Coronary Embolism in Patients with Mitral Valve Prosthesis*

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Coronary embolus resulting in acute myocardial infarction in three patients with mitral valve prosthesis is reported. These episodes occurred despite adequate anticoagulation in all three cases, and one patient had regular sinus rhythm. The clinical importance of this complication is discussed since one patient subsequently died in intractable heart failure as a direct result of this complication.

Myocardial infarction secondary to coronary embolization is infrequently encountered. Approximately 100 cases have been reported with the majority being associated with bacterial endocarditis or syphilis.1 Other causes are paradoxic embolization from atrial or ventricular mural thrombi, avulsed tumor or tumor mass and mitral stenosis with atrial fibrillation.3 Coronary embolization has also been reported in rare instances from thrombi or tumor invading the pulmonary veins,4 after pneumonectomy5 and in cases of tumor invasion of the left atrium.4-6 Since the advent of prosthetic valvular surgery, another source for coronary emboli has been introduced, ie fragments of the prosthetic material7 or more commonly thrombus formed at the surface of the prosthesis. In 1964, Bjork and Malers8 on discussing the late results of mitral valve replacement, reported the first case of coronary embolism arising from a mitral prosthesis. There have been five subsequent cases reported in detail of similar episodes.9-12 It is the purpose of this paper to report three cases of coronary embolus resulting in acute myocardial infarction in patients with prosthetic mitral valves.

CASE REPORTS

CASE 1

This 39-year-old woman was admitted to the Good Samaritan Hospital in November 1967 with sudden onset of severe chest pain radiating to the left shoulder and arm. The pain lasted one-half hour, and was accompanied by diaphoresis and nausea.

Past Medical History: She had had rheumatic fever at age 10; was asymptomatic until age 34, when she started to develop dyspnea on effort. In December 1964 she was hospitalized with congestive heart failure and had physical findings compatible with mitral insufficiency and stenosis, aortic insufficiency and tricuspid insufficiency; she was placed on digitalis and diuretics with very little improvement in her condition. In January 1965, open heart surgery was performed, and a No. 3M Z. Edwards prosthesis was placed in the mitral position; the aortic valve was replaced with a small McGovern prosthesis. The left atrium was found to be free of thrombi. The postoperative course was uneventful, and during the following three years she was maintained on digitalis and anticoagulants and was asymptomatic following the operation. Three years postoperatively the patient developed bilateral leg pain, weakness, urinary incontinence and vertigo, and the clinical picture was diagnosed as multiple sclerosis. She was, however, able to ambulate with crutches and perform light housework without difficulty until the onset of severe chest pain in November 1967.

Physical examination revealed an alert, moderately well-developed woman complaining of severe precordial pain and nausea. Blood pressure was 140/90 mm Hg on both arms; respiratory rate 18/min, heart rate 96/min and regular. Pertinent physical findings revealed the lungs to be clear to auscultation and percussion. The opening and closing clicks of the mitral and aortic valve prostheses were well heard and there was no evidence for valve variance. A grade III/VI systolic ejection murmur was heard at the aortic area which was interpreted as representing turbulent flow across the aortic prosthesis. There was loss of muscle strength in all muscle groups in both lower extremities.

The electrocardiogram at admission (Fig 1) revealed signs of an acute inferolateral wall myocardial infarction. The prothrombin time at admission was 18 seconds with a control of 13 seconds. Serial serum glutamic oxaloacetic transaminase (SGOT) rose from two units to a maximum of 120 units 12 hours later during the hospital course. The chest x-ray picture showed moderate left ventricular enlargement.

The patient was placed in the coronary care unit and, aside from occasional ventricular extrasystoles, her clinical course was essentially uneventful with only occasional recurrence of chest pain. She was discharged from the hospital three weeks later and remained free of cardiac symptoms.

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Figure 1. Case 1, serial electrocardiograms showing changes of an acute inferolateral myocardial infarction.

Figure 2a. Case 1, right coronary arteriogram with the patient in the left anterior oblique projection demonstrating normal right coronary artery. The aortic and mitral valve prostheses are seen.

Figure 2b. Case 1, left coronary arteriogram with the patient in the left anterior oblique projection demonstrating patent left anterior descending and circumflex arteries.
Cardiac catheterization was performed November 27, 1968, and the hemodynamic data are summarized in Table 1. Subsequent selective coronary arteriograms performed several months later showed normal and patent vessels (Fig 2 A and 2 B) without evidence for coronary atherosclerosis.

Case 2

A 37-year-old man was admitted to Good Samaritan Hospital in December 1968 complaining of a sudden onset of severe precordial pain which began one hour prior to admission.

