

Apical Ballooning Syndrome Secondary to Nasal Decongestant Abuse

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We describe a typical case of apical ballooning syndrome in an octogenarian female patient with left ventricular wall motion abnormality on electrocardiography, whose ventricular function returned to normal. The patient has allergic rhinitis and had used nasal decongestant excessively a few hours prior to the episode of pain.

Introduction

Since the apical ballooning syndrome (also known as broken heart and *tako-tsubo* syndrome) was first described in Japan, innumerable cases have been reported worldwide. This condition more often affects elderly female patients in the seventh and eighth decades of life, leading to a typical abnormality on left ventriculography in the absence of coronary obstruction¹. We report a typical case of apical ballooning possibly caused by nasal decongestant abuse.

Case Report

An 84-year-old female patient born in Curitiba – Paraná, Brazil, was admitted to the chest pain unit of *Santa Casa de Curitiba* with severe typical chest pain in the sternal region, with no radiation, accompanied by unspecific headache lasting for four hours. The patient reported that the pain had begun at rest and denied emotional stress prior to the episode. She also had hypertension and allergic rhinitis. She reported regular use of clonidine, bromazepam and omeprazole. The patient was a smoker for 30 years and had quit for one year. Physical examination revealed a patient in good general state of health, lucid and oriented, mucous membrane pink, no respiratory distress, blood pressure level of 110 x 70 mmHg on admission, heart rate of 78 bpm, and respiratory rate of 13 rpm. Cardiovascular and pulmonary examinations were unremarkable.

Key Words

Myocardial infarction; acute coronary syndrome; adrenergic agonists; myocardial stunning.

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The electrocardiogram (ECG) showed ST-segment elevation in the anterolateral region (Figure 1A). Based on the clinical and electrocardiographic findings, a hemodynamic study was immediately indicated, and a primary angioplasty was performed. The left ventriculography showed antero-infero-apical dyskinesia (Figure 2A). The right coronary and the circumflex arteries were tortuous and had no obstructive lesions, with right coronary dominance. The anterior descending artery was type III (continued across the left ventricular apex) and had no angiographic lesions (Figure 3). In view of this finding, intracoronary ultrasound was performed in order to establish the diagnosis. This showed a plaque in the mid-third of the anterior descending artery, with a lumen area of 8.7 mm² and vessel area of 13.9 mm² (Figure 4). Ultrasonography showed a plaque with predominantly fibrotic characteristics and no signs of rupture. Based on these findings, medical treatment was chosen.

During the hospital course, no elevation of cardiac enzymes was observed (maximum peak CK-MB 10 U/L, CPK 66 U/L) and the remaining laboratory tests were considered as being within normal limits. Another ECG was performed on the second day of hospitalization (Figure 1B), showing regression of the initial alterations. On the fourth day of hospitalization, two-dimensional transthoracic echocardiography still showed abnormal segmental wall motion of the anterior and apical walls, in addition to left ventricular hypertrophy and grade-I diastolic dysfunction. She had a favorable outcome and was discharged on the sixth day after the index event.

In the follow-up, two months later, the patient presented a new episode of severe chest pain similar to the previous episode, which lasted 2 hours. No significant changes were observed on the electrocardiogram. Worthy of attention, on that occasion, was the fact that the patient was presenting allergic rhinitis with significant nasal obstruction and using high doses of Afrin® (oxymetazoline hydrochloride 0.05% solution, 20-mL bottle) (one bottle in less than 12 hours). She underwent another hemodynamic study that showed that the alteration found in the baseline ventriculography had resolved (Figure 2B). When asked about the first event, the patient reported that she also had presented worsened allergy and used high doses of Afrin®. This medication was discontinued and the medical treatment with calcium channel blockers, angiotensin-converting enzyme, statin and antiplatelet drugs was maintained. Topical corticosteroid was prescribed for the allergic rhinitis. The patient improved and is currently asymptomatic.

