BRIEF REPORTS

Intermittent Atrioventricular Block in an Accessory Pathway Associated With Complete Infra-Hissian Block

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The appearance of syncope in a patient with an accessory pathway is often a sign of high conduction capacity along the pathway, and calls for urgent ablation. Serious complications may ensue if it is assumed that these patients have an underlying mechanism of tachyarrhythmia, and ablation of the accessory pathway performed hastily without careful electrophysiological evaluation may lead to serious complications. The case described here, despite the patient's unusual presenting features, illustrates that the causal mechanism of syncope is not always what it appears to be. Although our patient had an obvious accessory pathway that was clearly evident in the electrocardiogram, the actual cause of the problem was complete infra-hissian atrioventricular block.

Key words: Syncope. Accessory pathway. Heart block.

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Síncope por bloqueo auriculoventricular completo en un paciente con preexcitación

La aparición de síncope en un paciente portador de una vía accesoria suele ser un signo de la alta capacidad de conducción de la vía y, por tanto, motivar la realización de un procedimento de ablación de forma preferente.

Suponer en estos pacientes un mecanismo taquiarrítmico y proceder a la ablación directa de la vía accesoria sin una mínima evaluación electrofisiológica previa puede llevar a serias complicaciones.

El caso que aquí se muestra, aunque raro en su presentación, ilustra que el mecanismo causal del síncope no siempre es el que aparenta ser, y de ello es ejemplo este paciente en que, a pesar de la presencia de una vía accesoria muy manifiesta en el electrocardiograma, el verdadero mecanismo causal fue un bloqueo auriculoventricular completo infrahisiano.

Palabras clave: Síncope. Vía accesoria. Bloqueo cardíaco.

INTRODUCTION

Syncopal symptoms in a patient with manifest preexcitation in the electrocardiogram (ECG) usually indicate an accessory pathway contributing to the mechanism of syncope,¹ either through orthodromic or antidromic reentry or through atrial fibrillation, with fast conduction to the ventricle over the accessory pathway. Although these mechanisms directly or indirectly through extracardiac factors² explain syncope in a considerable percentage of patients with preexcitation, the search for the causal mechanism should be based on a rational interpretation of the full medical

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Received 30 October, 2003. Accepted for publication 8 January, 2004. history, and not only on the presence of an accessory pathway.

We present the case of a patient admitted for syncope, in which the causal mechanism was complete infra-hisian atrioventricular block (AVB), although atrioventricular (AV) conduction was maintained through an accessory pathway. In our literature review, we found only 2 cases with similar clinical and electrophysiological characteristics.^{3,4}

CASE STUDY

A 59-year-old man, with no prior history or relevant symptoms presented with an episode of sudden loss of consciousness while seated, with no prodromes, experiencing immediate spontaneous recovery and accompanying head injury.

An ECG (Figure 1) performed on arrival to the hospital showed sinus rhythm, a short PR interval and widened QRS complex, a positive delta wave in V1 and I

ABBREVIATIONS

ECG: electrocardiogram. AVB: atrioventricular block. AV: atrioventricular. EPS: electrophysiological study. AH: atrial-His bundle. HV: His-ventricular.

and negative delta in the inferior wall, indicating the presence of a left posterolateral accessory pathway.

The medical history indicated that the patient had never experienced palpitations, not even immediately before the syncopal episode.

Based on the ECG and the clear cardiogenic nature of the episode, the patient was admitted with suspected tachyarrhythmic syncope to undergo electrophysiological study (EPS) and ablation of the accessory pathway.

For the EPS, three quadripolar catheters were inserted using the Seldinger technique via the right femoral vein, then positioned in the right atrium, the bundle of His and the right ventricle. An octapolar catheter was also introduced through the left antecubital vein and advanced (with difficulty) to the coronary sinus for characterization of the mitral annulus.

The intracardiac recordings showed a normal atrial-His bundle (AH) interval and negative His-ventricular (HV) interval of -11 ms. Early ventricular activation indicative of a left posterolateral accessory pathway was noted in the catheter's distal pair, which had not advanced fully into the coronary sinus.

Atrial stimulation was initiated with a 500-ms cycle. At first, each atrial impulse was conducted to the ventricle over the accessory pathway (Figure 2A). After a few seconds, however, stimulated atrial activity followed by His activity was observed, although conduction to the ventricle by either the accessory pathway or the His-Purkinje system (Figure 2B) did not occur.

