Bezold-Jarisch reflex in a patient undergoing endoscopic sympathectomy for management of refractory angina pectoris: a case report

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Received 15 January 2015; accepted 4 March 2015
Available online 16 September 2016

Abstract

Background and objectives: Ischemic cardiomyopathy is characterized by imbalance between supply and demand of myocardial oxygen. Endoscopic transthoracic sympathectomy is a therapeutic option indicated in refractory cases. However, the patient’s position on the operating table may favor ischemic coronary events triggering the Bezold-Jarisch reflex.

Case report: A female patient, 47 years old, with refractory ischemic cardiomyopathy, admitted to the operating room for endoscopic transthoracic sympathectomy, developed the Bezold-Jarisch reflex with severe bradycardia and hypotension after placement in semi-sitting position to the procedure.

Conclusion: Bradyarrhythmia, hypotension, and asystole are complications potentially associated with patient placement in a semi-sitting position, particularly in cases with previous ischemic heart disease.

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PALAVRAS-CHAVE
Reflexo de Bezold-Jarisch; Simpatectomia transtorácica endoscópica;
Ischemic cardiomyopathy;
Endoscopic transthoracic sympathectomy;
Bezold-Jarisch reflex;
Ischemic cardiomyopathy;
Endoscopic transthoracic sympathectomy;
Bezold-Jarisch reflex;
Ischemic cardiomyopathy;
Endoscopic transthoracic sympathectomy;
Cardiomiopatia isquêmica

Introduction

Ischemic cardiomyopathy (IC) is a disorder resulting from the imbalance between supply and demand of myocardial oxygen, whose most common cause is atherosclerosis of the epicardial coronary arteries. It is the leading cause of death worldwide and heart failure in Brazil. Endoscopic transthoracic sympathectomy (ETS) has emerged as a safe and effective therapy to minimize angina pectoris, reduce myocardial oxygen consumption, and improve the quality of life of these patients, especially in refractory cases.

In this procedure, the patient is placed in a semi-sitting position, which may precipitate in some situations ischemic coronary events and predispose the occurrence of Bezold-Jarisch reflex (BJR). Because it is a reflex, its onset is immediate and may trigger severe bradycardia, with hypotension and asystole.

Case report

Female patient, 47 years old, with symptomatic and progressive ischemic cardiomyopathy (IC), refractory to coronary revascularization procedures, contraindicated for heart transplantation, admitted to the operating room for endoscopic transthoracic sympathectomy (ETS).

In the previous two years, the patient developed acute myocardial infarction with triple-vessel disease and underwent two coronary artery bypass surgeries without success. Over the past eight months, she remained confined to a hospital bed for treatment of angina pectoris and intense and persistent dyspnea, aggravated by the slightest effort. She had depressed mood and aggressive behavior attributed to prolonged hospitalization. Physical examination revealed hypophonic heart sounds, breath sounds decreased in lung bases, and presence of peripheral edema, without other significant changes.

Among the preoperative laboratory tests, we highlight: hemoglobin = 11.5 g.dL⁻¹; hematocrit = 36%; INR = 1.46; creatine phosphokinase (CPK) = 311 IU.L⁻¹, CK-MB isoenzyme = 42 U.L⁻¹, and troponin-1 = 0.006 ng.mL⁻¹. Chest X-ray (Fig. 1) and electrocardiogram (Fig. 2) were performed. Echocardiogram showed ischemic cardiomyopathy with severe systolic dysfunction (left ventricular ejection fraction = 34%). The patient was classified as class II (NYHA Functional Classification), group III (Goldman Risk Index), and class IV (ASA physical status).

In the operating room, monitoring was performed with ECG, pulse oximetry, invasive blood pressure, capnography; central venous access was achieved through the internal jugular vein. As premedication, we used intravenous midazolam 3 mg. Anesthetic induction was performed with target controlled infusion of remifentanil started at 6 ng.mL⁻¹, rocuronium 0.6 mg.kg⁻¹, and etomidate 0.3 mg.kg⁻¹, plus lidocaine 1 mg.kg⁻¹ and dobutamine 5 mcg.kg⁻¹.min⁻¹; uneventfully. Single-lung ventilation was achieved using a double-lumen endobronchial tube and maintenance of anesthesia performed with sevoflurane and remifentanil.

