Intramyocardial Calcification in the Elderly*

A Diagnostic and Therapeutic Puzzle

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A 70-year-old woman presented with anular and progressive intramyocardial calcification within a five-year period. She had become increasingly symptomatic with mitral regurgitation and coronary insufficiency during the same period. The subvalvular (mitral) calcified intramyocardial mass was found to be "grumous atherosclerosis." This was obliterated while the mitral valve was replaced with a prosthetic valve and the coronary arteries were bypassed × 3. She is surviving and well four years postoperatively.

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Calcification of the anulus fibrosus of the mitral valve is commonly found in older women.1-3 On roentgenograms, these calcific deposits are mostly found in or below the anulus of the posterior mitral leaflet.1 Pathologic evidence of previous inflammation is often absent in patients in this age group.4-6 The lesion is generally regarded as the benign end stage of a degenerative process within the cardiovascular fibrous skeleton.6 Its association with hemo-dynamically significant calcific aortic stenosis,7 heart block,8 bacterial endocarditis,9 connective tissue disorders, mitral regurgitation,10 and rheumatic endocarditis has been well documented.

Recently we encountered a patient in whom calcific deposits were not only located in this typical place but also extended into the ventricular wall forming a subvalvular intramyocardial calcific mass within a time frame of less than five years.

CASE REPORT

A 70-year-old woman was admitted to the Buffalo (NY) General Hospital with progressive symptoms of shortness of breath on exertion and breathlessness. She denied chest pain. Physical findings showed a blood pressure of 150/90 mm Hg and a regular pulse rate of 74. She had a bruit over the right carotid area. Her chest was clear to percussion and auscultation. The heart was not enlarged clinically. A systolic ejection murmur could be heard at the base radiating to the axilla. Results of abdominal examination were unremarkable. She was afebrile without any history of fever, chills, or elevated white blood cell count. The patient had no history of rheumatic fever.

In 1982, five years prior to this hospital admission, she underwent a work-up for complaints of episodes of numbness involving the right hand and shoulder. Coronary angiography demonstrated coronary artery disease but she was not a surgical candidate for coronary artery revascularization at this time. Her chest roentgenogram showed a nodular upper lobe fibrosis with no evidence of mitral prolapse, mitral regurgitation, or a mass within the heart. The left ventricle was normal size with an ejection fraction in the normal range. No source of emboli could be identified.

Her recent chest roentgenogram showed a spherical mass with a diameter of about 2.5 cm that appeared to be within the left side of the heart (Fig 1). The M-mode and 2-D mode echographic findings showed that there seemed to be a calcified mass on the ventricular side of the posterior leaflet of the mitral valve. It measured approximately 3 × 4.5 cm. There was no evidence of intracavitary thrombus. Her angiographic evaluation demonstrated similar findings. The mass appeared to be located below the posterior leaflet of the mitral valve and to be attached at the mitral anulus but within the ventricular side of the valve (Fig 2). There was a 2 to 3+ mitral regurgitation present seemingly due to the existing mass. This second angiogram revealed significant triple-vessel disease. The ejection fraction (EF) was estimated to be 70 percent. The patient was therefore recommended to have coronary artery bypass grafts with resection of the left ventricular mass and a mitral valve replacement.

Figure 1A (left) and B (right). Posteroanterior and left lateral chest roentgenogram demonstrating a calcified mass in the left side of the heart.

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Intramyocardial cells,

valve

2-D

differential and hypothermic prosthesis). The valve was also extensively damaged by the valve's own infective process.

Operative findings

On cardiopulmonary bypass through a mid sternotomy incision, the left atrium was entered. The posterior leaflet of the mitral valve was bulging. The posterior leaflet was seemingly attached to a mass underneath. The posterior cusp was removed. The mass was deroofed and white cheesy material was found inside. It looked like necrotic material, not like pus. It was cultured and sent for Gram stain.

The valve was fully excised; the cavity in the posterior ventricular wall was repaired with pledged mattress sutures incorporating these into the prosthetic valve ring (Carpentier Edwards, 29-mm prosthesis). The cavity had a firm fibrotic wall. Consequently, a coronary artery bypass graft (CABG)×3 was performed with hypothermic cardioplegia. The patient had an uneventful recovery and four years later is alive and well.

Discussion

This case represented a differential diagnostic dilemma preoperatively and postoperatively. The preoperative differential diagnosis of a calcified myxoma myocardial abscess, intramyocardial tumor, and/or atheromatous mass were entertained. Normal chest roentgenogram and M-mode and 2-D echocardiogram in 1982 opposed the diagnosis of myxoma but favored the diagnosis of grumous atherosclerosis, as described by previous reports.4 A 2+ mitral regurgitation and triple-vessel coronary artery disease (CAD) were the indications for operation.

Operative findings were impressive but excluded myxoma and were suggestive of a sterile intramyocardial abscess of the posterior ventricular wall. Similar findings were reported by Pomerance,19 who described seven cases of calcified mitral rings, which were not uniformly solid but showed extensive central caseation.

The active nature of the disease was questioned since it was absent in 1982 and became symptomatic in 1987. The five-year span of development and presentation were certainly not suggestive of a chronic disease.