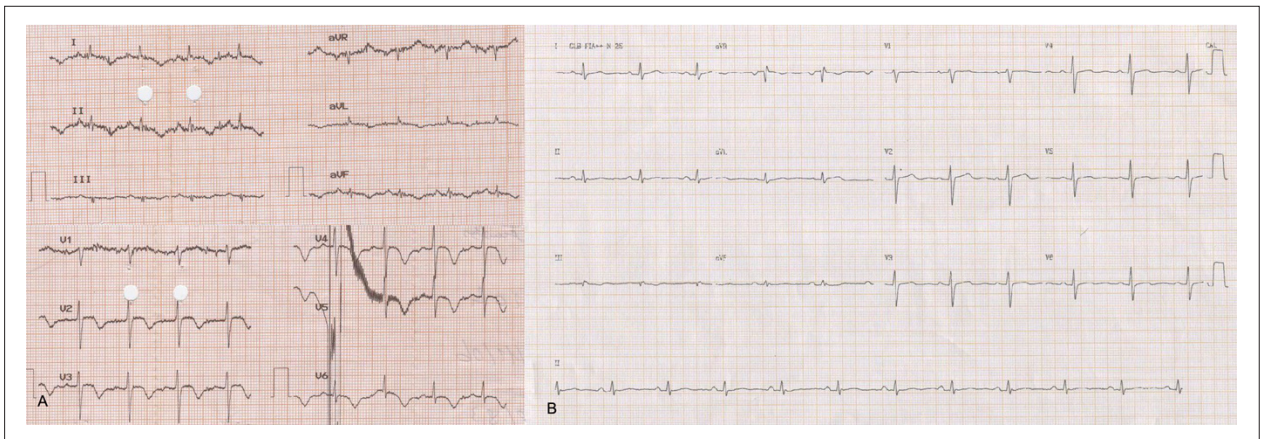


Figure 1 - 12-lead electrocardiogram; A - ECG at admission to the chest pain unit of Santa Casa. B - ECG one day after admission, showing that the previous alterations returned to normal.

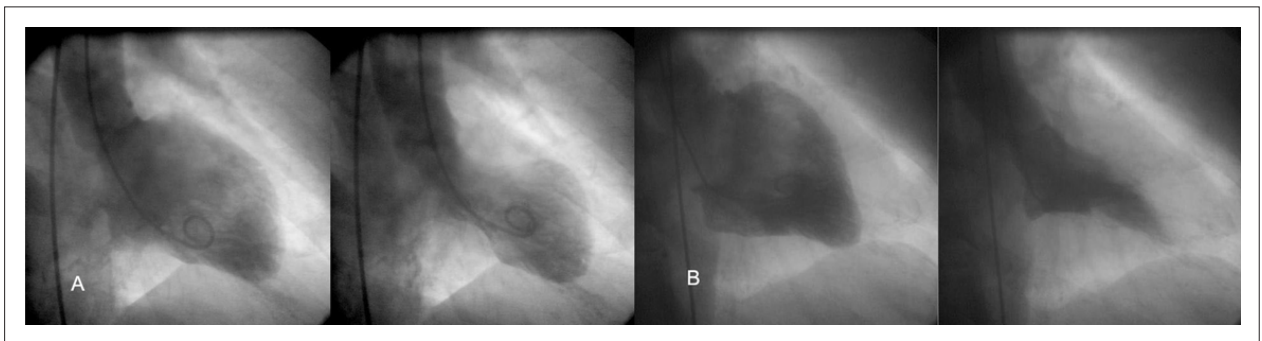


Figure 2 - Ventriculography in right anterior oblique view. A - Ventriculography performed in the acute event showing apical ballooning. B - Study performed at 2 months, showing normal myocardial contractility.

