

such cases, the evaluation should be individualized and aimed at ruling out possible cardiac sequelae of the radiotherapy.

Damage to the coronary arteries with the development of heart disease is one of the most common presentations in patients of this type, as reported in several earlier publications referred to previously. In the case of our patient, the presentation did not involve the classic symptoms that would suggest a coronary origin, and there was a low pretest probability of coronary artery disease. Likewise, the initial laboratory tests showed no elevation of biomarkers of myocardial injury, and the electrocardiogram revealed no evidence of dynamic changes indicative of ischemia.

Because there are no specific treatment protocols for these patients, during the hospital stay, we decided to perform tests that would progressively rule out possible cardiac mechanisms involved in the episodes of syncope, taking into consideration, to a certain extent, the risk associated with our patient's profession as a truck driver.

Concerning the electrophysiological study, as Unzué et al point out, the normal HV interval has a duration of 35 ms to 55 ms, and up to 60 ms in the case of left bundle branch block. In our patient, who had right bundle branch block, an HV interval of 65 ms cannot be considered normal. However, as the Wenckebach point was nearly normal and its location was suprahisian, and the predictive value of the progression of a baseline HV of less than 100 ms to atrioventricular block is low, we decided to perform the study with procainamide, with a negative result, as reported in our earlier letter. Thus, at the time, there was no indication for permanent antibradycardia pacing.^{2–4}

Due to the lack of specificity and low diagnostic yield of the initial tests, the decision was made to carry out exercise echocardiography to rule out ischemic heart disease (low pretest probability), while examining a possible dynamic obstruction in the left ventricular outflow tract due to valvular and subvalvular calcification detected on transthoracic echocardiography. During the test, there was no evidence of the usual symptoms suggestive of ischemia. The patient began to reproduce the symptoms that had led to his hospital admission, with a heart rate of 146 bpm, which was accompanied by the appearance of complete atrioventricular block at 45 bpm, with atrioventricular dissociation, which progressed to a 12-second asystole, the tracing of which is shown in Figure 2.¹ Because of limited space, we provided only the tracing that we had considered most relevant.¹

Subsequently, the decision was made to implant a permanent transvenous pacemaker. The choice of this approach was based on the belief that the causal mechanism was related to an infranodal block triggered by exercise.⁵

One month after discharge, the patient underwent cardiac catheterization because he had developed exertional dyspnea. The procedure revealed no obstructive coronary artery lesions. Spirometry confirmed the presence of GOLD stage II chronic obstructive pulmonary disease as the cause of the dyspnea.

Cardiac toxicity secondary to radiotherapy is usually difficult to demonstrate, and the diagnosis is reached after ruling out, to a reasonable extent, the most common causes of cardiac disease. In our patient, cardiac catheterization was not performed to assess the presence of coronary artery disease as the cause of the atrioventricular block because the initial tests and the absence of clinical signs of angina did not point in that direction, and because of the low pretest probability of coronary artery disease. However, its performance could have been an equally valid approach.

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Echocardiographic Diagnosis of Ventricular Tachycardia: Is There a Problem With Clinical and Electrocardiographic Diagnostic Criteria?



Taquicardia ventricular diagnosticada por ecocardiografía: ¿fallan los criterios diagnósticos clínicos y electrocardiográficos?

To the Editor,

We read with interest the *Image in Cardiology* case report by Dr Preza,¹ which summarizes the use of ultrasound imaging to diagnose ventricular tachycardia in a 60-year-old woman with known ischemic heart disease who presented to the emergency room after developing a hemodynamically stable regular tachycardia with a wide QRS. While recognizing the particular appeal of this diagnostic approach, we remain concerned about reliance on this method because of the risk that hemodynamic stability in a

patient with established ischemic heart disease might lead to misdiagnosis of a supraventricular origin. This misdiagnosis persists even though it is well established that > 90% of wide-QRS tachycardias in patients with ischemic heart disease are ventricular² and that hemodynamic tolerance is incapable of distinguishing between ventricular and supraventricular origin.³ In cases of ventricular tachycardia due to bundle branch reentry, the electrocardiogram morphology is normally similar to that in sinus rhythm, and therefore a normal electrocardiogram cannot exclude a ventricular origin. It would have been useful to compare the complete 12-lead electrocardiograms in tachycardia and sinus rhythm in this patient, but nonetheless the leads shown are clearly not identical in the 2 situations (higher S in DIII in sinus rhythm than in tachycardia, and changing peak AVR in DII). Atrioventricular dissociation is present in only 20% to 50% of patients and is sometimes difficult to recognize, so its absence does not aid diagnosis.⁴

In summary, without denying the appeal of ultrasound imaging as a method for diagnosing the origin of wide-QRS tachycardia, it is important to point out that this case was exceptional, and that an accurate diagnosis can be achieved in clinical practice based on patient history and electrocardiography. We should discard criteria such as hemodynamic tolerance that have no diagnostic value and can lead to inappropriate treatment of a regular wide-QRS tachycardia, with serious clinical and prognostic implications.

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Echocardiographic Diagnosis of Ventricular Tachycardia: Is There a Problem With Clinical and Electrocardiographic Diagnostic Criteria? Response



Taquicardia ventricular diagnosticada por ecocardiografía: ¿fallan los criterios diagnósticos clínicos y electrocardiográficos? Respuesta

To the Editor,

I am grateful for the interest shown in the published echocardiography images¹ and agree with the authors that hemodynamic stability in some patients during episodes of ventricular tachycardia can lead to a misdiagnosis of wide-QRS supraventricular tachycardia²; it is of the utmost importance to differentiate between ventricular and supraventricular origin because of the worse prognosis of ventricular tachycardia.³ Nonetheless, the many electrocardiographic algorithms in use have not achieved 100% sensitivity or specificity⁴; moreover, even widely accepted tools such as the Brugada and Verecke criteria do not achieve the sensitivity or specificity of the original reports when applied by emergency physicians or even cardiologists.^{5,6} Furthermore, the specificity of some criteria can be reduced in patients with complete left bundle branch block, as well as in patients with a structurally normal heart.^{4,7}

When present, atrioventricular dissociation is one of the most specific practical criteria for differential diagnosis of ventricular vs supraventricular tachycardia,⁷ and some authors have therefore suggested the potential diagnostic usefulness of echocardiography.^{8–10} The presented case provides an example.

The authors correctly state that clinical and electrocardiographic criteria can establish a diagnosis of ventricular tachycardia in most cases. However, we should keep in mind that resident and emergency physicians have to reach a diagnosis when confronted with acute cases of patients with hemodynamically stable wide-QRS tachycardia; with the echocardiography images presented, my intention was to remind them that they have an additional tool at their disposal for the diagnosis of atrioventricular dissociation.

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