Bacteriology

The bacteriology examination reported (4×) no bacterial growth of the patient's blood culture at seven days' incubation. The posterior leaflet of the mitral valve did not show any bacterial growth after five days' incubation. The heart valve was negative for acid-fast bacilli and for fungus, and the stain was not consistent with tuberculosis. Microscopic examination ruled out tuberculosis and/or myocardial abscess.

Pathology

The pathology findings described the mass as a "calcified amorphous debris covered by a thin fibrous membrane." In one focus, a permanent foreign body reaction with histiocytes, lymphocytes, and giant cells could be seen.

Neither neutrophil granulocytes nor fibroblasts surrounding the caseating mass could be found microscopically. Only lymphocytes were present, suggestive of chronic irritation by calcification. The valves did not show any residues like the appearance of muscular blood vessels within the valve due to "aging." Even the clinical appearance did not give any hint of an inflammatory or septic process. The patient had neither fever nor elevation of white blood cell count.

Calcified intramyocardial myxoma is usually pedunculated but may be sessile. Microscopic examination did not substantiate the diagnosis of myxoma.10,11 The Alcian blue staining for mucopolysaccharides was negative. The Verhof van Giesen stain showed no elastic fibers.

The Alizarin red stain for calcium was strongly positive with wide areas of calcification. Cardiac myxomas contain calcium only in small foci. One specimen was consistent with calcified plaques. These plaques had the feature of atheromatous tissue; therefore, this suggested the diagnosis of a variant of grumous atherosclerosis that probably arose from the mitral annulus growing by expansion, not by infiltration, into the tissue itself pushing the ventricular wall and the posterior mitral leaflet out. Mitral atherosclerosis is manifested by a subendothelial deposition of lipid mainly in the annulus and the posterior leaflet, and is regularly followed by calcification and central softening due to necrosis.6

Conclusion

The appearance of an intracardiac lesion in the elderly requires a thorough diagnostic workup, but alone it may not indicate surgery unless it is associated with valve malfunction, as in this patient. Previous chest roentgenograms and/or echocardiograms may be invaluable in assessing the time frame of the process. In the future, frequent screening and surveillance of elderly patients will encounter obviously more of these pictures; and "grumous atherosclerosis" and/or extensive anular and intramyocardial calcification as a differential diagnostic entity will have to be considered also.

References

We describe the features of a rare form of left dominant coronary circulation in which the left anterior descending wraps around the apex forming the posterior descending artery. Similar cases have been reported, but this is the first in which the artery extends beyond the crux into both atrioventricular grooves and supplies the AV node.

**Case Report**

A 54-year-old man presented after 1 h of chest pain, diaphoresis, and dyspnea. Electrocardiography revealed anterior and inferior ST-segment elevation, inferior Q-waves, and inferior T-wave inversion. He promptly received tissue plasminogen activator, his angina improved, and ST segment elevation resolved. The peak creatine phosphokinase was 2,418 U/L. Cardiac catheterization was declined, and he was discharged after nine days.

He returned two days later with chest pain and dyspnea. On examination, blood pressure was 134/86 mm Hg, and pulse was 92 beats per minute. Rales were present over the lung bases. An S₃, and a grade 1/6 apical holosystolic murmur were heard. Electrocardiography revealed sinus rhythm, inferior Q-waves, and a tall R-wave in lead V₁.

Cardiac catheterization demonstrated a large LAD (Fig 1) which wrapped around the apex forming the entire PDA, including a branch to the atrioventricular node. At the crux, the PDA bifurcated giving rise to branches traversing both AV grooves. The branch to the left AV groove in turn gave rise to posterolateral branches. There was a severe stenosis in the proximal LAD which appeared to have superimposed thrombus. A ramus intermedius branch had stenosis in its midportion, and a small right coronary artery had a proximal stenosis. A small left circumflex artery was free of disease. Left ventriculography revealed anterior, septal and apical akinesis and inferior hypokinesis with an ejection fraction of 0.26.

He underwent coronary artery bypass surgery with a left internal mammary artery graft placed to the mid-LAD and saphenous vein grafts to the ramus intermedius and first obtuse marginal branches. He was discharged after an uneventful postoperative recovery.

**Discussion**

There is considerable variation in the blood supply to the inferior and posterior myocardium as first described by

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**Left Coronary Dominance Due to Direct Continuation of the Left Anterior Descending to Form the Posterior Descending Coronary Artery**

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Continuation of the left anterior descending to form the posterior descending artery is rarely observed. We describe the first patient with this variant in whom the coronary artery extends beyond the crux, supplying branches to the atrioventricular node and both atrioventricular grooves.

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**Figure 1 A (left)** Right anterior oblique left coronary angiogram demonstrating a large left anterior descending artery which wraps around the cardiac apex, ascends along the posterior interventricular groove and forms the entire posterior descending artery. This artery gives a branch to form the atrioventricular nodal artery and branches which traverse both atrioventricular grooves. A proximal stenosis, which appeared to have superimposed thrombus, is present in the left anterior descending artery. **B (right)**. Left anterior oblique left coronary angiogram in the same patient. LAD, left anterior descending artery; PDA, posterior descending artery; AVNA, atrioventricular nodal artery; and L, lesion.