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Letter to the Editor

Myocardial stunning associated with a myocardial bridge

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A myocardial bridge is a discrete systolic constriction of a coronary artery, most commonly the left anterior descending, caused by some myocardial tissue ‘bridging’ the vessel [1]. Regarded for long time as innocent anatomic variants [1], myocardial bridges have been subsequently acknowledged as potential causes of angina, myocardial infarction and sudden death [2,3].

A 67-year-old woman with no coronary risk factor and a 2-year history of substernal pain on effort never previously evaluated, was referred to our Department after recurrent episodes of pain that had been attenuating or exacerbating over a 2-h period, with reduced or increased intensity of physical exercise.

On first examination, the pain had resolved and a 1–4-mm ST segment elevation in DI–DIII and V3–6 leads (Fig. 1A) was recorded, while echocardiography demonstrated a left ventricular antero-lateral akinesia with apical dyskinesia (LVEF 47%). At coronary angiography an isolated, 20-mm long myocardial bridge was causing a 75–80% systolic constriction of the mid left anterior descending branch (Fig. 2A,B). Serial myocardial enzymes were

normal and tests for viral infections screened negative. She received oral aspirin (100 mg daily) and propranolol (20 mg t.i.d.) and remained asymptomatic until discharge, 4 days later.

ECG evolutionary changes consisted of a slow regression of ST-segment elevation with appearance of negative T waves and no Q waves (Fig. 1B). At pre-discharge echocardiography, the left ventricular apex was no longer dyskinetic but rather akinetic, with other findings unchanged, while viability of

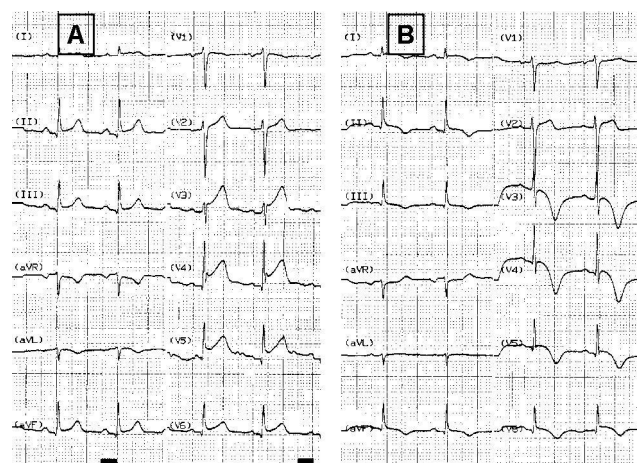


Fig. 1. Twelve-lead ECG on admission to CCU (A) and 4 days later, immediately prior to discharge (B).

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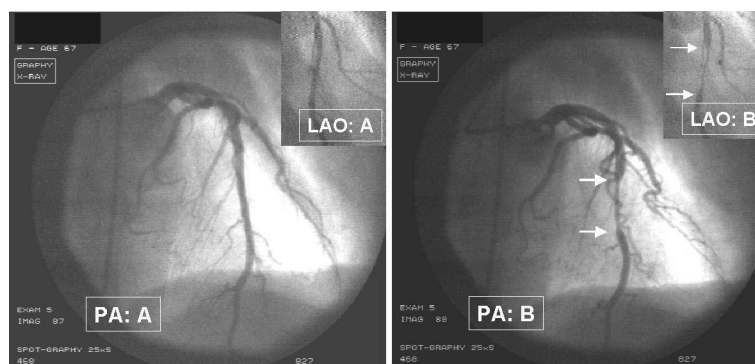


Fig. 2. End-diastolic (A) and end-systolic (B) frames of left coronary angiography (postero-anterior cranial [PA] and [small insert in the upper right corner] left anterior oblique, [LAO] cranial views) showing a myocardial bridge of the mid portion of left anterior descending coronary artery (white arrows).

anterior mid wall was suggested by a dobutamine stress-test. After 3 months, she had remained asymptomatic while still on aspirin and propranolol, and left ventricular wall motion was completely normalized (LVEF 54%).

Conflicting observations have been reported on the clinical consequences of myocardial bridges. In autopsical series, fewer myocardial infarctions were seen in patients with than in those without bridges [1], and an excellent long-term prognosis was reported for patients with angiographically detected, isolated bridges [4]. Conversely, others have reported that bridges can produce events [2,3]. Exercise-induced tachycardia exceeding 100–120 beats/min, such as presumably occurred in our case, has been identified as the main factor precipitating myocardial ischemia by increasing the relative importance of systolic coronary flow, which is limited by the bridge [5]. An alternative hypothesis is that myocardial bridges cause a systolic vascular kinking with endothelial damage leading to vasospasm or thrombosis [6,7]. Therefore, we might hypothesize that the ST-segment elevation observed in our patient was produced by ischemia secondary to a coronary spasm or thrombosis at the level of the bridge. The slow regression of ST segment elevation, and the normality of serum cardiac enzymes observed in our patient, contrast with both hypotheses of a prolonged spasm or of thrombosis with eventual spontaneous lysis. A coronary thrombosis complicating a myocardial bridging of the mid left anterior descending branch is also

unlikely since, in general, the closer the bridge to the left coronary ostium, the greater the extent of intimal injury [8]. Reversible left ventricular dyskinesia attributable to myocardial stunning has been previously described [9]. Thus, we hypothesized that the slowly resolving ST segment elevation was the expression of ventricular dyskinesia secondary to myocardial stunning associated with a prolonged ischemia that was produced by exercise-induced tachycardia. This interpretation is supported by the disappearance of ST segment elevation along with regression of apical dyskinesia, the detection of myocardial viability at dobutamine stress test, and the favorable long-term effects of β -blockers on both symptoms and ventricular asynergy.

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