Constrictive Pericarditis Causing Mitral Stenosis: A Case Report

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ABSTRACT

External compression of the cardiac valves giving symptoms is a known pathological entity¹. We report a 35-year old male patient who presented with a history of palpitations and shortness of breath on exercise of four months' duration. Clinical, radiographic, electrocardiographic, and echocardiographic examination, as well as cardiac catheterization revealed mitral stenosis and constrictive pericarditis. After a near radical pericardiectomy, all intraoperatively measured haemodynamic data normalised and the subjective and objective clinical signs and symptoms of mitral stenosis disappeared, and mitral valve surgery was therefore unnecessary and avoidable.

THE CASE

A 35 year old male patient, who previously had been in good health, was admitted to King Fahad Heart Centre with a four months' history of palpitations and shortness of breath on mild to moderate exercise. There was no past history of rheumatic fever. The physical examination revealed a blood pressure of 130/80 mmHg, and a heart rate of 80 beats per minute, with a sinus rhythm. The cardiac examination revealed a loud first heart sound with a grade 3/6 mid-diastolic rumbling murmur at the apex area, but no opening snap was audible. The remainder of the examination was normal. Electrocardiogram revealed a normal sinus rhythm with a right axis deviation, left atrial enlargement and low voltage complex.

Laboratory examinations revealed an erythrocyte sedimentation rate (ESR) of 15mm, an antistreptolysin titer of 100 Todd units and negative C-reactive protein.

Plain chest X-ray (Pa and lateral views) revealed enlarged left atrium and right ventricle. calcification was noted around the right ventricle and the posterior atrioventricular grove. Both lungs were clear. M-mode echocardiography revealed moderate to severe mitral valve stenosis with normal leaflets, free edges and commissures without fibrosis or calcification. The mitral valve was 1.6cm² and the left atrium was slightly enlarged (46 mm). The tricuspid, pulmonary and aortic valves were normal. The left ventricular performance was good. There was an obvious obliteration of the pericardial space, however, there was an obvious increase in the left ventricular dimensions during inspiration, suggestive of consitrictive pericarditis. The two-dimensional echocardiography revealed mitral stenosis and obvious pericardial constriction with high density echoes. Cardiac catheterisation revealed a significant end-diastolic gradient across the mitral valve (17 mmHg).

A median sternotomy was performed. Significant pericardial fibrotic constriction was found. The parietal and visceral pericardium were greatly adherent, thickened and partially calcified, therefore, stand-by cardiopulmonary bypass was made available, but was not instituted. Further dissection was then performed on the beating heart. An obvious line of constriction and calcification of the pericardium over the posterior atrioventricular groove was apparent. Anterior and posterior dissection of the constricting pericardium was performed carefully, first on the left ventricle and the four pulmonary veins. The constricting pericardium on the posterior atrioventricular region was meticulously dissected and removed as much as possible, until the earlier noted constriction almost totally disappeared. The constricting pericardium on the right ventricle, superior and inferior venae cavae, right atrium, pulmonary artery

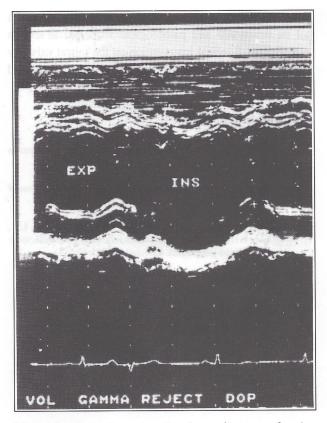


Figure 1: Two-dimensional echocardiogram showing thickened pericardium

and aorta was then carefully dissected and removed. After a near radical pericardiectomy, intracardiac pressures were measured intraoperatively and revealed complete normalisation. The preoperative end-diastolic gradient across the mitral valve of 17 mmHg was practically abolished. The post pericardiectomy left atrial pressure was 8 mmHg, while the left ventricular end-diastolic pressure (LVEDP) was 4 mmHg. The left atrial hypertension and the square root sign disappeared. Hemostasis was achieved, the anterior mediastinum drained, and the chest was closed.

Intraoperative auscultation using a disposable oesophageal stethoscope revealed normal heart sounds and absence of the diastolic rumbling murmur. These findings were confirmed by postoperative clinical examination. The patient was extubated on the first postoperative day without difficulty and had a smooth postoperative course. He was discharged from the hospital on the ninth postoperative day. He was then followed up for seven years. Serial echocardiographies were carried out and cardiac catheterisation four years after the surgery revealed normal mitral valve function and normal haemodynamic parameters, respectively.

DISCUSSION

Hippocrates and Galen in 460 BC and 130 AD respectively, described the pericardium and defined it as a protective covering of the heart.

The danger of cardiac compression following constrictive pericarditis has been reported by Morgagni¹. Chronic constrictive pericarditis can affect the orifice of the aortic valve as reported by Chevers², but to our knowledge, no such correlation with the mitral valve has been reported in the literature. Chevers observed that compression of the muscular tissue of the heart following constrictive pericarditis can affect the systolic and diastolic movement of the heart². Normalisation of hemodynamics and intracardiac pressures following adequate pericardiectomy for constrictive pericarditis has been reported in the literature³.

We report this case to illustrate that constrictive pericarditis can cause extracardiac compression of the mitral valve and therefore mimic various intracardiac valvular lesions including mitral stenosis.

In our patient, relief of mitral stenosis symptoms and signs was achieved primarily by pericardiectomy. Isolated organic mitral stenosis must, of course, be excluded. If constrictive pericarditis is confirmed to be the aetiological extra-cardiac mechanism of mitral stenosis, a near radical pericardiectomy must be carried out, and the success and efficacy of such a surgical therapy confirmed by measuring intracardiac pressures, pressure gradients and intra-operative use of esophageal stethoscope to detect any

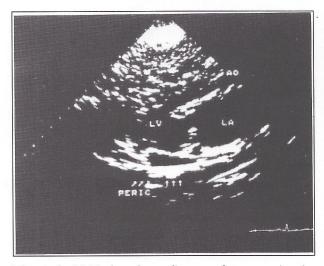


Figure 2: M-Mode echocardiogram demonstrating increase in LV dimension during inspiration, suggestive of constrictive pericarditis.

murmurs. The use of oesophageal echocardiography to evaluate the mitral valve orifice, competence and the cardiac function intraoperatively, is highly recommended, if available.

CONCLUSION

Mitral stenosis due to constrictive pericarditis is an uncommon pathological entity that is potentially curable by surgical excision of the constricting diseased, thickened and fibrotic pericardium alone. Mitral valve surgery may therefore not be needed for mitral stenosis of such aetiology. We believe that constriction of the pericardium along the posterior atrioventricular groove can affect the mitral

valve mechanism, causing extrinsic mitral stenosis with all its functional and pathological sequelae, as seen in our patient. Surgical intervention on the mitral valve by means of open heart surgery can therefore be spared and avoided.

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