Young Male With Incessantly Wide Complex Tachycardia

What Is the Substrate of the Arrhythmia?

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ECG CHALLENGE

A 26-year-old male with no past medical issues presented with 24-hour sudden onset palpitations, chest discomfort, and dizziness. He was hemodynamically stable and his heart rate was 189 bpm, with no other remarkable findings on physical examination. A 12-lead ECG (Figure 1) showed regular wide complex tachycardia with right bundle-branch block and extreme superior axis. Stepwise management was provided with vagal maneuvers, escalating doses of adenosine, uptitrated intravenous verapamil, and cardioversion with serial discharges ≤270 J of biphasic energy. However, all interventions were ineffective, and the tachycardia remained incessant. What is the arrhythmia, and what cardiac structure is involved in its origin?

Please turn the page to read the diagnosis.

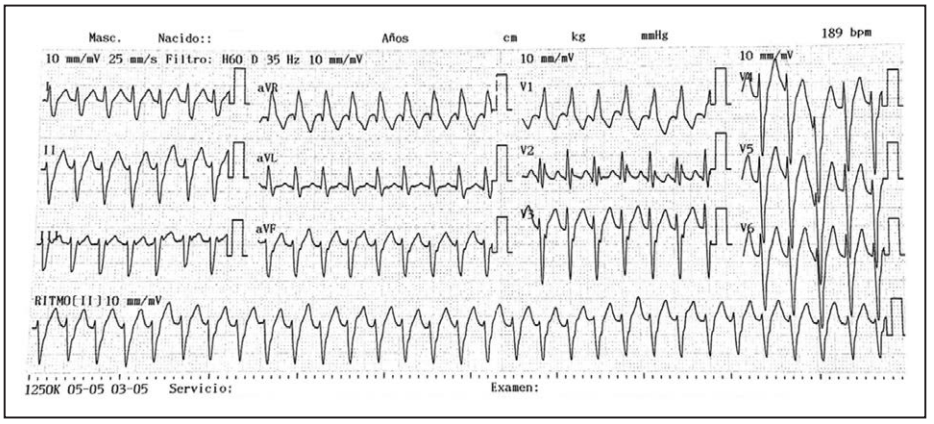

Figure 1. Twelve-lead ECG recorded in the emergency department showing a regular wide complex tachycardia with right bundle-branch block morphology and right superior axis deviation.

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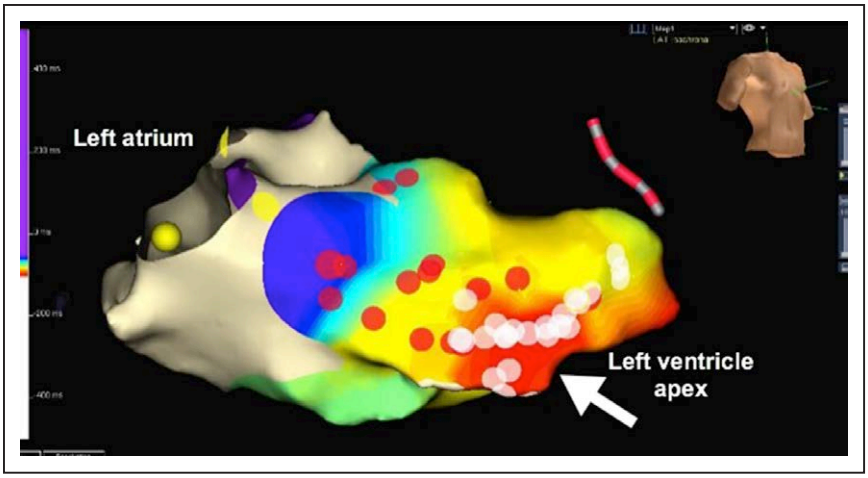

Figure 2. Electroanatomic mapping showing the earliest activation arising from the left ventricular apicoseptal region.
Apicoseptal region showing the earliest activation region of the VT (arrow), His potential (yellow dots), radiofrequency energy application (white dots), and Purkinje potential. VT indicates ventricular tachycardia (red dots).

RESPONSE TO ECG CHALLENGE

The ECG shows a regular wide complex tachycardia with right bundle-branch block morphology and right superior axis, suggesting a fascicular ventricular tachycardia (VT), poor responsiveness to intravenous verapamil, and cardioversion is not characteristic of this arrhythmia. Laboratories and echocardiography were normal.

Electrophysiological study with 3-dimensional electroanatomical mapping was carried out. A VT similar to the one previously described was induced with burst pacing from right atria. Several features, such as resistannce to cardioversion, suggested an automatic mechanism with focal exit in the apicoseptal left ventricle region (Figure 2). Intracardiac echocardiography showed a false tendon (FT) (Figure 3 and Video I in the online-only Data Supplement) that corresponded to the earliest site of activation. Radiofrequency ablation was applied in the proximity of interventricular septum insertion of FT with resolution of tachycardia. In addition, lines of ablation were created across the left posterior fascicle. The ablation was successful with no induction of ventricular arrhythmias with multiple protocols under isoproterenol infusion.

ECG is essential to approach ventricular arrhythmias (VAs). Its interpretation is necessary to identify the type and best treatment options. In the absence of structural heart disease, VAs most commonly arise from ventricles' outflow tracts. In addition, a subset of VAs originate from other endocardial complex structures such as parahisian myocardium, right ventricle moderator band, FT, and left ventricle papillary muscles (PMs)[1].

