Abstract

One of the less frequent underlying mechanisms of ventricular tachycardia (VT) is triggered activity. Triggered activity refers to an extrasystole due to a premature depolarization that occurs when the amplitude of an early or delayed afterdepolarization brings the cardiac membrane to its threshold potential. Hydrochlorothiazide and hydroxyzine can prolong repolarization and QT interval and are associated with early afterdepolarizations. Cyclic AMP-mediated, delayed afterdepolarizations can occur as a result of catecholaminergic surge. Delayed afterdepolarization is classically associated with outflow tract (OT) tachycardia, a type of VT that is uniquely defined by its termination with adenosine. We present a case of triggered OT tachycardia for which intravenous amiodarone through its antiadrenergic effect may have been effective. Infusions of magnesium and a cardioselective, β-receptor antagonist that does not prolong repolarization may have been more appropriate given the concurrent, acquired prolonged QT syndrome. After initial stabilization, considering the underlying VT mechanism may prompt the clinician to select the most appropriate, further treatment.

Case

A 50-year-old male with hypertension and anxiety disorder is brought to the emergency department (ED) by emergency medical services (EMS) complaining of intermittent dyspnea and palpitations for three weeks. The EMS rhythm strip was interpreted as “VT”, and 150 mg IV amiodarone had been administered. An urgent care center had recently started the patient on hydrochlorothiazide and hydroxyzine. Upon arrival to the

ED, the patient is awake, alert, and appears very anxious. His chest is clear to auscultation, and pulses are equal and strong. The cardiac monitor shows transient episodes of wide-complex tachycardia. The following ECG is obtained (Figures 1 and 2).

Clinical Course

The physicians administer two grams of magnesium, two milligrams of midazolam for anxiety, and a second 150 mg bolus of amiodarone for presumed recurrent ventricular tachycardia (VT). Minutes later, the patient's symptoms and episodes of tachydysrhythmia resolve. The troponin I is normal, potassium level is 2.7 mmol/L (low), and magnesium is 2.3 mg/dL (normal). Potassium is supplemented with 40 milliequivalents intravenously. An amiodarone infusion is started, and the patient is admitted to the coronary care unit. Echocardiography demonstrates mild concentric left ventricular hypertrophy and normal systolic function. Upon electrophysiology study, the VT is not reproducible with programmed stimulation or isoproterenol. Given the morphology of the observed premature ventricular complexes (PVC) during telemetry, the electrophysiologist maps the left ventricular outflow tract and then successfully ablates multiple PVC-initiating foci in the aorto-mitral continuity area. The QTc on the day of discharge is 464 milliseconds. The patient is instructed to discontinue hydroxyzine and hydrochlorothiazide and started on lisinopril and amlodipine. A zio-patch detects no further VT episodes upon outpatient follow-up.

Discussion

An acquired prolonged QT syndrome due to thiazide-related hypokalemia and hydroxyzine's antihistaminic effect may have predisposed the patient to early afterdepolarizations and VT episodes[1] that manifested as palpitations. In addition, cAMP-mediated, delayed afterdepolarizations may have occurred as a result of anxiety-related, catecholaminergic surges. One of the less frequent underlying mechanisms of VT is triggered activity. Triggered activity refers to an extrasystole due to a premature depolarization that occurs when the amplitude of an early or delayed afterdepolarization brings the cardiac membrane to its threshold potential. In short, a spontaneous action potential is generated at an inappropriate time in the cardiac cycle. In the presented ECG, sinus tachycardia precedes the monomorphic, wide-complex tachydysrhythmia, suggesting that increased catecholamine levels (possibly related to emotional stress) may have led to delayed afterdepolarization that then "triggered" the VT. Delayed afterdepolarization is classically associated with outflow tract (OT) tachycardia.[2]

The ECG in the case is consistent with a triggered, OT tachycardia, as the tachydysrhythmia starts with a PVC that is similar to the ensuing complexes (as opposed to reentrant VT where the initiating PVC is dissimilar)[3] and there is an inferior axis and left bundle branch block pattern. Baseline echocardiography will also help to distinguish between types of monomorphic VT, as OT tachycardia is the most common in patients without structural heart disease.[4,5] Lastly, electrophysiology study can assist with diagnosis and definitive therapy. When programmed electrical stimulation cannot reproduce the VT, reentry is excluded as the mechanism, but triggered activity

remains possible.[3] Additional, detailed ECG analysis to predict the site (i.e. right versus left ventricle) from which an OT tachycardia originates is only important for the electrophysiologist to accurately map and ablate the focus. The origin in the case was the aorto-mitral continuity area, which is known to trigger mitral annular VT[2], one of many subtypes of OT tachycardia.

Outflow tract tachycardias are somewhat defined by their termination with adenosine, however, β-receptor antagonists can also terminate OT tachycardias by antagonizing the dysrhythmogenic effect of adrenergic stimulation.[2,5] In the presented case, intravenous amiodarone through its antiadrenergic effect [6] may have reduced PVC burden from the outflow tract [5] and consequently suppressed the tachydysrhythmia. However, a cardioselective, β-receptor antagonist (i.e. esmolol) that does not prolong repolarization may have been more appropriate given the concurrent, acquired prolonged QT syndrome.

Timely determination of hemodynamic stability and treatment of unstable tachydysrhythmias take precedent over the actual diagnosis of monomorphic VT, and typical guideline-directed management[7] is likely to be effective for the majority of regular, wide-complex tachydysrhythmias. However, consideration of the underlying mechanism afterward may aid in selecting the most appropriate, further treatment. The recognition of an acquired long QT syndrome and suspicion of triggered activity may help the astute clinician to avoid the guideline-recommended agents, procainamide and amiodarone,[7] and their potentially deleterious effects (further prolongation of

repolarization), and instead initiate suppressive magnesium and β-receptor antagonist infusions.

This research did not receive any specific grant from funding agencies in the public, commercial, or not-for-profit sectors.

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Figure Captions

Figure 1. Presenting Electrocardiogram

The ECG demonstrates sinus tachycardia with prolonged QTc followed by the initiation of regular, monomorphic wide-complex tachydysrhythmia with AV dissociation. There is an inferior axis, left bundle branch pattern, and precordial transition at V3.

Figure 2. Simultaneous Tracings of Leads II (top), V1 (middle), and V5 (bottom)

The rhythm tracings demonstrate prolonged QT (green bracket; QTc 582 milliseconds) followed by the initiation (purple arrow) of regular, monomorphic wide-complex tachydysrhythmia with underlying P (blue arrow) suggesting AV dissociation, the hallmark of monomorphic ventricular tachycardia.