Case Report

Inferior Myocardial Infarction and Shock Associated with Left Ventricular Outflow Tract Obstruction

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We report the case of a patient admitted to the emergency room of our hospital with acute myocardial infarction in the inferior wall, who underwent primary angioplasty, complicated by shock that did not respond to the administration of catecholamines and intra-aortic counterpulsation balloon. The use of transesophageal echocardiography was crucial in showing systolic anterior motion of the mitral valve obstructing the left ventricular outflow tract.

Dynamic obstruction of the left ventricular outflow tract with systolic anterior motion of the mitral valve has been reported in association with several conditions, such as hypertensive concentric hypertrophy[1], excessive sympathetic stimulation[2,3], pericardial tamponade[4], dobutamine stress tests[5], postoperative mitral valvuloplasty[6], and aortic valve replacement[7], in addition to obstructive hypertrophic cardiomyopathy. Some cases of dynamic left ventricular outflow tract obstruction complicating anterior myocardial infarction have been reported[8-12]; however, no case of inferior myocardial infarction has been reported in association with that entity. The hemodynamic findings of myocardial infarction aggravated by left ventricular outflow tract obstruction have some implications in the treatment; these implications are not common in the treatment of the infarction without that complication. In the situation of hemodynamic deterioration, the use of intra-aortic counterpulsation balloon may have a deleterious effect on the patient diagnosed with that obstruction and may hinder improvement in clinical condition[13,14]. The use of vasodilators and some catecholamines may also worsen the state of shock [14]. We report the case of a patient with inferior myocardial infarction associated with left ventricular outflow tract obstruction aggravated by the use of intra-aortic counterpulsation balloon.

Case report

The patient was an 87-year-old male admitted to the hospital after an episode of syncope with no complaint of chest pain. On physical examination, his systemic blood pressure was 70/40 mmHg and his heart rate was 108 bpm. Cardiac auscultation revealed a regurgitant systolic murmur (III/VI intensity), better audible in the apex. The rest of the physical examination was normal. The electrocardiogram showed a 4mm elevation in the ST segment in the inferior leads. After the implementation of intra-aortic counterpulsation balloon, the patient was referred for immediate hemodynamic study. Coronary angiography showed a dominant right coronary artery, occlusion in the middle third of the posterior descending artery (fig. 1-A), and moderate obstruction in the middle third of the anterior descending artery. Left ventriculography showed akinesia of the inferior and septal wall and significant mitral regurgitation (fig. 2-A and B). Percutaneous transluminal coronary angioplasty was successfully performed in the middle third of the posterior descending artery (fig. 1-B), and abciximab was administered. The patient was referred to the Coronary Unit, and, due to the presence of refractory shock, dobutamine and noradrenaline were initiated, and mechanical ventilation was implemented. The values of creatine phosphokinase (CPK) and of its muscle-brain (MB) fraction reached peaks of 165 and 30 U/L, respectively. Due to the occurrence of mitral regurgitation and shock unresponsive to the treatment instituted, echocardiography was required to exclude possible mechanical complications of infarction. In addition, transesophageal echocardiography was performed after the demonstration of a transthoracic window of poor quality, which showed akinesia of the inferior septum and inferior wall, with no other abnormality of contractility. The estimated left ventricular ejection fraction was 45%, and no compensatory hyperkinesia of other segments was evident. Systolic anterior motion of the mitral valve (fig. 3) was observed in association with moderate regurgitation. The continuous Doppler evidenced a peak gradient in the left ventricular outflow tract of 186 mmHg (fig. 4-A).

The patient continued to use high doses of dobutamine and noradrenaline, and the frequency of the intra-aortic counterpulsation balloon was 1:1. As the high gradient of the left ventricular outflow tract was attributed to the presence of systolic anterior motion, the infusion of dobutamine was reduced to 5 mcg/Kg/min, and the administration of noradrenaline was interrupted. The frequency of intra-aortic counterpulsation balloon was reduced, and, after it was turned off, the left ventricular outflow tract gradients were measured again. At that time, the 2-dimensional images of the systolic anterior motion of the mitral valve observed on the longitudinal and frontal views of the transesophageal echocardiogram remained the same; however, the peak gradient measured in the left ventricular outflow tract decreased to 132 mmHg (fig. 4-B). Despite the interruption of those therapeutic measures, the mean systemic blood pressure remained the same (80 mmHg), as did the pulmonary capillary pressure measured with the Swan-Ganz catheter. Other hemodynamic instabilities associated with deterioration in the function

