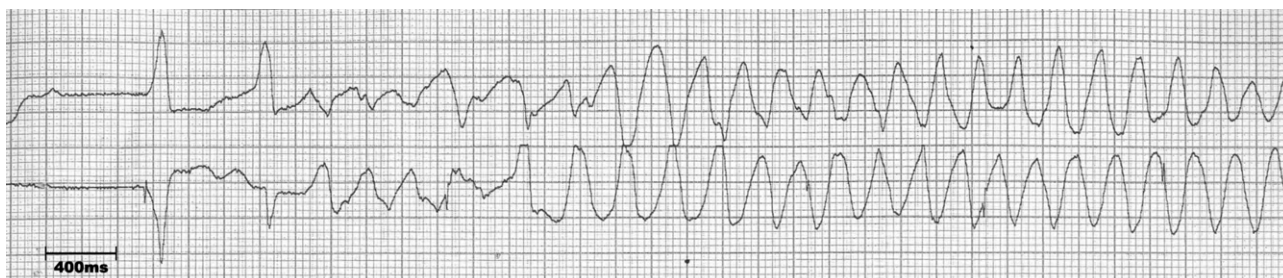
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**Figure 1.** Electrocardiogram obtained during the initial evaluation. Note the complete atrioventricular block and prolonged QT interval of 0.680 seconds (corrected QT is 0.591 seconds).

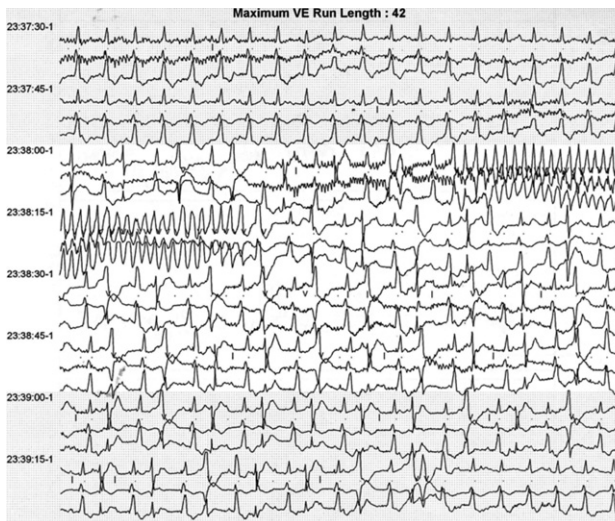


**Figure 2.** Rhythm strip demonstrating torsade de pointes.

Upon arrival at our hospital, the patient was afebrile and had a blood pressure of 120/80 mmHg, a heart rate of 48 beats/min, and a respiratory rate of 20 breaths/min. The heart sounds were irregular and slow, but no murmur was present. The initial electrocardiography (ECG) showed a complete AV block with a left-bundle-branch block pattern and a prolonged, corrected QT interval (0.591 seconds) (Fig. 1). A transthoracic echocardiographic study that was performed soon after admission revealed good regional wall motion, with a left ventricular ejection fraction of 54%. The chest X-ray revealed no abnormalities. The complete blood count was within normal limits, as were blood chemistries, including electrolytes (sodium 139 mEq/dL, potassium 4.5 mEq/dL, calcium 9.1 mEq/dL, phosphate 3.2 mEq/dL, magnesium 1.9 mEq/dL), CK-MB, and troponin-T. The results of thyroid function tests were also within normal limits. The pro-

thrombin time was prolonged (24.7 seconds, international normalized ratio 2.02) due to warfarin treatment. Previous medications taken by the patient included warfarin (3 mg daily), olanzapine (2.5 mg daily), diazepam (4 mg daily), valproic acid (300 mg twice daily), and topiramide (100 mg twice daily). The patient was transferred to the intensive care unit for continuous ECG monitoring. During the course of the illness, recurrent episodes of seizure-like activities connected with TdP were observed (Fig. 2). The patient's dysrhythmia was refractory to magnesium and a temporary pacemaker was implanted. Single photon emission tomography showed no abnormalities of the myocardium. A Holter test was conducted on the treatment that aided in making a correct diagnosis, and olanzapine, an anti-epileptic drug, was discontinued to exclude drug-mediated tachyarrhythmia. Until the 5th day of hospitalization, a sustained complete AV block and

intermittent TdP was still present, and serum electrolytes and biochemical markers of myocardial damage, including CK-MB and troponin-T, were repeatedly normal. Holter monitoring showed complete AV block with TdP (Fig. 3), which was noted even with the prolonged QT interval. Thus, we decided to implant a permanent pacemaker. During the days after permanent pacemaker implantation (ventricular pacing dual chamber sensing type), we set the ventricular rate at over 60 beats/min, resulting in a somewhat reduced frequency of syncope; however, intermittent sustained polymorphic ventricular tachycardia was still observed. Gradually, we set the ventricular rate at over 80 beats/min, which enabled the patient to return to high-level functioning, something that had not been possible since the onset of the illness. The



