Diagnosis and management of a wide QRS regular tachycardia

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Abstract

In spite of the available clinical and electrocardiographic criteria for the differential diagnosis of wide QRS complex tachycardias, distinguishing orthodromic supraventricular tachycardias is still a challenge. We present a case of a 63-year old patient admitted in our clinic after experiencing two episodes of syncope. Echocardiography showed left ventricular hypertrophy, grade 1 diastolic dysfunction and left atrial enlargement. A Holter monitoring revealed episodes of atrial fibrillation and paroxysmal narrow QRS tachycardia alternating with wide QRS tachycardia (170-180 bpm). During an electrophysiology study we induced self-limiting orthodromic supraventricular tachycardias with narrow and left bundle branch block patterns. Retrograde mapping near the mitral annulus identified a concealed accessory posteroseptal by-pass tract which was successfully ablated. After the procedure the patient developed atrial flutter and atrial fibrillation with rapid ventricular response (196 beats per minute) with a 3.9 s post-tachycardia pause. The patient underwent implantation of a cardiac pacemaker which allowed us to start antiarrhythmic treatment with amiodarone. This case shows that occult accessory posteroseptal by-pass tracts can have a late-onset presentation in a 63-year old male and explains why latent rhythm disturbances require a step-by-step medical approach.

Keywords*: wide QRS tachycardia, orthodromic supraventricular reentrant tachycardia, electrophysiology study, implantable cardiac pacemaker*

Introduction

The initial evaluation of every tachycardia should begin with determining the width of the QRS interval, thus separating narrow complex (<0.12 s) from wide complex tachycardias (WCT). Supraventricular tachycardias (SVT) arise from above or within the atrio-ventricular junction, allowing rapid ventricular activation through the His-Purkinje fibers and producing a narrow QRS complex [1, 2].

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However, SVTs that associate preexcitation, preexistent bundle branch block or functional aberrant conduction can present as WCTs [1]. Multiple electrocardiografic algorithms can be applied in distinguishing SVTs from ventricular arrhythmias, but the differential diagnosis of a WCT is a frequent diagnostic challenge [3]. VTs account for 80% of all cases of WCTs [1]. Distinguishing VT from SVT with pre-excitation can be especially difficult. However, pre-excitation is a rare cause of WCT and it should be suspected in young subjects with no structural heart disease or who have a previously diagnosed bypass-tract [1, 2].

The presence of clinical symptoms or hemodynamic instability should not be used to distinguish VT from SVT [2]. All hemodynamically unstable patients should undergo emergency cardioversion. Stable

patients can be treated with intravenous amiodarone or procainamide. Adenosine, diltiazem and verapamil are contraindicated in VT and should not be administered before the mechanism of WCT is certain [1, 2]. All unknown rhythms must be considered VTs and treated accordingly [1]. Long-term treatment options for patients with WCT include implantable cardiac pacemakers or defibrillators, antiarrhythmic drugs and catheter ablation [1, 2].

Case report

A 63 year-old male patient was admitted in our clinic after experiencing two episodes of syncope in the previous month, along with recurrent palpitations, sweating and shortness of breath. The patient had significant family history for cardiovascular disorders (hypertension and ischemic stroke) and had been previously diagnosed with type 2 diabetes and grade III hypertension.

