Rev Port Cardiol. 2011;30(11):837–843

Revista Portuguesa de
Cardiologia
Portuguese Journal of Cardiology
www.revportcardiol.org

CASE REPORT

Severe left ventricular outflow tract obstruction as a complication of mitral valve repair: Case report☆

Bruno Rodrigues*, Luís Ferreira Santos, Emanuel Correia, Rita Faria, Davide Moreira, Pedro Gama, Costa Cabral, João Pipa, Oliveira Santos

Serviço de Cardiologia, Hospital São Teotónio, Viseu, Portugal

Received 17 February 2010; accepted 7 April 2011
Available online 18 November 2011

KEYWORDS
Systolic anterior motion;
Mitral valve repair;
Pathophysiology

Abstract Systolic anterior motion (SAM) is a postoperative complication of mitral valve repair, with an incidence of 5–10%. Early recognition of the signs and symptoms of SAM is essential for the management of these patients. This article focuses on the pathophysiology and dynamics of SAM and the treatment strategies described in the literature. The authors present a case study and echocardiographic images illustrating the clinical relevance of the mechanism involved, in order to clarify whether surgical reintervention is necessary.

PALAVRAS-CHAVE
Movimento anterior sistólico;
Valvuloplastia mitral;
Fisiopatologia

Obstrução severa do tracto de saída do ventrículo esquerdo como complicação de valvuloplastia mitral: a propósito de um caso clínico

Resumo O movimento anterior sistólico (SAM) é uma complicação pós cirúrgica da valvuloplastia mitral, sendo a sua incidência de 5-10%. O reconhecimento precoce dos sinais e sintomas de SAM é imperativo no delinear de estratégia terapêutica nesses pacientes. Este artigo foca os principais mecanismos fisiopatológicos do SAM dinâmico e modalidades de tratamento descritas na literatura. Os autores descrevem um caso clínico e as imagens ecocardiográficas captadas ilustrando a relevância clínica do mecanismo envolvido, na tentativa de esclarecer uma questão suscitada: reintervenção cirúrgica necessária?

☆ Please cite this article as: Rodrigues, B. Obstrução severa do tracto de saída do ventrículo esquerdo como complicação de valvuloplastia mitral: a propósito de um caso clínico. Rev Port Cardiol. 2011;30(11):837–843.
* Corresponding author.
E-mail address: onurb80@sapo.pt (B. Rodrigues).

Introduction

Systolic anterior motion (SAM) is due to partial obstruction of the left ventricular outflow tract (LVOT) by the mitral valve (MV) anterior leaflet.[1] It has been reported in patients with hypertrophic cardiomyopathy, following myocardial infarction, and as a postoperative complication of MV repair.[2,3]

Case report

We describe the case of a 71-year-old female patient, Caucasian, admitted to our department for decompensated heart failure (NYHA class III/IV). Her personal history included hypertension, dyslipidemia, chronic atrial fibrillation, severe mitral regurgitation (posterior leaflet prolapse) and moderate to severe tricuspid regurgitation, with pulmonary hypertension. She had undergone cardiothoracic surgery 15 days before, with mitral valve repair (quadrangular resection of the posterior leaflet with implantation of a Carpentier ring) and tricuspid annuloplasty. Postoperative transesophageal echocardiography (TEE) showed good mitral valve competence and no regurgitation, and no other significant alterations.

The patient was medicated with furosemide (40+20 mg), enalapril 5 mg once a day, carvedilol 6.25 mg twice a day, amiodarone 200 mg once a day, spironolactone 25 mg once a day, potassium chloride (Retard) once a day, warfarin (for INR 2–3), omeprazole 20 mg once a day and sertraline 50 mg once a day.

