

Syncope in a Patient With a History of Radiotherapy: The Importance of a Comprehensive Assessment of Cardiac Involvement



Síncope en un paciente con antecedente de radioterapia: la importancia de una valoración global de la afección cardíaca

To the Editor,

We have read with great interest the case reported by Jorge-Pérez et al¹ concerning a 44-year-old man with a history of thoracic radiotherapy who presented with episodes of exertional syncope. Given the prolonged survival of patients with Hodgkin's lymphoma and the high radiation doses administered years ago, an increasing number of patients with these characteristics are being referred to cardiology departments. Thus, we consider the case to be of great relevance.

During an exercise echocardiogram, the patient experienced an episode that the authors describe, both in the text and the figure legend, as a "12-second episode of atrioventricular dissociation". This is an inaccurate description of the electrocardiogram since, far from independent atrial and ventricular rhythms, what the recording shows is asystole due to the development of complete atrioventricular block. The clinical picture is interpreted as a paroxysmal atrioventricular block secondary to fibrosis of the conduction system as a consequence of the radiotherapy. However, we consider that, in this case, it would have been advisable to carry out an exhaustive study to rule out the presence of coronary artery disease.

The effect of radiotherapy on the heart has been dealt with extensively and can take many forms: myocardial, coronary, valvular, pericardial, and conduction system involvement. Coronary artery disease is the most common cardiac condition, occurring in up to 8.4% of patients who undergo thoracic radiotherapy, according to the reported series.² The histological findings include interstitial fibrosis and luminal narrowing secondary to intimal proliferation,³ with a predilection for the coronary artery ostia because of the anterior position of the origin of the 2 coronary arteries in the thoracic aorta.^{4–7} In the case reported by Jorge-Pérez et al,¹ this possibility is even more probable because of the severe calcification the mitral and aortic valves and the subvalvular aortic calcification revealed by the echocardiogram.

As has been reported previously, paroxysmal atrioventricular block may be of ischemic origin, which in most published cases corresponds to a disturbance of the His-Purkinje system conduction.⁸ In the patient discussed herein,¹ the involvement of the His-Purkinje system is evident, not only because of the presence of right bundle branch block in the baseline electrocardiogram, but also because of the 65 ms-HV interval measured in the electrophysiological study. In this respect, it is also necessary to point out that the HV interval is by no means "within normal limits", as the authors report, given that this limit has been set at 55 ms (up to 60 ms can be accepted in patients with left bundle branch block).⁹

Atrioventricular block secondary to radiotherapy-induced coronary artery stenosis has been described in previous studies,¹⁰ and the consequences can be serious unless revascularization is undertaken (particularly in this patient because of his profession as a truck driver). Thus, we consider that, in this case of exercise-induced atrioventricular block in the His-Purkinje system, coronary angiography should have been performed to rule out an ischemic cause, before attributing it exclusively to the direct effects of the radiotherapy on the conduction system.

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Syncope in a Patient With a History of Radiotherapy: The Importance of a Comprehensive Assessment of Cardiac Involvement. Response



Síncope en un paciente con antecedente de radioterapia: la importancia de una valoración global de la afección cardíaca. Respuesta

To the Editor,

Having carefully read the contributions of Unzué et al,¹ we would like to add the following comments to ensure a greater understanding of the case.¹

The assessment of cardiovascular symptoms in patients with a history of radiotherapy is complex and, given the low incidence of

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