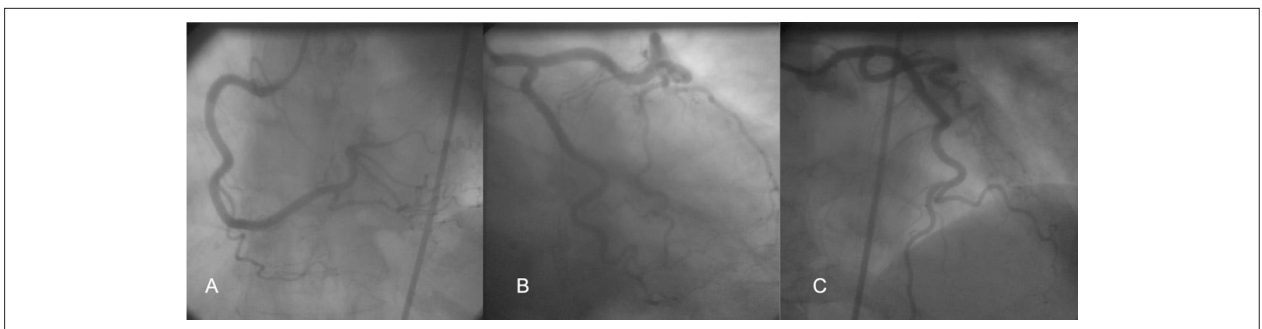


Figure 3 - Coronary angiography: A - right coronary in cranial LAO view; B - circumflex artery in caudal RAO view; C - anterior descending artery in cranial PA view; LAO - left anterior oblique; RAO - right anterior oblique; PA - postero-anterior.

Discussion

Oxymetazoline belongs to the imidazoline class. Thanks to its adrenergic properties (vasoconstrictor), it is frequently used as a component of nasal decongestants and is known as an over-the-counter medication² (no medical prescription required for purchase). At low serum levels, this drug stimulates mainly α_2 - adrenergic receptors of the central nervous system; at higher doses, peripheral alpha-adrenergic receptors are predominantly stimulated. At usual doses, the

major side effects are neurological manifestations; at higher doses, hypertension (secondary to vasoconstriction) and tachyarrhythmias arise.

To date, no acute coronary syndromes related to the chronic use of oxymetazoline have been described. Because of its vasoconstrictor effect, of the temporal relationship with the coronary syndrome, and of the absence of critical coronary lesions both on coronary angiography and ultrasonography, a cause-effect relationship is suggested. In the literature,

