

Left ventricular ballooning syndrome due to vasospasm of the middle portion of the left anterior descending coronary artery

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Abstract

We report a case that shows vasospasm to be one of the mechanisms responsible for left ventricular ballooning syndrome. Our case suggests that identical ventriculographic findings in patients with tako-tsubo syndrome and those with coronary vasospasm of a long left anterior descending coronary artery may be due to a common etiology. (Cardiol J 2012; 19, 3: 314–316)

Key words: left ventricular ballooning syndrome, vasospasm

Introduction

Since the first description of left ventricular ballooning syndrome (LVBS), otherwise known as tako-tsubo syndrome, coronary vasospasm has been put forward as one of the pathophysiological mechanisms involved [1–5]. This is a rare syndrome [6] and little information is available on its treatment and long-term prognosis.

Case report

We report the case of a 53 year-old woman, with hypercholesterolemia of 220 mg/dL as the only known cardiovascular risk factor, who originally consulted in 2003 due to nocturnal, prolonged angina-like chest pain, unrelated to any form of stress. Electrocardiogram (ECG) at admission showed a pattern of subepicardial lesion in precordial leads V1 to V6 and inferior leads II, III, and aVF (Fig. 1A). Emergency coronary angiography showed lesion-free coronary arteries, a long left anterior descending (LAD) artery that wrapped around the cardiac

apex, with reduced diameter from the mid-distal portion (Fig. 1B), and a typical image of tako-tsubo syndrome (Fig. 1C), left ventricular end-diastolic pressure of 21 mm Hg and an ejection fraction of 50%. Transthoracic echocardiogram showed akinesia of the septal-mid-apical, antero-apical and infero-apical segments. Peak CPK was 760 IU/L, CK-MB 74 IU/L and troponin I 20 ng/mL.

After initiating treatment with intravenous nitroglycerin (subsequently suspended due to headache), and diltiazem 300 mg/day, Q waves appeared in leads II, III, aVF, and V2–V6 with positive T wave persistence but also ST segment depression > 70%. However, over the following few days, vector recovery was observed in all the affected leads, along with improved echocardiographic parameters.

Until 2010, the patient was asymptomatic, receiving single daily doses of diltiazem 300 mg, aspirin 100 mg, enalapril 10 mg and simvastatin 40 mg. She then consulted for a new episode of anginal pain, similar to that previously experienced but less intense, and again unrelated to stress. The ECG showed no significant alterations (Fig. 2A), necrosis

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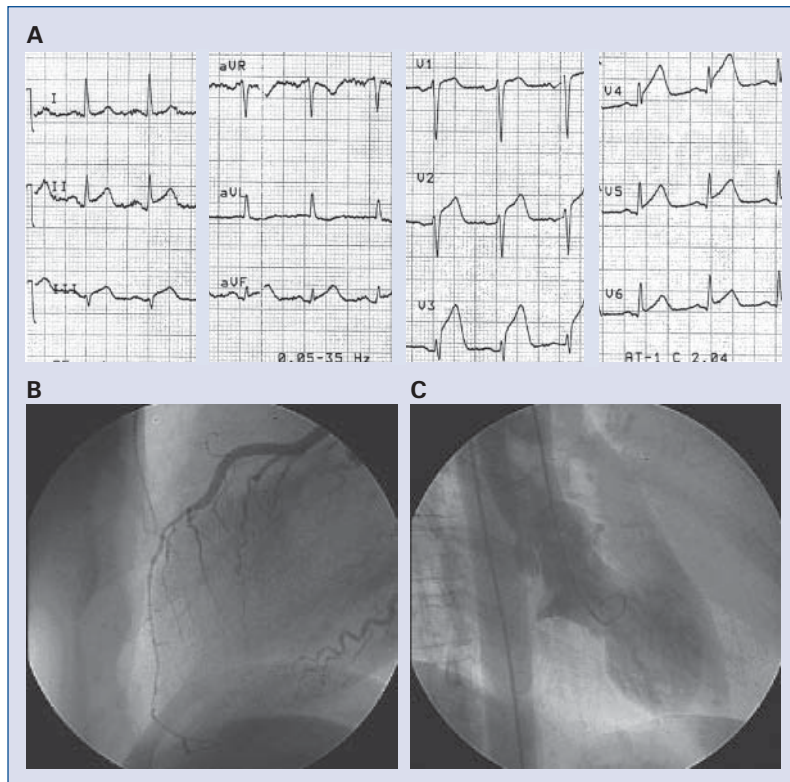


Figure 1. A. Electrocardiography at admission shows a pattern of subepicardial lesion in all precordial and inferior leads; B. Cardiac ventriculography shows a spasm of the middle portion of a long left anterior descending artery wrapped around the cardiac apex; C. Cardiac ventriculography shows a typical image of left ventricular ballooning.