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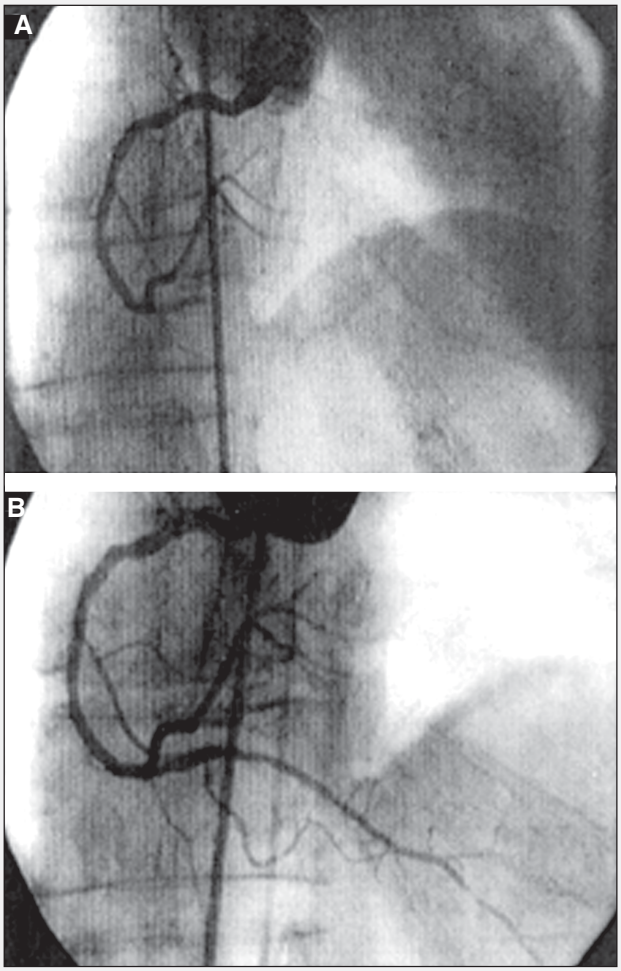

Fig. 1 - Coronary angiography of the right coronary artery showing: A) occlusion of the middle portion of the posterior descending branch; B) complete opacity after percutaneous transluminal coronary angioplasty.

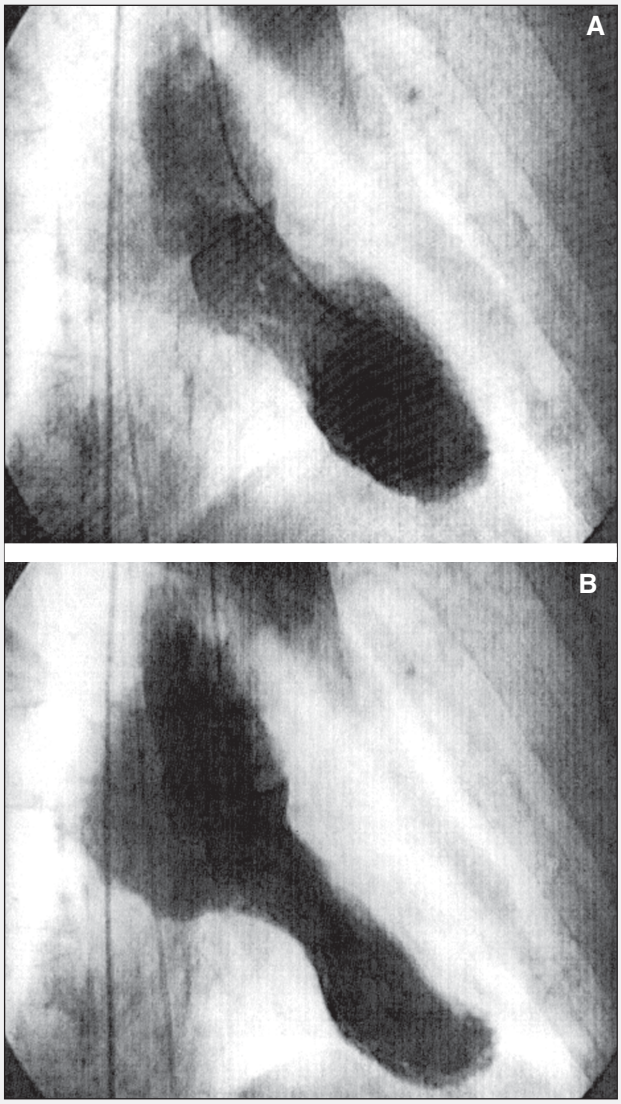

Fig. 2 - Left ventriculography showing: A) left ventricle in diastole; and B) left ventricle in systole, akinesia of the diaphragmatic wall, and significant mitral regurgitation.

of the cardiac pump occurred, requiring a new elevation in the dose of dobutamine and the restart of intra-aortic counterpulsation balloon.