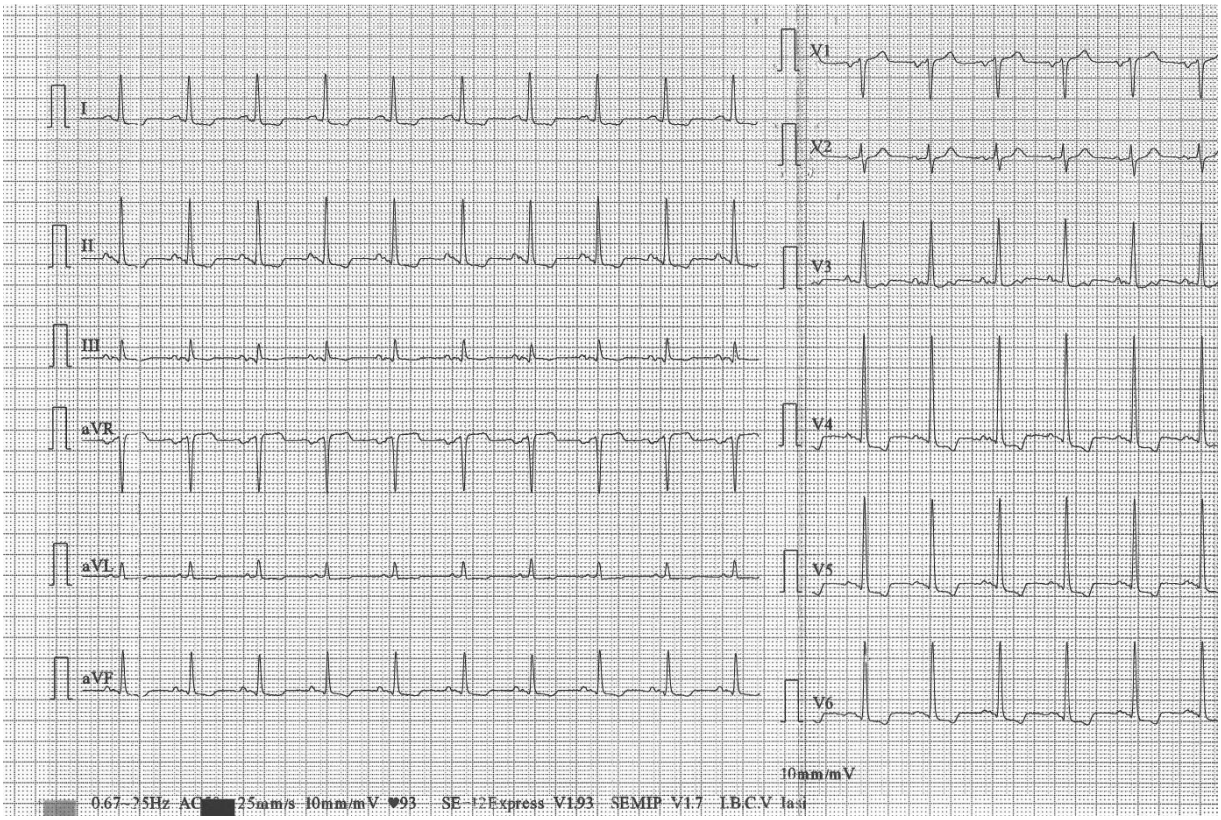

Fig. 1. Standard 12 lead ECG showing sinus rhythm with left ventricular hypertrophy

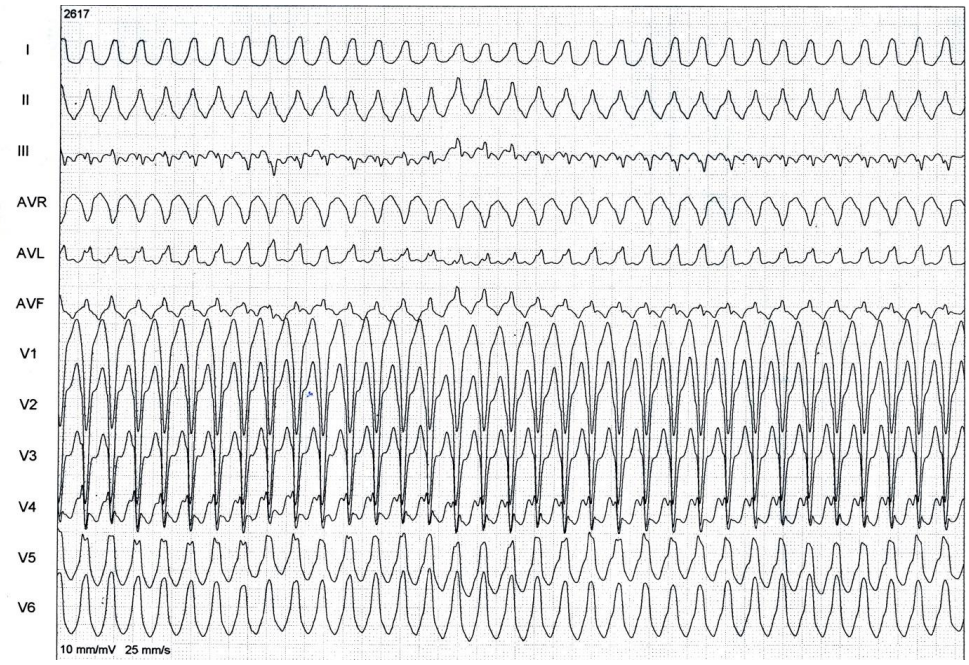

Fig. 2. Standard 12 lead ECG showing regular wide QRS complex tachycardia.