Case Report

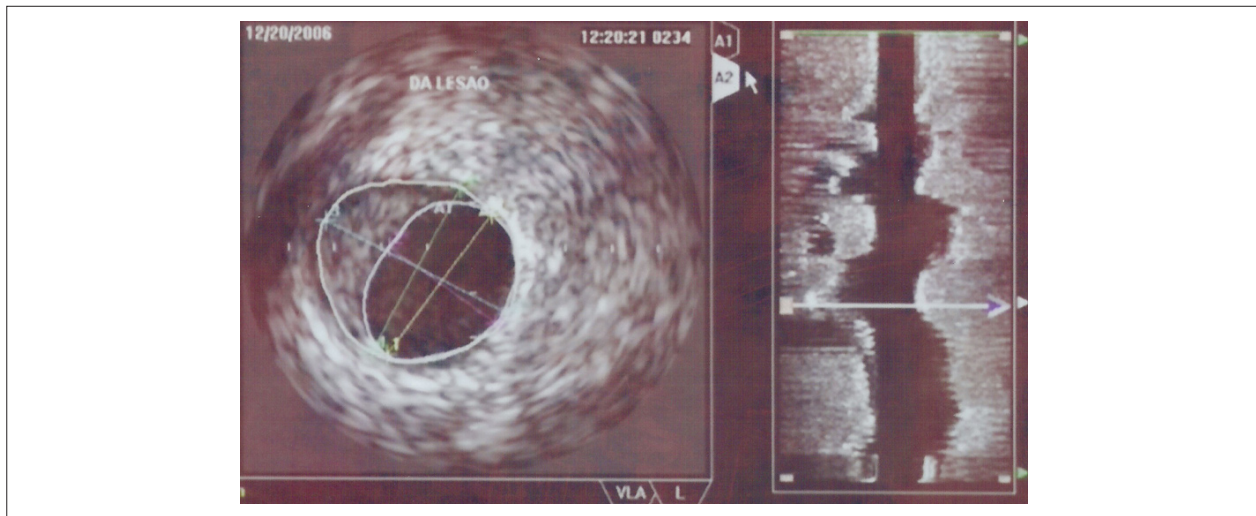


Figure 4 - Intracoronary ultrasound of the anterior descending artery showing an obstructive lesion in its mid segment, with lumen area of 8.7 mm².

several reports correlate vasoconstrictor substances with acute coronary syndrome³ and coronary spasm as the most probable mechanism. Recovery of myocardial contractility in the follow-up suggests myocardial stunning in response to an acute ischemic process.

Sympathomimetic drug abuse mainly leads to elevation of blood pressure levels due to the vasoconstrictor effect of these drugs. In general, intravenous medications such as sodium nitroprusside are mandatory to manage the event². Alphablockers such as phenoxybenzamine have also proven effective. In the management of tachyarrhythmias, betablockers should not be used alone, because the alpha-mediated effects may be exacerbated. Myocardial ischemia is caused by coronary vasoconstriction in most of these cases. We remark that vasoconstriction is a risk factor for coronary dissection, thrombosis due to stasis, and atherosclerotic plaque rupture.

Based on the diagnostic criteria proposed by Abe and Kondo⁴ (Chart 1), the presence of dynamic ST-segment abnormality on ECG, left ventricular apical ballooning on left ventriculography, and the patient's epidemiological characteristics (woman older than 70 years of age) establish the diagnosis of *tako-tsubo* syndrome^{5,6}. These authors suggest that catecholamines play a key role in the genesis of the syndrome. In the present case, these amines originated from external sources (nasal decongestants).

Approximately 0.5% to 1% of the patients admitted with chest pain and ST-segment elevation do not present with significant angiographic lesions. Because of the limitation of angiography in detecting atherosclerotic plaques, especially with positive remodeling, we chose the intracoronary ultrasonographic study which showed a small atherosclerotic plaque in the mid-third. This aspect was also observed in Ibanez et al's study⁶ of five cases presenting atherosclerotic plaque in the mid-third, thus suggesting that the acute event

results from the rapid resolution of both atherothrombotic and spastic coronary occlusion.

The prognosis was favorable in Bory et al's study⁷, in which 277 patients with coronary vasospasm were followed-up. In the 89-month follow-up of these patients, the following was observed: 6.5% presented acute myocardial infarction (AMI) and 39% had recurrent symptoms despite the use of medication. In the present case, only when the nasal decongestant was discontinued did the patient present improvement of the symptoms, thus showing a probable causal relation between the use of sympathomimetic drug and acute coronary syndrome and apical ballooning. The easy access to the medication, the fact that a medical prescription is not required for its sale, and its indiscriminate use may lead to the development of new cases.

Major criteria

- Transient apical ballooning with hypercontractility of the basal segments.
- ST-segment abnormalities on ECG mimicking acute myocardial infarction

Minor criteria

- Physical or emotional stress as triggering factors
- Minor cardiac enzyme elevation
- Chest pain

Exclusion criteria

- Ischemic myocardial stunning
- Subarachnoid hemorrhage
- Pheochromocytoma crisis
- Acute myocarditis.
- Tachycardiomyopathy

Chart 1 - Diagnostic criteria for the *tako-tsubo* syndrome

Potential Conflict of Interest

No potential conflict of interest relevant to this article was reported.

Sources of Funding

There were no external funding sources for this study.

Study Association

This study is not associated with any post-graduation program.

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