The patient also had the following complications: pulmonary infection, which required antibiotic therapy and 29 days of mechanical ventilation, renal failure, and vigil coma. Another transesophageal echocardiography was performed 19 days after the first one, but neither systolic anterior motion of the mitral valve nor a left ventricular outflow tract gradient was detected (fig. 5). Left ventricular function was normal, the estimated ejection fraction was 60%, and no alteration in the contractility of the walls was observed. The patient was discharged after 47 days of hospitalization.

Discussion

As far as we know, this is the first case report on the association of inferior infarction and systolic anterior motion of the mitral valve and left ventricular outflow tract obstruction. The cases reported in association with anterior or apical myocardial infarction were attributed to the hyperdynamic response of the left ventricular basal portions, leading to acceleration of blood flow through the left ventricular outflow tract, creating a Venturi effect and anterior suction of the mitral valve, with its obstruction. For this phenomenon to occur, the basal segments of the left ventricular walls should have normal myocardial perfusion[14]. In the case reported here, the infarction was not located in the antero-apical region, and no hypercontractility of the left ventricular basal segments was observed.

Systolic anterior motion is a condition of the left ventricle that results from an altered balance of forces that act on the mitral valve apparatus. The mechanism of its cause is still controversial. There are 2 theories to explain systolic anterior motion of the mitral valve: the Venturi hypothesis and the theory of geometrical alteration of the papillary muscle-mitral valve. The latter states that systolic anterior motion of the mitral valve results from the abnormal distribution of forces in the chordae tendineae, which causes the abnormal anterior motion of the mitral valve apparatus[15]. In patients with obstructive hypertrophic cardiomyopathy, the anterior dislocation of the papillary muscles, the anterior motion of the mitral leaflets and of its point of coaptation, with strengthening of its residual portion and stretching of the leaflets were evidenced. The anterior redirecting of the point of tension of the papillary muscles, causing loosening of the leaflets (related to the decreased posterior restriction), and increase in the length of the residual leaflet up to the point of coaptation may

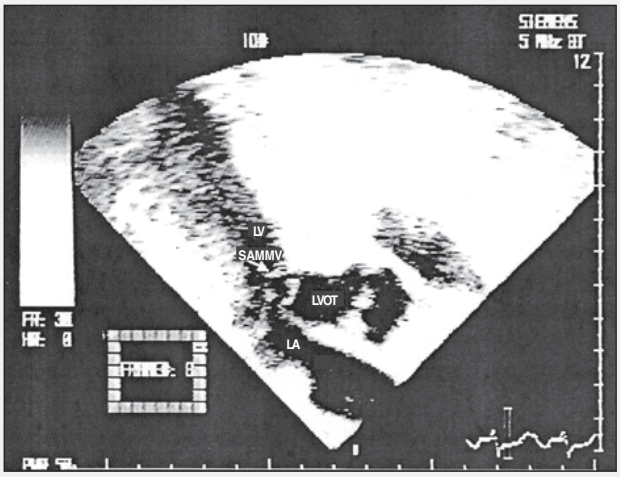

Fig. 3 - Transesophageal echocardiography: longitudinal view of the left cardiac chambers and systolic anterior motion of the mitral valve causing obstruction of the left ventricular outflow tract. LA - left atrium; LV - left ventricle; LVOT - left ventricular outflow tract; SAMMV - systolic anterior motion of the mitral valve.