General and cardiovascular physical examinations were unremarkable (ECG on admission illustrated in Figure 1). Routine laboratory evaluation showed midly elevated uric acid and slight hypertriglyceridemia. A 24 hour Holter monitoring revealed sinus rhythm with frequent atrial extrasystoles, episodes of atrial fibrillation (AF) and paroxysmal narrow QRS tachycardia alternating with WQT (170-180 bpm) (Figure 2). We recorded 6 significant pauses > 2 s with a maximum RR interval of 3.6 s.

Echocardiography showed left ventricular hypertrophy, mild left atrial enlargement and grade 1 diastolic dysfunction, but a preserved ejection fraction (65%). The patient accused anterior thoracic pain during the tachyarrhythmic episodes, but the coronarography revealed normal coronary arteries. Doppler ultrasound ruled out carotid stenosis and renal artery stenosis as potential causes of secondary hypertension.

During the electrophysiology study (Figures 3 and 4) the baseline ECG showed sinus rhythm, 88 bpm (beats per minute), AQRS=+30 degrees, left ventricular hypertrophy and supraventricular extrasystoles. AH 60 ms, HV 52 ms. Atrial stimulation protocol revealed no ventricular preexcitation but showed aberrant intraventricular conduction with both right and left bundle branch block (RBBB, LBBB) patterns. Two atrial extra-stimuli delivered at 240 and 260 ms induced self-limiting orthodromic supraventricular tachycardias with both narrow QRS and LBBB patterns, with the same cycle length of 286 ms.

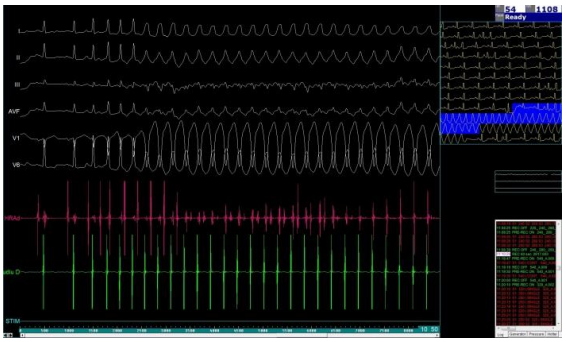

Fig. 3. Electrophysiology study. Induction of the orthodromic atrio-ventricular reentry tachycardia through 3 atrial extra-stimuli with 1:1 ventriculo-atrial conduction. Colored in red – mapping catheter positioned in the right ventricle. Colored in green – catheter positioned in the right atrium

Fig. 4. Electrophysiology study; Surface and intracavitary ECG during WCT showing a cycle length of 286 ms

During a tachyarrhythmic episode, we performed retrograde mapping in the region of the mitral annulus (anteroposterior and 40 degrees left anterior oblique) and found that the shortest VA interval was located in the left posteroseptal region. We performed radiofrequency ablation in the posteroseptal region of the mitral annulus and acquired AH 60 ms, HV 52 ms, antegrade Wenckebach point 340 ms and retrograde Wenckebach point 400 ms, confirming the cease of conduction through the accessory pathway. After 15 minutes we repeated atrial and ventricular pacing protocols which did not reveal any manifest accessory pathway conduction.

