Idiopathic Ventricular Tachycardia Associated With AV Reciprocating Tachycardia
Charles-Lwanga K. Bennin, MD, Avinash Chandra, MD

Case Report
A forty-six year old female patient with a medical history of asthma and seasonal allergies presented with a two day history of shortness of breath, associated with palpitations. She denied chest pain or diaphoresis. Patient reported past history of similar complaints that usually lasted less than two minutes and were relieved with aspirin. The longest episode of palpitations lasted approximately twenty minutes.

Her social history was significant for tobacco and alcohol abuse, but negative for substance abuse. Her family history was significant for cardiovascular disease.

Pertinent findings on physical examination were significant for elevated blood pressure at 156 mmHg/112 mmHg and tachycardia at 112 beats per minute. Her electrocardiogram (EKG) showed tachycardia at a rate of 150 beats per minute with a long RP interval concerning for atypical AV reciprocating tachycardia (AVRT). The EKG also revealed right bundle branch block with a left axis deviation (Figure 1) and non-sustained ventricular tachycardia (Figure 2) indicating a diagnosis of idiopathic ventricular tachycardia. A urine drug screen was positive for cocaine.

Discussion
Idiopathic fascicular ventricular tachycardia has been reported in literature by Cohen in 1972\(^1\) and Zipes in 1979\(^2\). These electrophysiologic findings are unique in that the QRS complexes are narrow, especially when compared to the typical wide QRS complex ventricular tachycardia and a right bundle branch morphology. The fascicle activated on re-entry determines the axis - left axis deviation (left posterior fascicle) and right axis deviation (left anterior fascicle). Some case have been reported of familial presentations of idiopathic ventricular tachycardia.\(^3\)

Eighty percent (80%) of idiopathic ventricular tachycardia originates from the ventricular out flow tracts or the coronary cusp.\(^4\) The origin of these arrhythmias has been attributed to the posterior inferior left ventricle in a region of the left posterior fascicle.\(^5\) This region probably has a high degree of reentrant or triggered automaticity.\(^6\) The adjacent posterior left bundle branch may or may not be involved in the anterograde limb of the reentrant circuit.\(^7\)

Idiopathic ventricular tachycardia, having a morphology of left bundle branch block and right axis deviation, is believed to originate in the right ventricular outflow tract, but may arise from the right ventricular outflow tract in about 18% of cases.\(^8\) According to Francis et al. idiopathic fascicular ventricular tachycardia has been limited to three subtypes in order of prevalence; left posterior fascicular ventricular tachycardia with a right bundle branch block morphology and superior axis configuration; left anterior fascicular ventricular tachycardia with right bundle branch block and right-axis

Figure 1. EKG on admission showing right bundle branch block with a left axis deviation.
deviation configuration; and the rare presentation of upper septal fascicular ventricular tachycardia with a narrow QRS and normal axis configuration. It appears that fascicular ventricular tachycardia are sensitive to phenylalkylamine class L-Type calcium channel blockers such as verapamil as have been well described by Belhassen. Calcium channel blockers also suppress conduction through atrio-ventricular (AV) node, and are effective on both AV reciprocating tachycardia (AVRT) and AV nodal reentrant tachycardia (AVNRT).

Atrial pacing as well as supraventricular tachycardia has been shown to induce ventricular tachycardia due to either reentry or triggered automaticity. It is not uncommon to find co-existing tachyarrhythmia and a few cases have been reported of ventricular tachycardia initiated by atrial arrhythmias including AV reciprocating tachycardia also known as AV reentrant tachycardia (AVRT). A literature search using PubMed with the following keywords “Idiopathic ventricular tachycardia, AV reentrant tachycardia, AV reciprocating tachycardia, AVRT” produced 2 articles. One case series reported seven patients without structural heart disease in which AVNRT spontaneously triggered VT in three cases. Another case report illustrated a patient with Wolff-Parkinson-White (WPW) syndrome. Ventricular tachycardia originating from the right ventricular outflow tract was induced during isoprenaline infusion. This also led to ativoventricular reentrant tachycardia (AVRT). This case report was significant in that the ventricular tachycardia was possibly driven by catecholamine stimulation.

In our case report we present a patient with possible cocaine induced idiopathic ventricular tachycardia and AV reentrant tachycardia. Cocaine works on by increasing release of norepinephrine and dopamine and blocking reabsorption of norepinephrine, dopamine and serotonin. Cocaine also blocks sodium channels, thereby interfering with the propagation of action potentials. The release of catecholamines that occur in cocaine intake may exacerbate changes in the action potential threshold and hence stimulate automaticity. Management of idiopathic ventricular tachycardia includes radiofrequency ablation and intravenous verapamil.

Figure 2. EKG on admission showing non-sustained ventricular tachycardia.

![EKG on admission showing non-sustained ventricular tachycardia.](image)

**Reference**


