CORONARY ARTERY EMBOLUS CAUSED BY CALCIFIED MITRAL VALVE DISEASE

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A 34 year old male patient whom ebaloon valvuloplasty was previously performed due to mitral stenosis was admitted to the hospital with complaints of dyspnea and paroxsysmal nocturnal dyspnea in the past one year period. Echocardiography demonstrated severe fibrocalcific mitral stenosis with multible vegetative nodules on the mitral valve. While the patient was being prepared for MVR, an acute inferior myocardial infarction developed. Coronary angiography was performed, and a intracoronary mass which caused 80-90% occlusion in the mid portion of right coronary artery was found. The patient was operated 2 months later and a mobil, calcified mass was found to occlude the coronary vessel 1.5 cm lenght. The mass was removed and arterioplasty was performed with a saphenous vein patch. Mitral valve was excised and mitral valve replacement was performed. The patient was discharged on the 14th day without any complaint.

Key words: Coronary artery embolus, CABG, coronary artery patchplasty.

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Spontaneous coronary artery embolism causing acute myocardial infarction (AMI) is a rare encountered clinical situation. Coronary artery embolism caused by calcific vegetations setteled on a stenotic mitral valve has not been reported before in the literature as for as we could have searched.

CASE REPORT

A thirty five year old male patient was admitted to a refferring hospital with a complaint of exertional dyspnea, and palpitation for the last one year. Orthopnea, hemoptysis, paroxismal nocturnal dyspnea arised for the previous 45 days. The patient had a prior medical history of mitral baloon valvulotomy performed one year ago. On physical examination the patient had a blood pressure of 130/85

mmHg, a rhythmic pulse rate of 112 beats/min, and a temperature of 36.9°C. There was evidence of an accentuated first heart sound, and a rumbling mid-diastolic murmur with auscultation at the apex of the heart. The pulmonary component of the second sound was also accentuated at the upperleft sternal border.

Electrocardiography (ECG) revealed a normal sinus rhythm with broad and notched p waves in leads II and V_1.

Radiographic manifestations were characteristic "double density" caused by the dilated left atrium, enlargement of the pulmonary artery, and increase in bilateral upper lobe density.

A two -dimensional echocardiographic and Doppler ultrasonographic study demonstrated left atrial dilatation (LAD's: 6.62 cm), thickened and fused mitral valve leaflets with annular calcification, chordal shortening restricting the leaflet motion, and a mobile vegetation attached to the left atrial surface of the anterior mitral leaflet. The mean diastolic gradient across the mitral valve was estimated to be 25 mmHg, and the valve area was calculated to be 0.8 cm2. The aortic valve was found to be normal (Figure I).

All of the serological tests were in normal ranges.

A MVR was planned, and while the patient was beeing prepared for open heart surgery, a sudden complaint of anginal pain reflecting to the left upper extremity begun. A control ECG revealed ST segment elevation more than 5 mm at D_{II}, D_{III} and AVF leads (Figure II). Accepting the patient having a AMI, he was transferred to the ICU and thrombolytic and vasodilator therapy was started immediately. At the 9th hour pathologic Q waves and ST segment depression in leads D_{III} and AVF developed, subseqmently at the 24th hour T wave inversion in leads D_{III} and AVF was seen. During this period the cardiac enzymes were elevated (Table I).

Coronary angiography was performed on the 6th week of AMI, and as seen in Figure III an intracoronary formation causing a 80-90% stenosis was detected at the right coronary artery at the proximal of the acute margin. There was also an additional 50-60 percent stenosis at the third marginal branch of the circumflex system. Left ventriculography was

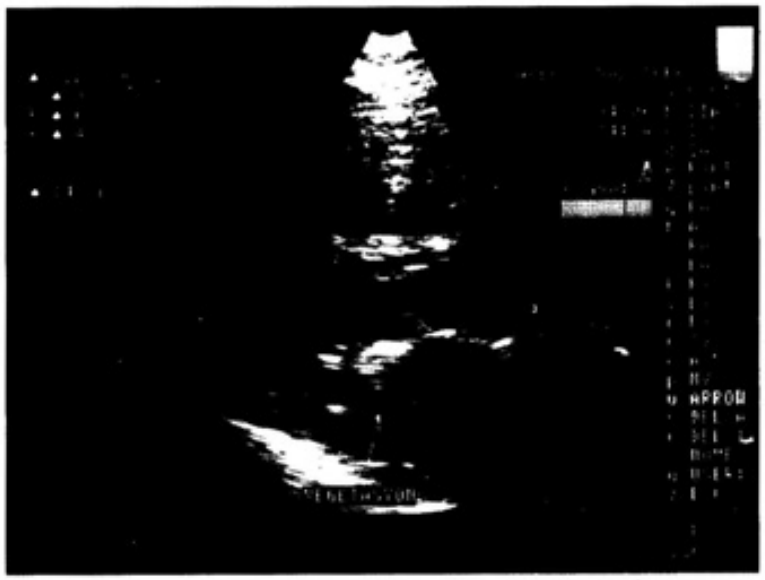

Figure I. Thickened and fused mitral valve with annular calcification. A mobile vegetation attached to the left atrial surface of the anterior mitral valve is seen.

