Cardiac arrest due to torsades de pointes in a patient with complete heart block: the “R-on-T” phenomenon

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Figure 1. P waves are shown without correlation to QRS complexes.

A 59-year-old white man with type 2 diabetes mellitus and no regular medical care presented to the emergency department with syncope while driving. The patient denied palpitations, chest pain, or blurry vision prior to the event. After passing out, he reported no postictal symptoms. He reported having a “slow heart rate” over the last year. His heart rate in the emergency department was about 30 beats per minute, and an electrocardiogram showed intermittent complete heart block (*Figure 1*).

On examination he was afebrile, hypertensive (blood pressure of 189/78 mm Hg), bradycardic (heart rate of 46 beats per minute) with a normal respiratory rate (20 breaths per minute), and had a normal arterial oxygen saturation (SpO₂ of 100% on 2 L/min nasal cannula). The patient was alert and oriented. He had no murmurs, rubs, or gallops. Complete blood count was within normal limits. His blood glucose was 220 mg/dL and serum potassium was 3.2 mEq/L, which was being repleted. His thyroid-stimulating hormone level was within normal limits and hemoglobin A1C was 7.7%.

Upon arrival in the intensive care unit, he went into cardiac arrest. An electrocardiogram showed ventricular premature complexes (VPCs) triggering torsades de pointes (TdP) (*Figure 2*). Chest compressions, atropine (1 mg intravenously), magnesium sulfate (1 g intravenously), and defibrillation were applied before he returned to his baseline rhythm of third-degree heart block. At the time of the cardiac arrest, the patient’s potassium and magnesium levels were 3.8 mEq/dL and 2 mg/dL, respectively. The patient was chemically paced

with isoproterenol and transcutaneously paced following the event.

Following the cardiac arrest, the patient’s transthoracic echocardiogram showed normal left ventricular systolic function, borderline left ventricular hypertrophy with associated mild impaired relaxation, and mild mitral regurgitation. The patient received definitive treatment with an implantation of a Medtronic Sensia DDD pacemaker and was discharged home 2 days later.

DISCUSSION

The “R-on-T” phenomenon was first described by Smirk in 1949. He observed that abnormal electrocardiogram morphology preceded the start of ventricular fibrillation. Smirk described this morphology as an R wave superimposed on T waves, representing a ventricular depolarization of an ectopic beat on the preceding T wave. He believed that these early beats were precursors for ventricular fibrillation and sudden death (1, 2).

Fries et al looked at the incidence of this phenomenon in patients with implantable cardioverter-defibrillators. The

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Figure 2. (a) Initiation of torsades de pointes (TdP) with a ventricular premature complex (VPC) falling on the downslope of the T wave. Classic polymorphic ventricular tachycardia is seen after initiation. (b) TdP ultimately devolves into coarse ventricular fibrillation. After defibrillation, there is resolution of the ventricular fibrillation and resumption of the patient's sinus rhythm with complete heart block. (c) Continued complete heart block is seen after shock, with dissociation of P waves and QRS complexes.

investigators looked at the incidence of a VPC preceding ventricular tachycardia (VT) as well as the location of the VPC on the T wave: on the ascending limb, top of the wave, or descending limb. The result of the study was that 15% of VPCs initiated VT. Of the VPCs that led to VT, 16% occurred on the ascending limb, 23% on top, and 61% on the descending limb of the T wave. The authors concluded that the “R-on-T” phenomenon is rarely a cause of sustained VT; however, when it does occur it is more likely to occur on the descending limb of the preceding T wave and leads to TdP (3).

Kurita et al observed that complete heart block patients with heart rates of <60 beats per minute would have widening of their QT intervals. In addition, they found that patients with QT intervals ≥ 550 ms had an increased incidence of TdP (4). The descending limb of the T wave represents the transmural dispersion of repolarization and is considered to be a vulnerable period for electrical interruption. This period is increased in prolonged QT. The transmural dispersion of repolarization functions as a reentrant substrate to initiate and maintain a TdP

when a VPC occurs in this period due to phase 2 reentry or phase 2 early after depolarization (5, 6). To prevent reoccurrence of TdP, Kurita et al recommended (as in our patient) ventricular pacing above 70 beats a minute (4).

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