Short Communication

Diagnostic Superiority of Continuous-Loop Electrocardiographic Recording over Other Testing in a Patient with Recurrent Syncope and Underlying Coronary Artery Disease with Severe Left Ventricular Dysfunction

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Summary: Dysrhythmic causes of syncope may elude diagnosis in postinfarction patients despite elaborate testing, including electrophysiologic study. For a correct diagnosis, capture of cardiac rhythm during patient's typical symptoms is crucial. This report describes a patient with coronary artery disease and decreased left ventricular ejection fraction who experienced recurrent syncopal episodes without obvious precipitating factors. The 12-lead electrocardiogram showed left bundle-branch block indicating a possible conduction abnormality as the underlying cause of syncope. Twenty-four-h Holter monitoring exhibited no sinus rhythm or conduction disturbances but revealed a nonsustained run of ventricular tachycardia. Findings at electrophysiologic testing led to a presumptive diagnosis of tachyarrhythmic cause of syncope; however, the correct diagnosis was only made with use of a loop monitor which documented a 15-s sinus pause during a syncopal episode.

Key words: syncope, left ventricular dysfunction, loop electrocardiogram recorder, electrophysiology

Introduction

Judicious diagnostic evaluation of syncope in patients with underlying coronary artery disease (CAD) and depressed left

ventricular (LV) ejection fraction is crucial to identify the cause and to direct proper therapy. In the vast majority of these patients, a methodological work-up with newer noninvasive and invasive testing including electrophysiologic study (EPS) can determine the correct diagnosis.^{1,2} The remaining patients who are designated as having "syncope of unknown origin" pose a diagnostic challenge and therapeutic dilemma for all clinicians.^{1–3} These patients may portend a poor prognosis with increased mortality if serious arrhythmic causes have been missed.^{3–5} On the other hand, confirmation of a nonarrhythmic etiology will reassure both physician and patient.

We report the usefulness of the loop electrocardiographic (ECG) recording in verifying a bradyarrhythmic cause of recurrent syncope in a patient with CAD and severe LV dysfunction in whom all other tests failed to identify the correct etiology and pharmacologic treatment toward a presumptive diagnosis, guided by EPS, did not avert syncopal recurrences.

Case Report

A 73-year-old man was admitted to our hospital because of three syncopal episodes in the preceding 5 months, one of which resulted in serious head injury. These spells developed abruptly and were not preceded by physical exertion or palpitations. His past medical history included an old myocardial infarction and coronary bypass graft surgery 5 years earlier. He had never taken antiarrhythmic drugs, and laboratory investigations were normal. During sinus rhythm, the 12-lead ECG exhibited a complete left bundle-branch block (LBBB) with left axis deviation. Twenty-four h Holter monitoring exposed an episode of nonsustained monomorphic ventricular tachycardia (VT) of 9 QRS complexes with a mean cycle length of 350 ms. There was no treadmill exercise-induced tachyarrhythmia, and the upright tilt-table testing was negative. Due to a positive thallium scanning, coronary angiography was performed which showed triple-vessel disease with patent grafts (left internal mammary and two saphenous vein grafts) and a dilated and hypokinetic left ventricle with a low ejection fraction of 25%.

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FIG. 1 Rhythm strip with continuous recording from the loop monitor in the patient with recurrent syncope, showing sinus arrest of 15 s duration during a syncopal episode.

During the baseline EPS there was no evidence of sinus node dysfunction or atrioventricular conduction disturbances. The H-V interval measured 55 ms. Programmed right apex ventricular stimulation using up to two extrastimuli elicited reproducible runs of nonsustained VT, and a polymorphic VT with two extrastimuli which rapidly degenerated into ventricular fibrillation which was restored by direct current (DC) shock. Amiodarone therapy was then instituted at an oral dose of 1.800 mg/day for 1 week. The follow-up EPS evaluation showed no conduction abnormality or infra-His block and reproduced no ventricular arrhythmia. However, the patient experienced two more syncopal episodes after his discharge, which prompted repeat Holter monitoring and a second follow-up EPS that were entirely negative. He was then provided with a cardiac continuous-loop event recorder ("King of Hearts" monitor, Instromedix, Inc., San Diego, Calif., USA). Two weeks later, he experienced one more syncopal episode during which sinus arrest with a 15-s pause was documented that was preceded and followed by junctional bradycardia (Fig. 1). In light of this finding, we proceeded to a dual-chamber DDD pacemaker implantation.

Discussion

During the last decade, we have witnessed major advances in diagnosing previously "unexplained" syncope with the expanded use of signal-averaged electrocardiography, head-up tilt testing, and EPS.^{1,2} In particular, EPS has evolved as the procedure of choice for exposing symptomatic tachyarrhythmias in patients with previous myocardial infarction and LV dysfunction.^{6,7} However, EPS has certain limitations,⁸ and findings during these tests provide only presumptive diagnoses, since they fail to document the cardiac rhythm problem during the clinical event or to reproduce the patient's symptoms accurately.

In the present case, ECG findings of bifascicular block (LBBB) indicated a possible bradyarrhythmic cause of syn-

cope related to potential high-degree atrioventricular block; however, this was not supported by EPS. Findings on Holter monitoring (nonsustained VT) pointed to a ventricular tachyarrhythmic cause of syncope, particularly in view of the underlying CAD and severe LV dysfunction. Subsequently, EPS further supported such a presumptive diagnosis with easily induced sustained ventricular tachyarrhythmia by a less aggressive stimulation from the right ventricular apex with only two extrastimuli. Thus, the decision was made to treat the patient with amiodarone, and the result of follow-up EPS was satisfactory since amiodarone treatment rendered the patient's arrhythmia noninducible.

However, the patient's clinical course disproved all these presumptive diagnoses, despite the fact that there were all based on "solid" evidence, since syncope recurred 3 weeks later, shortly after his discharge from the hospital. Due to pre-existing LBBB, a new presumptive diagnosis of possible aggravated conduction abnormality was initially suspected. However, repeat EPS failed once more to document intra- or infra-His conduction block. Subsequently, during a recurrence, the correct diagnosis, never suspected before, was only revealed by the loop monitor, which documented a 15-s pause as the patient's cause of syncope (Fig. 1).

Loop ECG recorders offer the ability to "store" the cardiac rhythm at the time of patient's symptoms, providing a direct causal correlation for confirming or excluding serious rhythm abnormalities. These monitors continuously record and erase, preserving in digital memory maximal 5 min of ECG rhythm for a programmable time period before and after the patient activates an event button. The recorded strip can also be transmitted over the telephone for interpretation. These devices have been found to provide significant diagnostic yield of 6 to 36% in patients with recurrent syncope or presyncope during the first 2 to 4 weeks of monitoring.^{9,10}

This case underscores the superiority of loop ECG recording in identifying the exact cause of syncope in a patient post infarction with LV dysfunction portending a high tachyarrhythmia risk, whereby other tests including EPS were totally misleading.

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