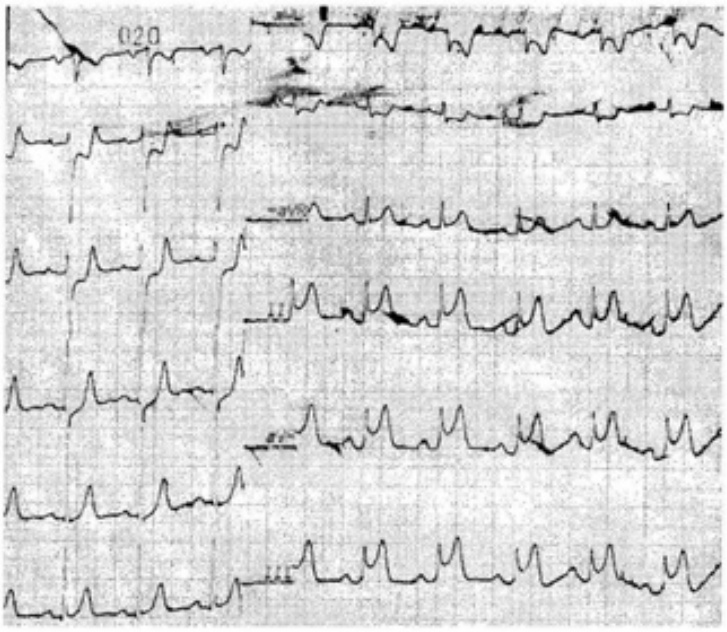

Figure II. ST Segment elevation at D_{II}, D_{III} and AVF leads suggesting an acute myocardial infarction.

not performed because of the mobile vegetation attached on the anterior mitral valve leaflet.

An open heart operation was performed on the seventh week following AMI. Standart cardiopulmonary techniques were instituted by aortic and bicaval cannulation. Myocardial preservation was obtained by isothermic retrograde coronary sinus blood cardioplegia. After moderate hypothermia was obtained, aortic occlusion was performed and cardiac arrest was induced. The right coronary artery was explored and at the proximal of the acute margin, partially mobile, hard and calcified modular mass with a 1.5 cm length was found. The mass was removed from the coronary artery and a saphenous patch plasty was performed to the arteriotomy. An additional aortocoronary saphenous vein bypass was performed to the third marginal branch of the circumflex system.

Mitral exploration revealed a severe stenosis and moderate insufficiency of the valve. Calcified modular vegetations were visible on the

Table I. Cardiac enzyme levels during and 2 weeks after AMI*

	6th hour	24th hour	2nd week
LDH (IU/L)	703	594	348
CPK (IU/L)	865	1128	170
CPK-MB (IU/L)	68	46	17

** Normal ranges of the enyzme were accepted as LDH: 210-500 IU/L, CPK : 20-240 IU/L, CPK-MB : 0-25 IU/L*

Figure III. Right coronary artery obstruction caused by a calcified embolus from the mitral valve.

III- A. Left oblique view.

III- B. Right oblique view

atrial side of the valve. The mitral valve was excised carefully, and a 29 mm size. Medtronic mechanical valve prosthesis was replaced. The patients postoperative course was uneventful.

The pathological examination of the intracoronary mass revealed a calcified vegetation originating from the mitral valve.

The patient was discharged from the hospital on the 14th postoperative day long term controls (18 month) revealed a completely normal life course.

COMMENT

Spontaneous coronary embolisation is a rare cause of AMI. The most common cause of coronary embolism is reported to be liberated cholesterol crystals following PTCA[1]. Additionally, in the literature calcified aortic valve stenosis[2], mitral valve endocarditis[3], bioprosthetic valve degeneration at the aortic location[4], pellet embolism due to gunshot wounds (especially to the right coronary artery[5], systemic lupus erythematosus[6], left atrial myxomas[7], are reported to be rare causes of spontaneous coronary artery embolism.

In this case spontaneous coronary artery embolism due to calcified vegetations on the mitral valve has been reported. The cause of the vegetations might be occult native valve endocarditis. As for as we have searched the literature, we could not encounter this kind of a clinical case.

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