The patient would be placed in a semi-sitting position for the procedure. Immediately after the head elevation, with a gap in the vertical plane of 30 cm between the base of the
brain (higher) and heart, she developed severe bradycardia and hypotension, which spontaneously stopped immediately after returning the patient to the neutral position in relation to the vertical plane.

The fluid situation, by pulse pressure variation, and the doses of current drugs have been checked and adjusted; norepinephrine was added starting at 0.2 mcg.kg⁻¹.min⁻¹. Then, another attempt to place the patient in a semi-sitting position was made, but without success due to the development of the cardiovascular events reported. On that occasion, the monitoring pattern was recorded before and after positioning (Figs. 3 and 4).

The procedure continued with the patient in the supine position and chest lateralization to facilitate the management of endoscopic instruments, but without any gap in the vertical plane. The left sympathetic chain was dissected, and complete blocking of the nerve impulses at the level of T1 and T2 was achieved using clips; there was no intervention in the right left sympathetic chain to minimize possible hypotension. The patient was transferred to the intensive care unit (ICU) without vasoactive drugs and discharged five days after the procedure, reporting significant improvement of symptoms 30 days after surgery.

**Discussion**

Ischemic heart disease (IHD) is a predominantly atherosclerotic disease due to the imbalance between oxygen delivery (DO₂) and consumption (VO₂) by the myocardium. Ischemic heart disease is the leading cause of death worldwide, accounting for 12.8% of deaths,¹ and the most common cause of heart failure in Brazil.²

Endoscopic transthoracic sympathectomy (ETS) may be a minimally invasive, effective, and safe therapy to improve the quality of life of these patients, especially in cases of coronary artery bypass grafting (CABG) failure or percutaneous coronary intervention (PCI). Studies have shown decreased angina pectoris³ and myocardial oxygen consumption by decreasing the baseline heart rate, plasma

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**Figure 2** Electrocardiogram 12-lead showing inactive area in the inferior wall, isolated ventricular extrasystole, and diffuse changes of ventricular repolarization.

**Figure 3** Monitoring pattern before the positioning.
Bezold-Jarisch reflex: case report

levels of norepinephrine, and occurrence of ventricular extrasystoles after the procedure.

In ETS, the patient should be placed supine, in a semi-sitting position, with the chest elevated at about 45°, and two small cushions under the shoulders and the back. These maneuvers intend to deviate armpits from the operating table, facilitate the manipulation of the endoscopic instruments, push the shoulders forward, and prevent stretching of the brachial plexus.

In this position, the accumulation of blood in capacitance vessels leads to decreased effective circulating volume and reduced atrial filling pressure. Consequently, stroke volume and cardiac output decrease, thus compromising coronary perfusion and oxygen delivery to the myocardium (DO₂). Moreover, there may be a reflex increase in sympathetic tone, with increased heart rate and oxygen consumption by the heart (VO₂), which predisposes to ischemic coronary events.

This decrease in venous return associated with sympathetic hyperactivity may be the mechanism responsible for the Bezold-Jarisch reflex (reflex sympathetic hyperactivity) onset, which starts by subendocardial sensory receptors in the inferoposterior region of the heart during ischemia and culminates in the vagal efferent action, determining reduction of oxygen consumption by reducing heart rate.

Some studies have reported association between the BJR and sitting position for shoulder arthroscopy. In our case, positioning the patient in a semi-sitting position for ETS triggered, twice, severe bradycardia and hypotension of sudden onset and spontaneous regression after returning to horizontal position. This demonstrates that the registered cardiocirculatory collapse was intimately related to the positioning.

The acute imbalance between myocardial oxygen delivery and consumption due to venous return reduction and reflex sympathetic hyperactivity may have exacerbated the prior ischemic impairment of the lower cardiac wall seen on preoperative electrocardiogram and, thus, triggered a BJR reflex through the stimulation of subendocardial sensory receptors sensitive to ischemia, located in the heart inferoposterior region.

Conclusion

Bradyarrhythmia, severe hypotension, and asystole that are established with the Bezold-Jarisch reflex onset are complications potentially associated with placing the patient in a semi-sitting position for surgery, particularly in cases with previous ischemic heart impairment.

Conflicts of interest

The authors declare no conflicts of interest.

References