On admission to the emergency department, the patient complained of precordial discomfort and worsening dyspnea on minimal exertion, as well as paroxysmal nocturnal orthopnea and dyspnea. Physical examination showed blood pressure of 86/64 mmHg and mean heart rate (HR) of 150 bpm; cardiac auscultation revealed arrhythmia and a grade III/VI systolic murmur over the aorta. Pulmonary auscultation revealed absence of breath sounds in the left lung base. There was no lower limb edema.

Laboratory tests showed normocytic and normochromic anemia (Hb 10.9 g/dl) and worsening baseline renal function (urea 146 mg/dl; creatinine 1.9 mg/dl; creatinine clearance [by the MDRD formula] 27.69 ml/min). The ECG revealed atrial fibrillation with mean ventricular response of 150 bpm and poor R-wave progression in V1–V2. The chest X-ray showed cardiomegaly and moderate left pleural effusion.

The patient was admitted for decompensated heart failure. Transthoracic echocardiography (TTE) performed on the first day of hospitalization (with HR 120–150 bpm) (Figure 1) revealed aortic valve fibrosis with no restriction of opening, together with mild regurgitation.

The MV presented fibrocalcification, with increased echogenicity of the annulus; the anterior leaflet and subvalvular apparatus were obstructing the LVOT, resulting in an intraventricular gradient of 110 mmHg and moderate paroxysmal regurgitation (probably related to the intermittent nature of the LVOT obstruction). The left atrium was severely dilated (6.1 cm), and the left ventricle was hypertrophied (diastolic diameter 4.3 cm) but with good global systolic function. The right chambers were of normal size, with pulmonary artery pressure estimated at 40 mmHg.

For a more accurate assessment of MV function, TEE was performed (with HR 120–150 bpm) (Figures 2 and 3), which showed the MV with a Carpentier ring and leaflet degeneration and redundancy, good opening in diastole but with SAM leading to LVOT obstruction by the anterior leaflet, and severe regurgitation (vena contracta 8 mm). The aortic valve was tricuspid, with good opening and mild regurgitation. The left atrial appendage was free of thrombi.

Since the patient's clinical condition was extremely unstable during hospital stay, systolic blood pressure remaining below 90 mmHg and with clear signs of heart failure in NYHA class IV, the patient was transferred to a surgical center 14 days after admission to be evaluated for surgical reintervention.

Six weeks after her initial admission to our department, the patient was seen at the outpatient clinic; she was hemodynamically stable, in good general health and with no signs of heart failure. The report from the surgical center, where she had remained for three weeks, revealed that surgical reintervention had not been necessary. TTE at discharge showed significant improvement in echocardiographic parameters (mild mitral regurgitation and no LVOT obstruction by the mitral anterior leaflet). Repeat TTE a week after reassessment, with optimized HR, revealed good MV function (mild regurgitation), with no LVOT obstruction (Figures 4 and 5).

Pathophysiology of SAM

The literature indicates that SAM, which has been reported after mitral valve repair in various studies,[3] is caused by the velocity of the blood flow drawing the ventricular surface of the MV anterior leaflet into the LVOT. The position and any abnormalities of the two leaflets contribute to the phenomenon.

Firstly, the distance between the MV coaptation point and the septum is shortened due to elongation of the posterior or anterior leaflets during surgical repair, increasing the area of the anterior leaflet exposed to LVOT flow.[4]

Secondly, during surgical repair of the papillary muscles, the MV may be displaced anteriorly around the LVOT, thus directly exposing the anterior leaflet to the outflow stream.[5–7] Fluid overload in the pre- and postoperative period causes the septum to bulge leftwards and restrict the LVOT, while postoperative hypovolemia reduces left ventricular diastolic dimensions, thus decreasing LVOT diameter. All these pathophysiological conditions contribute to the development of SAM following mitral valve repair.[1]

However, there is some debate as to how and why the MV anterior leaflet is pushed towards the LVOT once the above-mentioned conditions are present.[8–12] One theory is that it is due to a Venturi effect, the result of a fall in pressure distally to an obstruction. Pressure can be restored if there is dilatation distally to the stenosis with an angle of