Past Medical History: At age eight, the patient had rheumatic fever. Symptoms and signs of congestive heart failure first appeared in 1954 at which time he was started on digitals. In 1957 he developed atrial fibrillation, and cardiac catheterization revealed mitral stenosis and insufficiency and a large left atrium. In 1958 he suffered left-sided hemiplegia but recovered from the episode without neurologic deficit. In February 1958 a mitral commissurotomy was attempted, but the left atrium was found to be large and its wall heavily calcified and immobile. The left atrium endocardium was lined with adherent thrombi. Because of technical difficulties, it was elected not to attempt a mitral commissurotomy. He was, thereafter, placed on anticoagulant therapy and remained severely limited in his activity. In April 1967 cardiac catheterization was performed, and the hemodynamic data are summarized in Table 1. The cineangiocardiograms revealed a dilated right ventricle and moderate degree of tricuspid insufficiency. The mitral valve was heavily calcified and revealed moderate regurgitation. In July 1967 an open heart procedure was performed and the mitral valve replaced with a No. 9 Kay-Shiley prosthesis. Thrombi were removed from the left atrium. The postoperative period was uneventful. However, the patient subsequently developed hemolytic anemia which was successfully controlled with steroids. Subsequently, the patient had several brief episodes of hemiparesis without major sequelae. Throughout this time the prothrombin time was found to be in the therapeutic range of 20 to 25 seconds.

Physical examination in December 1968, at the time of episode of chest pain, revealed an acutely ill, pale, diaphoretic man with a blood pressure of 80/60 mm Hg. The heart rate was 102/min and irregular; the respiratory rate was 24/min and labored. The lungs were clear to auscultation and percussion. Examination of the heart revealed normal opening and closing clicks of the mitral prosthesis and a third heart sound; no murmurs were heard (Fig 3).

The electrocardiograms and vectorcardiograms (Fig 4) in December 1968 showed atrial fibrillation and signs of an acute anterolateral myocardial infarction. Serial SGOT rose from initial level of 30 units to a maximum of 655 units; the CPK rose to a maximal of 296 units 24 hours following admission. His prothrombin time at the time of admission was 24 seconds with a control of 13 seconds. While in the coronary care unit, aside from frequent ventricular extrasystoles, his course was satisfactory, and he was discharged three weeks after admission. In February 1969 the patient again began to have increasing exertional dyspnea and marked fatigability. A grade III/VI, regurgitant, systolic murmur was heard at the tricuspid area and was interpreted as representing tricuspid insufficiency. His liver was enlarged and a large regurgitant "V" wave was seen in the jugular venous pulse. Increasing digitalization was to no avail and diuretic therapy gave no improvement. Cardiac catheterization was performed in April 1969, and the results are summarized in Table 1. Selective cineangiocardiograms revealed enlargement of all heart chambers and poor left ventricular contraction. Marked degree of tricuspid insufficiency was present, but the mitral valve prosthesis appeared to be functioning well without evidence of insufficiency. Selective coronary arteriogram showed a large filling defect in the proximal third segment of the left anterior descending coronary artery which was attributed to the presence of an organized thrombus (Fig 5). The vessel appeared, otherwise, free of atherosclerotic disease. The left circumflex and the right coronary arteries were normal.

The patient remained confined to bed; however, he continued to have heart failure which did not respond to medical

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**Table 1—Hemodynamic data.**

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<th>PA S/D/M v/ Mean</th>
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**Figure 3.** Case 2, phonocardiogram at the mitral area (MA), tricuspid area (TA), pulmonic area (PA), aortic area (AA), carotid tracing (CT) and lead II of the electrocardiogram demonstrating the normal opening and closing clicks of the prosthetic mitral valve. Phonocardiograms were recorded with filters 50 to 500 cycles per second (CPS). There is short early high frequency systolic ejection murmur recorded in the mitral area.
therapy, and he died with a low output syndrome in April of 1969. Postmortem studies were not done.

Case 3

A 32-year-old woman was seen at another hospital for severe dyspnea, chest pain, nausea, vomiting and hypotension. Digitalis and furosemide were administered and she was transferred to the Good Samaritan Hospital.

Past Medical History: The patient had rheumatic fever in childhood and a heart murmur was first detected at age 16. Congestive heart failure occurred during the secondary pregnancy in 1954. The patient was placed on digitalis for congestive heart failure in 1958, and in 1961 she developed atrial fibrillation. While hospitalized in 1962 for tubal ligation, signs of mitral stenosis and insufficiency were present. Because of increasing limitation of activities, she underwent open heart surgery in October 1966 when the mitral valve was replaced with a No. 7 Cutter prosthesis. No clots were found in the left atrium at the time of operation. She had an uneventful recovery and, in 1967, cardioversion was attempted unsuccessfully. The patient remained on anticoagulants and her prothrombin time was frequently checked and found to be within therapeutic range (20 to 26 seconds). She

Figure 4. Case 2, serial Frank vectorcardiograms in the frontal plane (FP), sagittal plane (SP) and horizontal plane (HP) with the electrocardiogram. Note the characteristic evolving vectorcardiographic and electrocardiographic changes of an extensive anterior wall myocardial infarction. The tracing taken October 11, 1967 shows right ventricular hypertrophy (type C).

