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ORIGINAL ARTICLE

CASE REPORT OF ACUTE PULMONARY EDEMA DUE TO SYSTOLIC ANTERIOR MOVEMENT AFTER LIPOSUCTION

MATEUS MOREIRA DE MELO SILVA[1], DANIEL FERREIRA GUNDIM[1], FABRÍCIO RODRIGUES DE SOUZA[1], GIULLIANO GARDENGHI[1,2,3], GUSTAVO SIQUEIRA ELMIRO[1,3]

ABSTRACT

The systolic anterior motion is the dislocation of one or more of mitral valve leaflets anteriorly causing obstruction during the left ventricular outflow. Diagnose can demand transesophagic echocardiography or magnetic resonance and its treatment is majorly clinical. The objective of this text is to report a challenging case in plastic surgery postoperative, the clinical presentation was an acute pulmonary edema and recieved the diagnosis of Systolic Anterior Motions just after. It led to a necessity of a particular menagement, contrasting the menagement based on the nonspecific primary diagnosis.

KEYWORDS: PULMONARY EDEMA; LIPECTOMY; SYSTOLE; MITRAL VALVE; VENTRICULAR OUTFLOW OBSTRUCTION

INTRODUCTION

Systolic anterior motion (SAM) is a peri or intraoperative complication where there is movement of one or more leaflets of the mitral valve in the anterior direction, causing obstruction during the exit of systolic content from the left ventricle (LV) [1]. In the absence of previous heart disease, it can be caused by decreased preload, vasodilation caused by general anesthesia or neuraxial block [2]. SAM is a heart condition that has a varied manifestation from asymptomatic to severe cases [3]. It is clinically manifested by hypoxemia, hypotension and acute pulmonary edema (APE), being little remembered in the first differential diagnoses in the face of these clinical alterations, mainly in non-cardiac surgeries [1,3]. Its diagnosis can be made through transesophageal echocardiography (TEE) or magnetic resonance imaging [3] and the treatment varies according to the severity of the manifestation, with cardiac surgery being the last option, but the ideal approach remains without consensus in the literature [4]. The causal relationship between anesthesia and the condition in question has not been epidemiologically proven. The aim of this work is to present a clinical case of SAM managed in the postoperative period of plastic surgery on the day following the anesthetic procedure.

CASE REPORT

Patient 49 years old, denies allergies, reports controlled systemic arterial hypertension, associated with diabetes mellitus also compensated, with a history of right eye loss. On physical examination: blood pressure 128x82 mmHg and cardiac auscultation with regular rhythm in two stages, normophonetic sounds, systolic murmur of light intensity in the mitral focus. Transthoracic echocardiography (TTE) showing an ejection fraction of 67% by Teichholz, with mild mitral and tricuspid regurgitation, with more pronounced concentric LV hypertrophy in the septal region with 16 mm.

Patient admitted to the hospital for a liposculpture surgical procedure, fasting for eight hours and asymptomatic. In the operating room, monitoring with electrocardiography (ECG), plethysmography and non-invasive blood pressure was performed, followed by 20G jelco venoclysis. Patient sedated with midazolam 5 mg and sufentanil 5 micrograms, sitting, performed asepsis of the lumbar region, followed by sterile lumbar puncture with a 27 G Quincke needle at L3-L4 with spinal infusion of heavy bupivacaine 20 mg, after the sensory block level test analgesic on T4.

Anesthetic induction with sufentanil 10 micrograms, propofol 120 mg and cisatracurium 10 mg. Orotracheal intubation with a 7.5-inch cuffed cannula was performed, without adverse events. Anesthetic maintenance with 2% inspired sevoflurane and dexmedetomitin in a continuous infusion pump (CIP) 0.3 microgram/kg/minute. Surgical procedure performed without adverse events. Extubation after decurarization with 2 mg of neostigmine without physiological repercussions. Patient referred to the ward after two hours of post anesthetic recovery, without complaints.

1. Clínica de Anestesia (CLIANEST), Goiânia/GO, Brasil.
2. Hospital ENCORE, Aparecida de Goiânia/GO, Brasil.
3. Hospital de Urgências de Goiás, Goiânia/GO, Brasil.