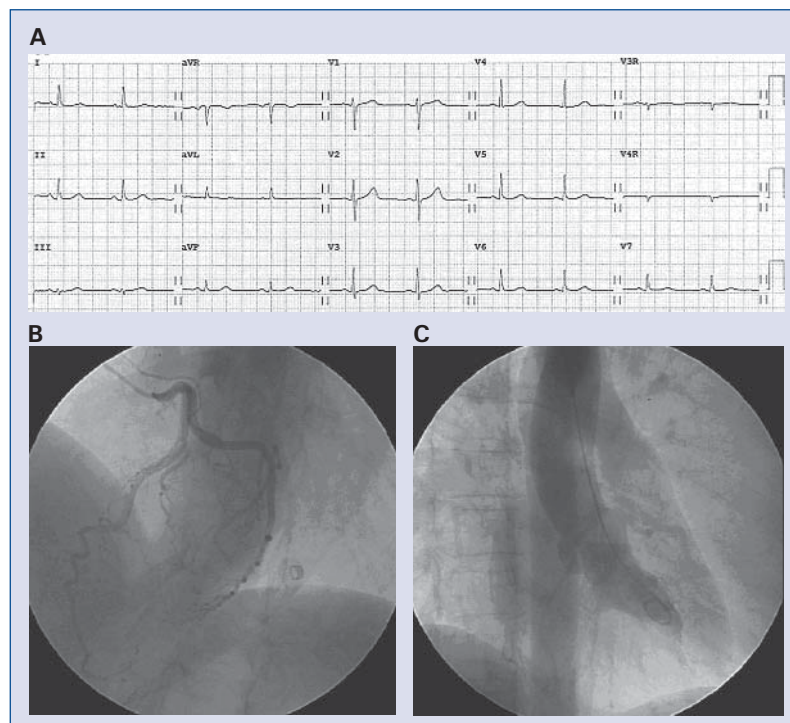


Figure 2. A. Electrocardiographic tracing with normal characteristics; B. Cardiac ventriculography shows no evidence of the coronary artery spasm observed on the previous admission; C. Cardiac ventriculography shows no evidence of the left ventricular ballooning observed on the previous admission.

markers were negative, there was no evidence of coronary vasospasm (Fig. 2B), and no signs of tako-tsubo syndrome in the ventriculogram (Fig. 2C).

Discussion

Ibanez et al. [7] suggested a common etiology in ischemic tako-tsubo syndrome and acute myocardial infarction secondary to LAD artery occlusion, both with a long wrap-around LAD artery, since cardiac ventriculography findings are identical. Furthermore, in five cases of tako-tsubo syndrome [8] with insignificant angiographic atherosclerosis, the same authors reported that intravascular ultrasound detected a non-occlusive plaque in the middle portion of the LAD artery, which suggests that in some cases the appearance of LVBS is actually an acute coronary syndrome with early reperfusion (aborted infarct) in patients with a long wrap-around LAD artery.

According to some authors [9], vasospasm in a single artery as the pathophysiological mechanism of LVBS is improbable because the affected territory is too limited. However, in our case, a spasm located in the middle portion of a long LAD artery that wrapped around the cardiac apex gave rise to the LVBS.

This case illustrates that vasospasm is one of the mechanisms responsible for LVBS, and that treatment with calcium antagonists is effective in maintaining the patient asymptomatic for long periods of time, but also that there may be subsequent episodes of less intense chest pain without hemodynamic compromise.

Several aspects of this case are noteworthy. First, angina-like chest pain was not preceded by an episode of emotional or physical stress, which may play a key role in the pathogenesis of the disorder. Secondly, the recurrence of chest pain occurs in no more than 10% of cases, according to previous studies [10]. Thirdly, the prognosis was good [10].

Our case suggests that the identical ventriculographic findings in patients with tako-tsubo syndrome and those with coronary vasospasm of a long LAD coronary artery may be due to a common etiology.

Conflict of interest: none declared

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