Figure 5. Case 2, left coronary arteriogram with the patient in a right anterior oblique projection. Note a filling defect in the proximal third segment of the left anterior descending artery at a place indicated by an arrow. The prosthetic mitral valve is seen.
was readmitted in March 1967 because of hepatitis from which she recovered. In December 1967 she noted decrease in her exercise tolerance, and cardiac catheterization was subsequently performed in January 1968; the hemodynamic data are summarized in Table I. Cineangiography revealed a dilated left ventricle and poor ventricular contractions. There was no evidence of insufficiency around the mitral prosthesis.

On physical examination in March of 1967 she showed moderate dyspnea and she was complaining of severe precordial and epigastric pain. Her skin was cold and clammy; the blood pressure 80/50 mm Hg, the heart rate was 106/min and irregular (atrial fibrillation). The respiratory rate was 28/min and labored. Bales were heard over both lung bases. The auscultation of the heart revealed normal closing and opening clicks of the mitral valve prosthesis. A third heart sound was present. Her white blood cell count (WBC) on admission was 30,300. Her prothrombin time was 24 seconds with 13 seconds control. The chest x-ray picture was compatible with pulmonary edema and moderate cardiomegaly. The electrocardiogram showed signs of an acute subendocardial myocardial infarction (Fig 7). Serial SGOT rose from 106 units to 130 units 24 hours after admission, and this figure subsequently fell to 28 units on the third day of hospitalization. She gradually improved on the standard therapy for acute myocardial infarction, and she was discharged three weeks after admission.

Discussion

The present study emphasized an uncommon but clinically important complication of mitral valve replacement. It is reasonable to assume that these episodes of myocardial infarction in the three patients were secondary to coronary embolization from thrombus originating in the prosthetic valve.

This is based on the fact that all patients were below age 40, two of the three were women and menstruating and without previous clinical evidence of coronary artery disease. In addition, selective coronary arteriograms in all three cases failed to reveal evidence for coronary atherosclerosis; in one case a definite filling defect in the left anterior descending artery was interpreted as evidence for thrombosis of that vessel. It is apparent that neither

![Figure 6. Case 3, phonocardiogram at the mitral area (MA), tricuspid area (TA), pulmonic area (PA), aortic area (AA), carotid tracing and lead II of the electrocardiogram demonstrating the normal opening and closing clicks of the prosthetic mitral valve.](image)

![Figure 7. Case 3, serial electrocardiograms demonstrating marked abnormality of T waves and ST segment in the precordial leads, highly suggestive of an acute subendocardial myocardial infarction.](image)
adequate anticoagulation or the presence of a normal sinus rhythm prevented the occurrence of this complication in the presently reported cases as they have been shown not to prevent systemic embolism to an absolute degree. 13-14 Nevertheless, it is reasonable to assume that the presence of atrial fibrillation and/or inadequate or no anticoagulation may favor the occurrence of coronary embolization.

The exact incidence of coronary embolization in patients with mitral valve prosthesis remains unknown. The present study was not intended to provide a statistical incidence of this complication, since no adequate follow-up could be obtained in all cases subjected to mitral valve replacement performed at this institution. Approximately 120 mitral valve replacements have been performed at this hospital over the past five years.

Duvoisin and co-workers13 found several incidences of coronary embolus in a total of 90 embolic episodes in 166 patients with Starr-Edwards mitral prosthesis. It follows that this problem is probably more prevalent than is reported.

The importance of this problem appears to have great clinical significance because of the increased numbers of this type of operation. Obviously the occurrence of an acute myocardial infarction in patients who already have various degrees of depression of ventricular performance may carry grave prognostic significance. One of the presently reported cases eventually died from intractable left ventricular failure which developed following the episode of coronary embolization. The second patient who had two episodes of acute myocardial infarction is doing poorly because of repeated episodes of heart failure.

It is of importance to emphasize that coronary arteriography in these cases may be normal probably due to recanalization of the thrombus. However, areas of left ventricular dyskinesia are almost always present as demonstrated in the left ventricular angiograms in these three cases representing loss of contraction of the infarcted segment of the ventricular wall. In addition, it is interesting to note the high incidence of inferior wall myocardial infarction in these cases as opposed to the other location.

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