ADDRESS
GIULLIANO GARDENGHI
Clianest, R. T-32, 279 - St. Bueno, Goiânia - GO, 74210-210
Telefone: +55 (62) 3604-1100
E-mail: ggardenghi@encore.com.br

After discharge: she ate and, while still in the ward, she developed a complaint of dyspnea followed by chest pain. The hospital's emergency team was called. According to the emergency room physician, the patient was tachydyspneic with oximetry at 70%, complaining of excruciating retrosternal pain, radiating to the neck and back, ECG with ST-infralevelling (3-way ECG) and hypotension, and O2 was offered in a mask with reservoir at 15 l/min and norepinephrine in CIP.

Patient evaluated by an anesthesiologist who found acute respiratory failure and proceeded with emergency orotracheal intubation in rapid sequence, at laryngoscopy patient with abundant pink foamy secretion in the trachea. Bedside TTE showing moderate mitral regurgitation, concentric LV hypertrophy and pulmonary hypertension, with preserved LV function. Next, patient was transported for chest angiotomography, with signs of cor pulmonale, without signs suggestive of pulmonary thromboembolism as shown in Figure 1 and without signs suggestive of aortic dissection.

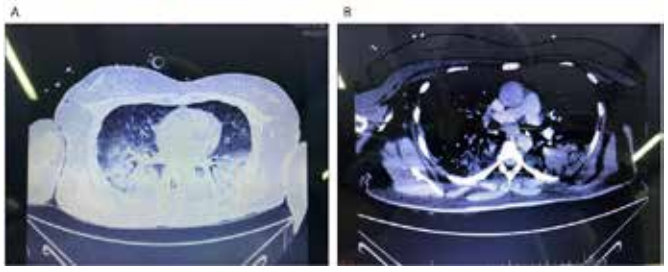

Figure 1. Chest angiotomography image. A: Ground-glass opacities and thickening of the peribronchovascular interstitium, compatible with pulmonary edema; B: Enlarged pulmonary arteries, attenuation of peripheral pulmonary vasculature and enlargement of the right ventricle, suggestive of pulmonary hypertension, without signs of arterial occlusion of grids or medium vessels.

She was also transported for coronary catheterization, which showed a sub-occlusive lesion of the marginal branch of the right coronary artery, already with collateral circulation, without signs of acute lesions in activity. Patient maintained clinical worsening during coronary catheterization, then arterial catheterization was performed to assess invasive blood pressure and started dobutamine 2.5 mcg/kg/min, without improvement. At the end of the procedure, the patient was referred to the Intensive Care Unit (ICU) without a diagnosis of the cause of clinical decompensation. The patient in the ICU was studied again, however, this time by TEE, which found moderate mitral regurgitation, LV concentric hypertrophy and pulmonary hypertension, with preserved LV function and tightening of the anterior leaflet of the mitral valve, producing marked obstruction of the outflow tract. of the LV (figures 02 and 03).

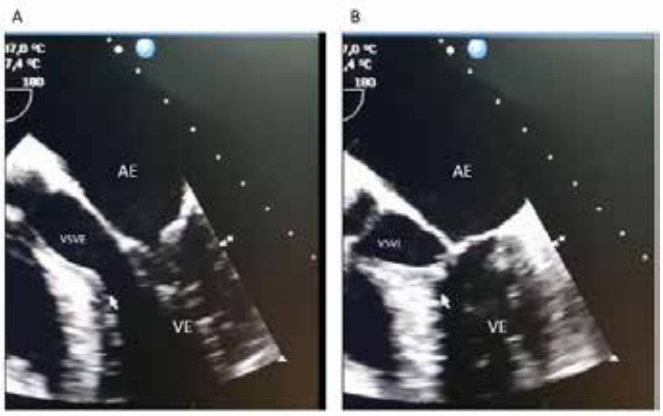

Figure 2. TEE image. A: Left ventricular outflow tract (LVOT) opened during ventricular diastole. B: LVOT obstructed by the anterior leaflet of the mitral valve during ventricular systole. C: Post-intervention result. Image of residual thrombus is observed, but without flow impairment.