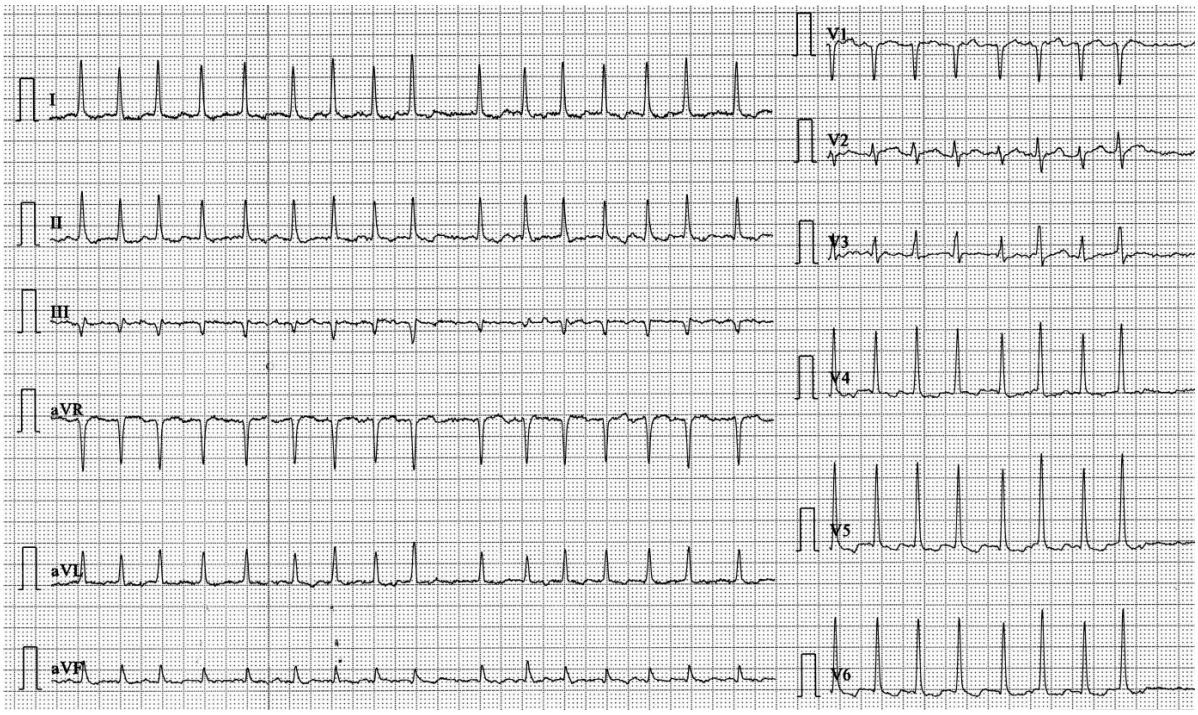

Fig. 5. Standard 12 lead ECG - atrial fibrillation

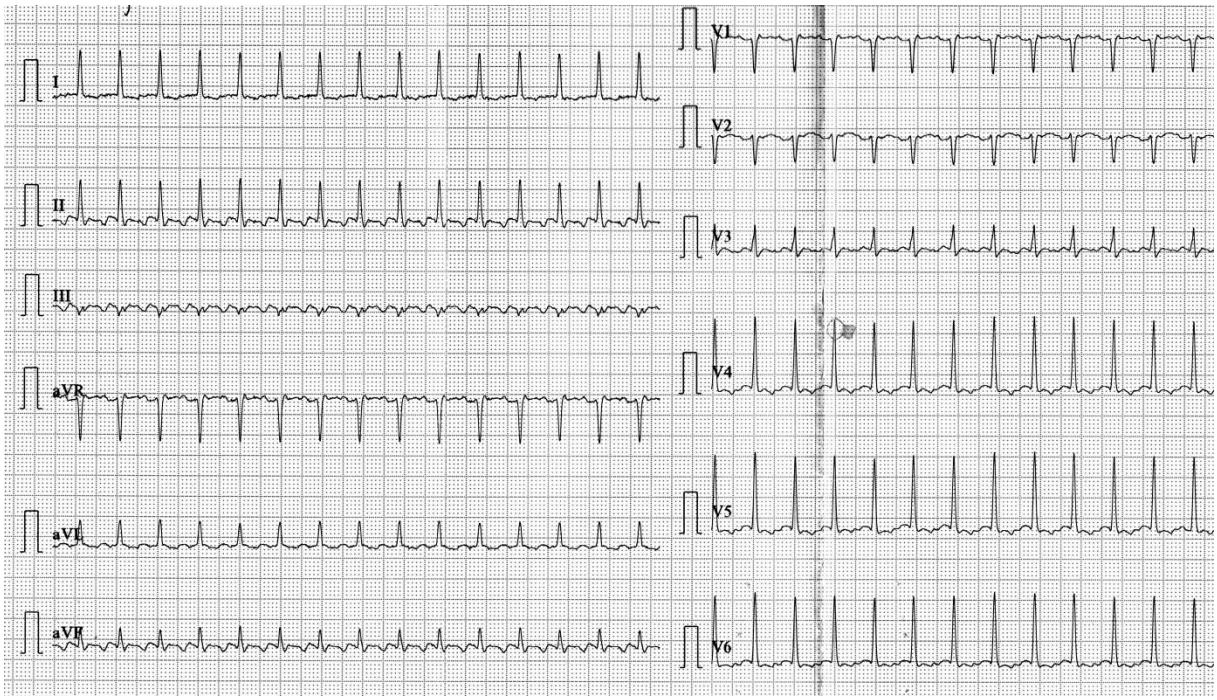

Fig. 6. Standard 12 lead ECG - atrial flutter

A day after the procedure the patient developed palpitations, sweating and dizziness an expression of rapid atrial fibrillation (Figure 5) and atrial flutter (Figure 6). A 24 hour Holter monitoring (performed under 2.5 mg bisoprolol) showed sinus rhythm alternating with atrial flutter and atrial fibrillation with rapid ventricular response (196 bpm) with a 3.9 s pause at the end of an arrhythmic episode and a minimum heart rate of 40 bpm. No regular wide QRS tachycardia was detected. Our patient was anxious, fearing recurrent syncopes, and demanded a speedy solution to his symptoms. A secondary radiofrequency ablation (for atrial flutter and atrial fibrillation) was not immediately available, so we started antiarrhythmic treatment with amiodarone under the protection of an implantable cardiac pacemaker. The procedure was successful without any local or systemic complications and the patient was discharged clear of symptoms.

Discussions

The first challenge in our case was to determine the cause of the recurrent episodes of syncope. The most frequent cause of loss of consciousness is neurally mediated (reflex) syncope, with cardiovascular syncope cited as the second most frequent etiology [4]. Reflex syncope is much more common amoung young subjects, has a typical prodrome and is usually triggered by emotional stress. Syncope in elderly patients is usually of cardiovascular cause, such as arrhythmias, orthostatic hypotension, cardiac ischemia or severe structural heart disease [4].

We excluded acute cardiac ischemia (cardiac enzymes within normal range, normal epicardial coronary arteries) and carotid artery stenosis (normal carotid Doppler ultrasound). Since our patient associated recurrent palpitations, we suspected arrhythmias to be the substrate of syncopes. In such cases, ECG monitoring should be performed (class I) [4]. Diagnostic criteria include the presence of a correlation between the syncope and an arryhthmic episode or the existence of sinus pause ≥ 3 seconds [4].