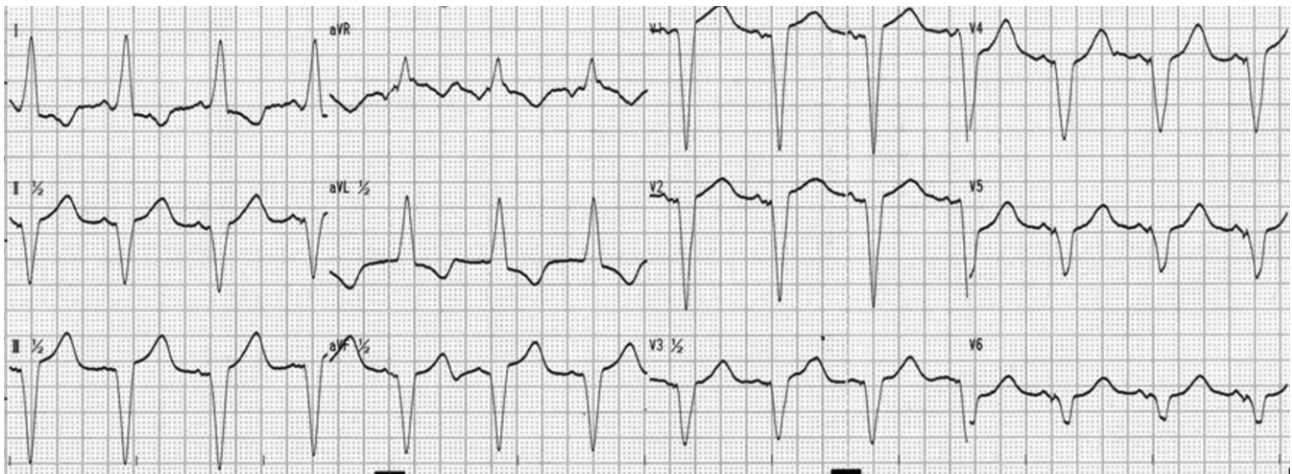
**Figure 3.** Holter test during hospitalization; note the torsade de pointes with underlying complete atrioventricular block.

follow-up ECG showed regular pacing beats (Fig. 4). The patient was discharged with a prescription of only 3 mg/day of warfarin. During the seven months following the implantation of the permanent pacemaker, the patient did not undergo any syncope attacks.

## DISCUSSION

Bradyarrhythmia-induced torsade de pointes is primarily seen during complete AV block. Additional risk factors e.g., female gender, hypokalemia, hypomagnesemia, diuretic use (independent of electrolyte serum concentrations), high drug concentrations (except for quinidine), bradycardia (especially recent heart rate slowing), congestive heart failure, cardiac hypertrophy, congenital long QT syndrome, and history of open heart surgery increase the risk of torsade de pointes during bradyarrhythmias [4].

In the case reported herein, complete heart block following open-heart surgery should be considered. However, although the patient had had open-heart surgery, no symptoms had manifested in the past 13 years. As such, we suggested that the antipsychotic drug olanzapine was the culprit. Toxic amounts of non-antiarrhythmic drugs may cause torsade de pointes [4]; however, olanzapine, a thienobenzodiazepine derivative antipsychotic agent mainly metabolized via cytochrome P4501A2 (CYP1A2) and cytochrome P4502D6 (CYP2D6) isoenzymes, exhibits a very low binding affinity with the human-ether-a-go-go-related-gene-(HERG)-mediated potassium, as compared with other antipsychotic agents, and thus does not contribute to significant QT interval



**Figure 4.** The follow-up electrocardiogram showed normal ventricle capture by the pacemaker at a rate of 80 beats/min (QT interval is 0.480 seconds, corrected QT is 0.515 seconds).

prolongation [5]. Long QT due to a high dose of olanzapine has been reported once in the literature [6]. Olanzapine was thus discontinued, but the recurrent episode of seizure-like symptoms and polymorphic ventricular tachycardia was observed until day 5 in the hospital. As a result we could exclude the presence of drug-mediated TdP.

Kurita et al. [7], who studied patients with complete AV block with TdP, found that these patients had a bradycardia-sensitive repolarization abnormality, which remained after pacemaker implantation. They suggested that the critical heart rate that induced abnormal QT prolongation was less than or equal to 60 beats/min, and that it had a potential to develop TdP [7]. In a patient with complete AV block, a QT interval of above 0.60 seconds on the ECG seems to indicate an increased risk of developing polymorphic ventricular tachycardia [8].

As mentioned above, there are numerous causes of TdP with long QT intervals. Aside from correcting any of these predisposing conditions, the treatment of long QT syndrome is directed at regulating heart rate, normalizing the QT length, and preventing early ventricular depolarizations [9]. Since we could not determine any predisposing factor, we thus considered primary conduction system disease. Permanent cardiac pacing is effective with TdP with complete AV block and long QT intervals [9,10]. Heart rate acceleration resulted in the immediate suppression of all arrhythmias. Pacing was continued until the condition producing QT prolongation disappeared. It is important to increase the pacing rate to over 70 beats/min or maintain the QT interval at less than 0.440 s to prevent TdP in the presence of a cardiac pacemaker [2,10].

In the case reported herein, a permanent pacemaker had been implanted and the absolute QT interval shortened by overdrive pacing from a mean value of 0.680 seconds to 0.480 seconds (corrected QT = 0.515 seconds). The placement of a transvenous right ventricular pacemaker with pacing set at a rate of 60 beats/min could not terminate TdP for the entire day. As discussed above, we could terminate TdP and syncope with overdrive pacing at a rate of 80 beats/min. Overdrive pacing was able to suppress these symptoms. Patients with bradycardia-mediated TdP and complete AV block can develop syncope,

which is related to TdP, even though they are under cardiac pacing. Therefore, overdrive pacing may be the treatment of choice for bradycardia-mediated TdP associated with complete AV block.

### Conflict of interest

No potential conflict of interest relevant to this article was reported.

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