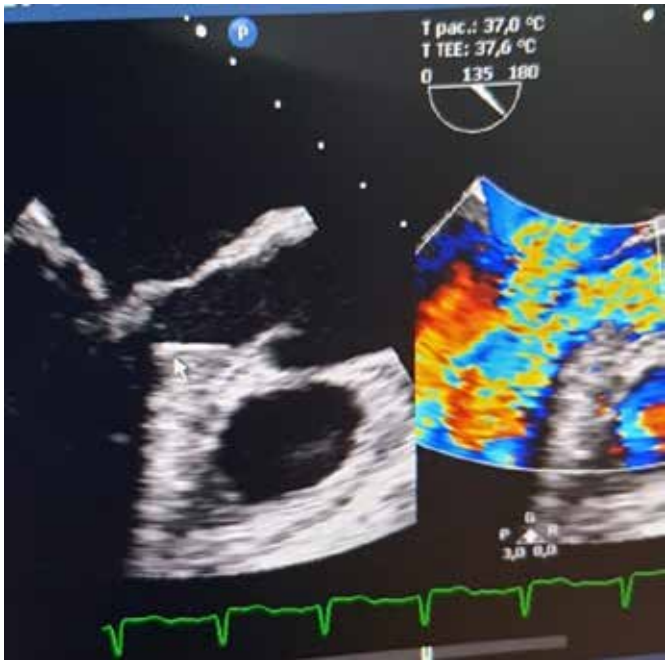

Figure 3. Transesophageal echocardiogram image demonstrating turbulent and disorganized flow due to LVOT narrowing.

The treatment was performed by discontinuing dobutamine, starting carvedilol and volume expansion with crystalloids aiming at a positive fluid balance. Due to the diagnosis of acute pulmonary edema, special follow-up and attention was needed to prevent them from restarting beta-adrenergic drugs and taking measures to negatively affect fluid balance.

DISCUSSION

The diagnosis of LV outflow tract obstruction is rare, but should be considered in patients who present with symptoms and ECG changes consistent with hypoxemia,

acute coronary syndrome, systolic murmur, PAE, and hypotension in the presence of a small elevation of serum creatine phosphate kinase, which were subjected to hypovolemic states, inotropic drugs, cardiac surgery and anesthetic states. [5]

The pathophysiology of SAM still remains uncertain, but the most accepted idea would be a Venturi effect (air flowing at a higher speed as it circulates through a thinner passage, creating negative pressure in the process, which causes a partial vacuum leading to fluid impulsion) through the narrowing of the LV outflow caused by the bulging of a hyperdynamic and hypertrophic septum, in a hypovolemic state [5]. Knowing this, the initial idea of treatment could involve volume increase, rate controls and cardiac contractility. Considering the perioperative period, neuraxial block, general anesthesia, hypovolemia, use of inotropes and bleeding can increase the risk or worsen this pathology [5] and are the daily routine of the anesthesiologist.

For diagnosis, TTE should be considered, however, if the images are suboptimal for diagnosis, TEE should be used [6]. In the case reported here, the use of TEE was necessary to properly perform the diagnosis of SAM.

Reasoning from the point of pre-anesthetic evaluation, the findings found by the physical and complementary exams were not considered sufficient for cancellation or postponement of the procedure, being released by the assistant team both before and on the day of the procedure, after reassessing the medical records.

Taking into account the anesthetic procedure itself, both general anesthesia and neuraxial block via spinal anesthesia are possible causes of SAM by causing relative or absolute hypovolemia; vasodilator effect, which in turn can cause an LV with low volume, reducing its own diameter and increasing the speed of blood flow in the region. However, other causes cannot be ruled out, nor can it be affirmed that anesthesia was the real cause of all the problems, because the patient already had some degree of mitral regurgitation and concentric LV hypertrophy.

Once the diagnosis is made, the clinical management of SAM, according to Ibrahim et al, consists of two steps, namely: step 01, which consists of ceasing inotropes plus volume expansion, and step 02, where beta-blockers should be started. In the clinical case, the patient presented progressive clinical worsening until the measures foreseen by the first step were taken. Once the diagnosis was made and treatment started in step 01, another challenge was to convince the ICU on-call team to maintain the previously discussed approach, since this APE was a consequence of pulmonary hypertension caused by a not so prevalent pathology, where inotropism added to the use of diuretics and consequent hypovolemia is exactly the opposite of ideal for SAM cases.

CONCLUSION

The case demonstrates that, faced with a challenging clinical presentation, one must have a range of differential diagnosis options. Multidisciplinary assistance and accessibility to complementary exams add up to a positive outcome, being of paramount importance for the case. Both the emergency, intensive, cardiological, anesthesiological and echocardiographic teams formed a vital group for the diagnosis and treatment of this patient and despite the potential diagnosis with TTE, the availability of TEE was essential. Without it, the etiological diagnosis would remain uncertain and without the multidisciplinary work, the clinical measures necessary to modify the outcome could be delayed or even not taken, resulting in a worse prognosis for the patient.

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