After performing a 24 hour Holter monitoring, we were faced with a regular WCT with significant pauses, confirming our diagnosis of an arrhythmic syncope. The substrate of the WCT can be suggested by several clinical features. 70% of young patients develop SVTs, while 85% of the subjects over the age of 35 present VT. History of myocardial infarction or other structural heart

diseases are strongly associated with ventricular arrhythmias [1]. Despite the fact that our patient had normal epicardial coronary arteries, he did have a history of diabetes, severe hypertension and left ventricular hypertrophy, which can cause coronary microvascular disease.

Wellens et al reported several electrocardiographic criteria that advocate for a ventricular origin of a WCT including atrio-ventricular dissociation, left axis deviation, enlarged QRS width [5]. Kindwall et al evaluated 4 criteria, recommending their combined use for improved sensitivity and specificity: a Q wave in V6 and S wave notching, S wave nadir >0.06s or an R wave >0.03s in V1-V2 [6]. Akhtar et al introduced other VT criteria: LBBB-like pattern with right axis deviation, a mininum QRS width of 0.12s for RBBB-like patterns and 0.14s for LBBB-like patterns and positive QRS concordance [7]. Brugada *et al.* offered a step-by-step approach to the differential diagnosis of WCTs [8] but the first decision-tree algorithm that focused on the aVR lead was published by Vereckei et al [9, 10]. The authors proposed simple and effective criteria, appropriate for emergency situations: any initial R wave in aVR lead can be found only with VTs [9–11]. A recent analysis concluded that the Brugada and simplified Vereckei algorithms have comparable sensibility and sensitivity in the differential diagnosis of WCT [11]. Our electrocardiographic recordings of the WCT showed no evidence of A-V dissociation, and did not meet the morphologic criteria for VT. Our patient had intermediate QRS axis, QS morphology (absence of notching) in aVR with (vi/vt)>1 in the same derivation, QS morphology in V1-V2 (no visible notching) and monophasic R wave morphology in V6.