Continuing with the same cycle, a gradual 4:1 increase in the accessory pathway block was observed (Figure 3), showing that only AV conduction was possible over this pathway.

Based on these data, it was evident that the true mechanism of syncope was complete infra-hisian AVB in a patient with an abnormal accessory pathway, as shown by intermittent advanced accessory pathway block with long stimulation cycles (500 ms).

Because of the reduced capacity of anterograde conduction over the accessory pathway, atrioventricular dissociation with ventricular stimulation, and concomitant infra-hisian block, pathway ablation was ruled out and a DDD pacemaker was implanted the following day.

DISCUSSION

Two aspects of our case were of interest: age and the absence of palpitations. It is known that the conduction properties of the accessory pathways worsen



Fig. 1. 12-lead electrocardiogram showing a short PR interval and a QRS complex with highly evident delta wave suggestive of left posterolateral accessory pathway.

Fig. 2. A: atrial activity stimulated with 500-ms cvcle and early ventricular activation in the distal coronary sinus. All stimulated atrial beats are followed by the bundle of His (H) and ventricle ("S" is the stimulation artifact in the surface ECG and "QRS" is the ventricular complex in the surface ECG). B: the stimulation of atrial beats followed by the His bundle, but without conduction to the ventricle (the arrows indicate the absence of ventricular activity). From top to bottom, bipolar intracardiac electrocardiographic and electrophysiologic recordings (100 mm/s) obtained from the high right atrium (HRA), distal His region (HIS ds) and proximal His region (HIS px), from the distal coronary sinus (CSd) to the proximal (CS4) and from the right ventricular apex (RVA).







with age, the development of tachycardia decreases over time and preexcitation can even disappear.⁵ If, in addition to patient age, there is no previous history of palpitations, the initial diagnostic orientation should probably have focused on mechanisms other than the accessory pathway. Several retrospective studies, such as one by Farré et al,⁶ show that syncope unrelated to the accessory pathway accounts for a large percentage of patients with preexcitation. The presence of manifest preexcitation in the ECG may also indicate a conduction disorder, as this finding is not usual in left-sided accessory pathways because of the distance from the sinus node.

All of these considerations show the value of an accurate medical history to assist in the diagnosis, as well as the importance of always considering syncope to be of unknown origin unless a direct relationship between an electrocardiographic or electrophysiological feature and the syncopal episode can be clearly established. In this case, if we had assumed a tachyarrhythmic mechanism without question because of the preexcitation found in the ECG, the patient would have been treated with a Class IC antiarrhythmic agent, thereby suppressing conduction over the accessory pathway and iatrogenically unmasking the AVB.

In the baseline intervals of the EPS, we found a long local HV interval in the His-bundle catheter, leading us to suspect that the septal ventricle was being activated over the accessory pathway and not over the specific system, as the result of some kind of block in this location.

The conduction properties of the accessory pathway of this patient merit several comments. Although the usefulness of EPS prior to ablation in patients with Wolff-Parkinson-White syndrome7 has been questioned, this case shows that a simple electrophysiological assessment prior to direct accessory pathway ablation can completely change the therapeutic option. The conduction capacity of this patient's accessory pathway was so deteriorated that continuous atrial stimulation alone demonstrated advanced block and indicated complete infra-hisian AVB. Although no pre-admission ECG was available for this patient, the clinical data indicate that the accessory pathway would only manifest when complete infra-hisian AVB appeared. In this case, the accessory pathway was an alternative, opportune mechanism that maintained AV conduction, preventing repeated syncopal episodes. In addition, ventriculoatrial dissociation with ventricular stimulation showed no capacity for retrograde conduction. This led to a major change in the approach, as accessory pathway ablation was excluded.

Although EPS prior to ablation provides valuable information, the concomitant block might not have been shown in the EPS if good accessory pathway conduction had been present. Thus, ablation would have been performed, immediately leading to the rhythm of complete atrioventricular block.

The latter assumption/supposition is a risk that must be assumed when performing ablation of an accessory pathway with higher conduction capacity and an effective refractory period theoretically shorter than that of the AV node, as in a case study reported by García Pinilla et al,⁸ in which the infra-hisian conduction disorder became evident only after accessory pathway ablation.

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