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Journal of Electrocardiology 48 (2015) 450 – 454

JOURNAL OF Electrocardiology

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Torsade de pointes in a patient with complete atrioventricular block and pacemaker failure, misdiagnosed with epilepsy

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Abstract A case of torsade de pointes (TdP) with complete atrioventricular block and pacemaker failure that was misdiagnosed as epilepsy is presented herein. An 82-year-old female with recurrent seizure-like attacks showed epileptiform discharge during an electroencephalogram recording. A long QT interval and severe hypokalemia induced runs of TdP, which was related to pacemaker lead fracture, was detected during Holter recording and accompanied with episodes of seizures. After a DDD pacemaker with a new ventricular lead was replaced, there was no recurrence of any seizure-like attacks. Bradycardia-mediated TdP associated with complete atrioventricular block should not be missed in patients with recurrent seizure-like attacks even after pacemaker implantation.

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

Torsade de pointes (TdP) may be a significant complication of atrioventricular (AV) block associated with QT prolongation. TdP and associated long QT syndrome (LQTS) can precipitate syncope, seizures or sudden cardiac death. Patients may be misdiagnosed with a seizure disorder or epilepsy and treated with antiepileptic drug medication. We report a case initially diagnosed as having epilepsy, but Holter recording revealed runs of TdP tachycardia, connected with episode of seizure-like activities, leading to a further diagnosis of AV block associated LQTS and TdP.

Case

An 82-year-old woman was admitted to the emergency room after suffering several seizure-like episodes. Family members reported that she had sudden loss of consciousness accompanied by jerking of the upper left limb, paroxysm of involuntary mouth twitching and upward gazing. Her consciousness recovered spontaneously without postictal confusion, and the episode subsided within 10 seconds. The patient had no tongue biting or incontinence with the episode. The long-term electroencephalogram (EEG) recorded in the local hospital documented the presence of right frontal and central epileptiform discharges (Fig. 1). Consequently, the patient was diagnosed with partial seizure disorder and was treated with levetiracetam combined with valproate, which was later substituted by carbamazepine. The seizure-like attacks continued despite adequate doses of anticonvulsant drugs. The patient ate less and less, and suffered from nausea and fatigue. Her past medical history included a VVI pacemaker implantation due to third-degree atrioventricular (AV) block in a different hospital 13 years ago. The generator was replaced 5 years ago and she often missed her follow-up appointments. She denied history of hypertension or diabetes. There is no family history of seizures, early cardiac disease, or sudden death.

When the patient arrived at the emergency department, she was awake, alert, and oriented, with no complaints of headache, chest pain, or palpitations. Her blood pressure was 108/75 mmHg, a heart rate of 65 beats per minute (bpm), and a respiratory rate of 20 breaths per minute. Her physical and neurologic examinations were unremarkable. Laboratory studies were normal, with the exception of serum potassium concentration of 3.21 mEq/L. Computed tomography of her head showed lacunar infarction. Echocardiography showed normal cardiac structure and function. Electrocardiogram (ECG) on admission showed sinus rhythm with complete AV block and ventricular paced rhythm at a rate of 65 pulses per minute (ppm) and the QT interval was 0.60 seconds (QTc 0.53 seconds) (Fig. 2).

The patient was admitted to the neurology service for further evaluation of her presumed seizures. On the third day of hospitalization, an ambulatory ECG was performed, during which several episodes of seizure-like attacks were witnessed. The 24-hour Holter monitoring revealed a sinus rhythm with third-degree AV block and ventricular paced

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http://dx.doi.org/10.1016/j.jelectrocard.2015.03.007
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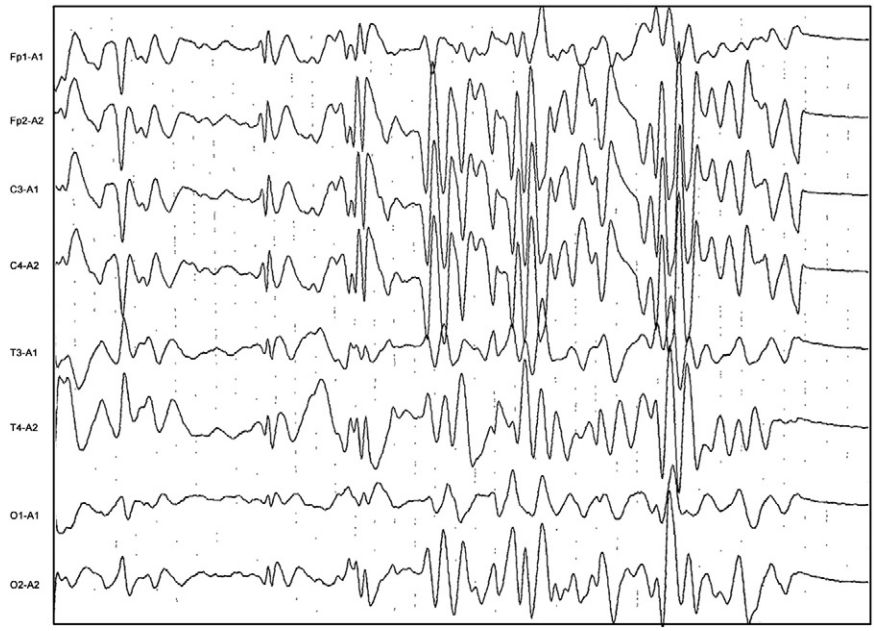