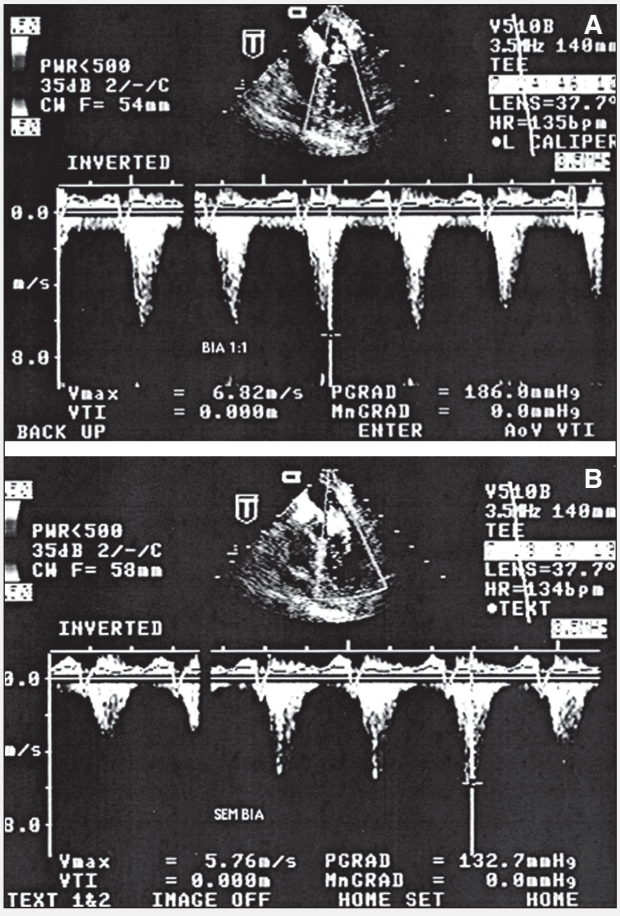

Fig. 4 - A) Transesophageal echocardiography: frontal view showing blood flow in the left ventricular outflow tract; peak gradient (186 mmHg) obtained with continuous Doppler when the frequency of the intra-aortic balloon of counterpulsation was 1:1 and the patient was still using high doses of dobutamine; B) decrease in the peak gradient (132 mmHg) after turning off the balloon and reducing the dose of dobutamine.

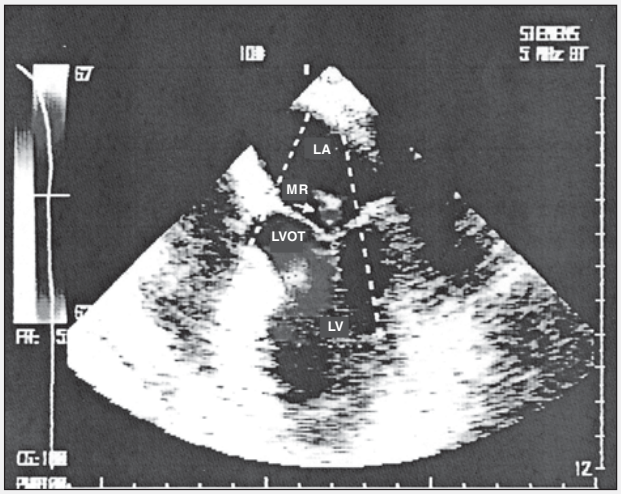

Fig. 5 - Transesophageal echocardiography: frontal view showing insignificant mitral regurgitation (MR) and absence of turbulent flow in the left ventricular outflow tract. LA: left atrium; LV: left ventricle; LVOT: left ventricular outflow tract.

lead to interposition of the leaflets in the direction of the flow of the left ventricular outflow tract, causing their anterior and superior dislocation. This combination of forces, determining the relative relaxation and redundancy of the middle portion of the affected leaflets, makes them prone to being anteriorly pulled by the forces of the Venturi phenomenon, or anteriorly pushed by the forces of flow tension[16].

The rearrangement in the left ventricular geometry of our patient may have altered the relation between space and forces in the papillary muscles, the mitral valve leaflets, its point of coaptation, and the left ventricular outflow tract. Together, these alterations could cause the precipitation and appearance of systolic anterior motion of the mitral valve. The use of intra-aortic counterpulsation balloon, causing a reduction in afterload, the use of catecholamines, and a possible hypovolemia, diminishing the ventricular cavity, may have aggravated the state of the left ventricular outflow tract obstruction and the patient's hemodynamic condition. The temporary suspension of these therapeutic measures caused a decrease in the left ventricular outflow tract gradient. The patient's recovery with resolution of the regional abnormalities of contractility and weaning from the intra-aortic counterpulsation balloon and from the vasoactive amines coincided with the complete disappearance of the systolic anterior motion of the mitral valve and the left ventricular outflow tract obstruction.

As the first report on the association of non-anterior infarction and systolic anterior motion of the mitral valve and left ventricular outflow tract obstruction, this case emphasizes the importance of widening our diagnostic range when treating patients with infarction, refractory shock, and significant mitral regurgitation. Transesophageal echocardiography plays an important diagnostic role in this clinical condition.

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