Accessory by-pass tracts that exhibit only retrograde conduction are known as occult or concealed [12]. The patient's ECG is consistent with SVT associating rate-dependent LBBB which is common in orthodromic atrio-ventricular reentry tachycardias [3]. A left sided accessory pathway allows retrograde conduction from the ventricles to the atria while antegrade conduction occurs through the atrio-ventricular node and the right Tawara branch [3]. Orthodromic tachycardia is the most frequent form of atrio-ventricular reciprocating tachycardia, accounting for 95% of cases [12]. In the absence of associated preexcitation, accessory by-pass tract ablation should be performed as a first line treatment option for severely symptomatic patients (class I) but symptomatic individuals with rare arrhythmic episodes can also benefit from the procedure (class IIa) [12].

In our case, the retrograde mapping performed during a tachyarrhythmic episode isolated a concealed left-sided accessory pathway which was successfully ablated. After the procedure our patient developed symptomatic paroxysmal episodes of atrial flutter and fibrillation with rapid ventricular response. A second 24 hour Holter monitoring showed a significant post-tachyarrhythmic pause, marking the presence of a latent sick sinus syndrome (SSS).

Treatment options for arrhythmic syncope include implantable cardiac pacemakers, defibrillators, antiarrhythmic drugs and catheter ablation. The latter is a class I indication for patients with VT- or SVT-induced symptoms [4]. The procedure can also be performed in subjects where AF with rapid ventricular response triggered the syncope (IIb), although in such cases antiarrhythmic drugs have a class I indication [4]. Implantable cardiac pacemakers are a class I indication for SSS with asymptomatic pauses ≥ 3 seconds (excluding pauses recorded during sleep-time or under antiarrhythmic treatment) and SSS with syncopes that are clearly induced by sinus pauses [4].

Catheter ablation was another option in the management of both arrhythmic syncopes and atrial fibrillation. Isolation of the pulmonary veins is considered a Class IA indication for symptomatic paroxysmal AF recurring under antiarrhythmic therapy [13]. The technique is at least as effective in maintaining sinus rhythm as the use of antiarrhythmic drugs. Therefore, catheter ablation for AF should be considered a first line treatment option for symptomatic paroxysmal atrial fibrillation, as an alternative to available antiarrhythmic therapy (Class IIa) [13]. Symptomatic patients with AF refractory to antiarrhythmic drugs can benefit from catheter ablation even if the arrhythmia is persistent (class IIa) or long-standing persistent (class IIb) [13]. Our patient also had

documented atrial flutter which could have been managed through cavotricuspid isthmus ablation during the same procedure (Class I) [13].

Our subject was eager to achieve rapid control of his symptoms and prevention of recurrent syncopes. A second ablation was not immediately available and its success rate would have been debatable due to the presence of atrial enlargement. Therefore, we chose antiarrhythmic drug therapy, indicated in subjects with syncope due to rapid AF (Ic) [4]. Beta blockers had already proven ineffective in preventing our patient's tachyarrhythmias, both prior to hospital admission and after the catheter ablation. The underlying structural heart disease (left ventricular hypertrophy, grade 1 diastolic dysfunction, left atrium enlargement) and also the possible association of coronary microvascular disease argued in favor of amiodarone instead of propafenone (flecainide being unavailable). The second Holter monitoring showed tachycardia-bradycardia syndrome, which supports our choice of implanting a cardiac pacemaker. We will perform a second catheter ablation if the arrhythmia reoccurs or if the patient develops side effects to amiodarone.

Conclusions

Clinical features can suggest the substrate of the WCT but electrocardiographic criteria and electrophysiology studies are indispensable for the definite etiological diagnosis. Occult accessory posteroseptal by-pass tracts can become manifest in patients over 60 years of age, coinciding with the development of sick sinus syndrome, thus requiring a step-by-step therapeutic approach. The cycle length of narrow QRS and LBBB pattern orthodromic supraventricular tachycardias can be similar if the bypass tract is located in the septum, particularly in the left posteroseptal region.

List of abbreviations:

WCT - wide complex tachycardia
SVT - supraventricular tachycardia
VT - ventricular tachycardia
RBBB – right bundle branch block
LBBB - left bundle branch block
AF – atrial fibrillation
SSS – sick sinus syndrome
A-V - atrio-ventricular
bpm - beats per minute

Consent

Written informed consent was obtained from the patient for publication of this case report and accompanying images. A copy of the written consent is available for review by the Editor-in-Chief of this journal.

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