Fig. 1. Ambulatory electroencephalogram recording with bursts of high amplitude sharp waves over the right frontal and central regions.

rhythm (Fig. 3a). Intermittent loss of ventricular capture (Fig. 3b), which resulted in a very slow junction escape rhythm at the rate of 18–30 bpm, was detected, accompanied with a prominent TU abnormality and an extremely prolonged QTU interval (QT 0.88 seconds) (Fig. 3c). A ventricular premature beat initiated runs of TdP with repeated cycles of 10–20 beats and terminated spontaneously, with the longest lasting 10.4 seconds (Fig. 3d), coinciding with the symptomatology. Blood samples showed severe hypokalemia (2.52 mEq/L) and normal serum magnesium level (0.83 mEq/L). She was immediately treated with parenteral magnesium sulfate and potassium chloride. Battery depletion, lead fracture, or dislodgment resulting in intermittent pacemaker failure was suspected. The interrogation of the device revealed that the battery status was within normal ranges (voltage 2.78 V, impedance 474 Ω). A ventricular pacing threshold test was performed showing the right ventricular capture threshold of 2.65 V at 0.4 milliseconds. The ventricular pacing lead impedance was 419 Ω. A chest radiograph revealed fracture and insulation breaks at the angulation of the lead (Fig. 4). A detailed real-time fluoroscopy along the course of the lead further confirmed the abnormality. On the next day, a pacemaker replacement was performed and a DDD generator (Medtronic RELIA RED01, USA) with a new ventricular lead (Medtronic 5076-58 cm, USA) was implanted (Fig. 5). After 2 days of potassium supplementation, the serum potassium level rose to 4.13 mEq/L. The patient was discharged the following day and the ECG showed VAT mode with a shortened QT interval of 0.48 seconds. No symptoms were noted by the patient at the follow-up evaluation despite that she had discontinued antiepileptic drugs since her discharge.

Discussion

In the case reported herein, epileptic seizure was considered first based on the patient's presenting symptoms. However, although the patient showed epileptiform discharge during ambulatory EEG, no symptoms had improved after anticonvulsant therapy. Holter recording documented episodes of TdP tachycardia leading to seizure-like attacks. The interrogation of pacemaker and the chest X ray indicated lead angulation and insulation breaks, which are commonly

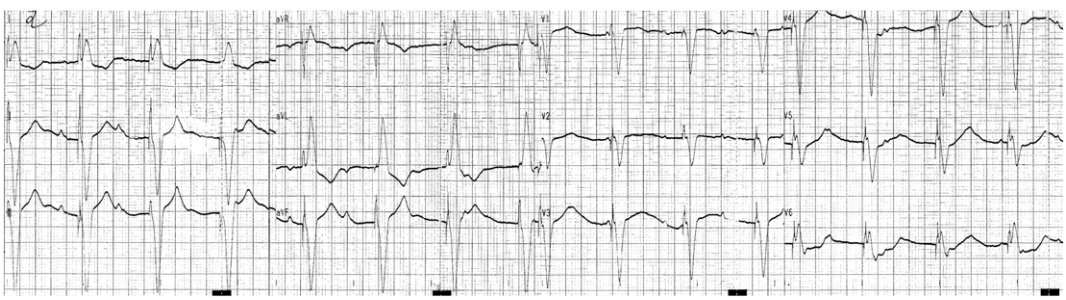

Fig. 2. Electrocardiogram obtained during initial evaluation. Note the complete atrioventricular block, ventricular paced rhythm and prolonged QT interval of 0.60 seconds (QT_C 0.53 seconds).