Asystole During Exercise Stress Test: Analysis of Cardioinhibitory Response

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This is the case of asystole during stress test at the stage of physical stress in a 30-year-old man with previous history of syncope. The episode was related to a cardioinhibitory response with asystole during the exercise test stress stage, which is not frequent in the literature.

Introduction

Episodes of vasovagal reaction are described as part of the main complications during exercise test and may range from a brief episode of hypotension to extreme cases, translated by asystole.

Cardiac asystole related to exercise in patients without structural heart disease is uncommon in clinical practice. In the literature, cases of asystole most often occur outside the period of stress, most commonly in the recovery stage of the exercise test.

We report the case of a young patient without a structural cardiac disease who presented cardiac asystole in the stress stage of the exercise test.

Case Report

Hypertensive, dyslipidemic 30-year-old man with obstructive sleep apnea and grade III obesity (BMI = 41 kg/m²). The patient reported an episode of syncope during exertion 13 days before the exercise stress test.

Electrocardiogram at rest showed sinus rhythm with no significant abnormalities. During the exercise test, the patient showed no signs of ischemia or tachyarrhythmia. The patient presented Mobitz II 2nd degree atioventricular block (Figure 1) and evolved with cardiorespiratory arrest (CRA) in asystole (Figure 1) lasting 20 seconds in the fifth minute of the test. Immediately submitted to cardiopulmonary resuscitation (CPR), followed by endotracheal intubation with reversion to sinus rhythm. Post-PCR electrocardiogram showed signs of ischemia on the inferior wall. Transthoracic echocardiogram performed afterwards revealed global preserved segmental contractility without structural abnormalities.

The patient was sent by plane to Instituto de Cardiologia de Santa Catarina (ICSC), a state reference for high complexity cardiology cases. Soon after admission, the patient underwent coronary angiography, which showed the circumflex artery originating from the right coronary artery (Figure 2) and absence of obstructive lesions. Chest computed tomography angiography showed no signs of pulmonary thromboembolism. Myocardial perfusion scintigraphy with technetium produced images after pharmacological stress with dipyridamole and at rest, which showed no signs of ischemia or myocardial fibrosis and showed normal left ventricular function (left ventricular ejection fraction = 65%). Permanent pacemaker implantation was chosen due to prolonged asystole requiring CPR during the stress stage of the exercise test. The patient progressed well and was referred to outpatient follow-up.

Discussion

The prevalence of anomalous origin of the circumflex artery is around 0.6% and is associated with increased incidence of myocardial ischemia and sudden death, especially among athletes. The most common complications related to exercise testing include serious tachyarrhythmia (4.78/10,000 tests), infarction (3.58/10,000 tests) and deaths (0.5/10,000 tests). Asystole during exercise in young people without any heart...
disease is described as a rare event in the literature and it is believed that one of the main mechanisms responsible is a vasovagal component with increased vagal stimulus.\textsuperscript{5} The mechanisms responsible for syncope and asystole in patients with anomalous origin of the circumflex artery are unknown, but possibly the same ones present in patients without a structural disease: bradycardia occurs by a gradual reduction in the end-diastolic and end-systolic volumes, with maximum increase in fractional shortening before syncope. Increased sympathetic tone in the scenario of a relatively empty heart leads to vigorous myocardial contraction with activation of mechanoreceptors on the ventricular wall and consequent response of the afferent vagus nerve. This vagal stimulus can trigger a sudden withdrawal of sympathetic efferent activity (negative circular feedback) and increase the parasympathetic efferent tone with vasodilatation and subsequent hypotension.

Hypotension usually precedes bradycardia, hence the inactivation of the sympathetic efferent and consequent vasodilation may be the primary mechanism of syncope. Bradycardia and asystole as markers of increased parasympathetic tone would play only a secondary role.
in most patients with vasodepressor syncope and may even prevent the pacemaker from avoiding hypotension and syncope in most patients; however, cardioinhibition with consequent bradycardia or asystole may be the primary mechanism in some patients.5

**Author contributions**

Conception and design of the research: Luciano LSC, Mallmann FB. Acquisition of data: Luciano LSC, Mallmann FB, Moreira DM, Frederico GN, Fracasso M. Writing of the manuscript: Luciano LSC, Moreira DM. Critical revision of the manuscript for intellectual content: Luciano LSC, Mallmann FB, Moreira DM, Frederico GN, Fracasso M.

**Potential Conflicts of Interest**

This study has no relevant conflicts of interest.

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**References**