FTs (Figure 4) appear to act as substrates of VA with specific electrophysiological features and particular technical difficulties for mapping and ablation.[1] Abouezzeddine et al[2] analyzed 190 patients presenting for VT ablation and found that in 15 (8%), direct radiofrequency energy delivery over endocardial complex structures was necessary to eliminate the arrhythmia; implied structures were the right ventricle moderator band (7/15), PM (5/15), and FT (3/15). Normal cardiac structure was seen in 90% of patients with right ventricle moderator band or FT-originated arrhythmias.

It is a challenge to differentiate FT-related VA from other septal arrhythmias such as fascicular VT (Belhassen tachycardia) and PM-originated VA. Fascicular VT is exceptionally susceptible to intravenous verapamil, and wavefront goes from basal to posteroinferior septum and predominantly reentrant mechanism. Conversely, our case was detected in the apicoseptal region, and it was resistant to intravenous verapamil and automatic. Arrhythmias arising from PM are difficult to discriminate from FT-mediated VA, requiring electroanatomical mapping guided by intracardiac echocardiography to prove early site of activation. PM VA are mainly reentrant and more related with a structurally abnormal heart.[2] Fascicular VT has been related to the presence of FT because of ECG similarities, but this is now matter of controversy[3].

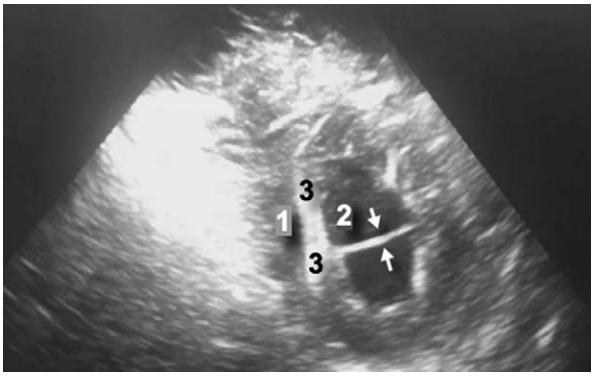

Figure 3. Intracardiac ultrasound with left ventricle false tendon closely related with high-voltage apicoseptal region shown on 3-dimensional mapping.
Left ventricle false tendon is marked between arrows: 1, right ventricle; 2, left ventricle; 3, interventricular septum (see Video I in the online-only Data Supplement).

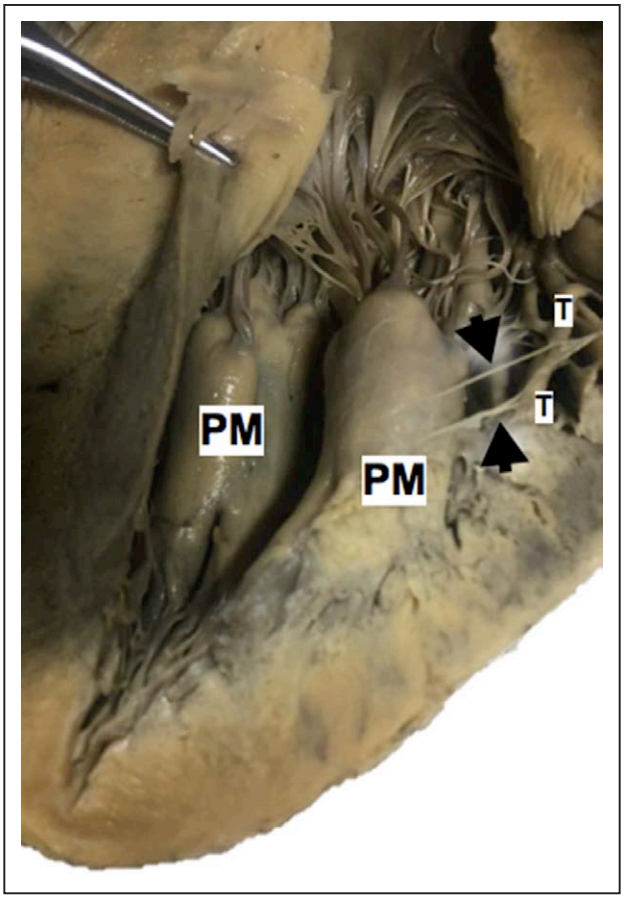

Figure 4. Anatomic dissection of human heart opening in the left ventricle.
Complex endocardial anatomy with multiple structures in different directions within left ventricle. Arrow heads point to false tendon connecting trabeculae (T) with papillary muscle (PM), which may result in simultaneous activation at different points of the LV conferring to difficulties in diagnosis and ablation of these arrhythmias. Figure courtesy of Dr Rojas, Department of Morphology, Faculty of Medicine, Universidad de Antioquia.

This case illustrates an increasingly recognized VT syndrome related with special and intriguing structures as FT. Classic features of previously reported FT arrhythmic syndromes include right bundle-branch block pattern and superior axis, poor response to verapamil, structurally normal heart, and electrophysiological study with mapping proving an automatic VT related with FT identified on intracardiac echocardiography. The patient was successfully treated with radiofrequency. Ablation therapy in this subset of patients reduces related morbidity and avoids the use of implantable cardioverter defibrillators.

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DISCLOSURES

None.

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FOOTNOTES

The online-only Data Supplement is available with this article at http://circ.ahajournals.org/lookup/suppl/doi:10.1161/CIRCULATIONAHA.117.032445/-/DC1.

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