Recurrent Calcium Emboli in a Patient with Aortic Stenosis*

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We report a patient with moderate aortic stenosis who had two episodes of calcium embolus to the left popliteal artery within ten months. This case suggests that calcium emboli can be recurrent and that major calcium emboli should warrant consideration of valve replacement, even in patients with mild aortic stenosis. (Chest 1989; 96:1433-34)

Little information regarding the natural history of calcium emboli from diseased heart valves is available and it is not known whether this complication should be considered an indication for surgical valve replacement. We report a unique case in which a patient had two calcium emboli to the same location in a period of ten months.

Spontaneous calcific emboli have been considered rare; however, necroscopy examination of patients with aortic stenosis reveals an incidence of 19 percent. The majority of these emboli were to the coronary arteries. Clinically obvious cases seem to be much less common.

In a series of 735 embolectomies for major arterial emboli, the heart was the source of the embolus in 90 percent of the cases. Seven percent of the patients gave a history of rheumatic heart disease. The case we report is the only instance in which calcium embolic material was an operative and pathologic finding. The incidence of recurrence following a calcium embolus is not known and thus, whether to recommend valve replacement in such patients is problematic, especially if their aortic stenosis is not severe.

CASE REPORT

First Visit

A 38-year-old man was admitted to Good Samaritan Hospital on May 6, 1987, with the tentative diagnosis of arterial embolus to the left lower extremity. After mowing his lawn the day previously, he had become dizzy and had dull retrosternal pain that lasted only seconds. He then noticed pain in his left calf when he walked. On standing still, the pain immediately went away. He gave a history of five separate hospital admissions for rheumatic fever as a child. Since that time he had been asymptomatic and had received no regular medical care. He reported no other significant medical history.

Physical examination showed that the patient's left limb had normal color and was slightly cool to palpation. The femoral and popliteal pulses were easily palpable, but the posterior tibial and dorsalis pedis pulses were absent on the left side. On the right side, all pulses were of normal amplitude. Auscultation of the heart revealed a grade 3/6 systolic ejection murmur audible at both upper sternal borders, with radiation to the neck. No diastolic murmur was heard. The electrocardiogram was normal. An echocardiogram showed thickened aortic leaflets (Fig 1) but was otherwise normal. The Doppler examination revealed a peak systolic velocity of 4 m/s, indicating a 64-mm gradient across the aortic valve. The estimated valve area using the continuity equation was 1.0 cm². A duplex scan was obtained which showed a normally beating left popliteal artery; however, the artery could not be visualized at its termination. Because the aortic stenosis appeared to be moderate and the patient was asymptomatic, valve replacement was not recommended.

The operative findings were as follows: The popliteal artery was exposed with the standard medial incision at the knee level. The proximal artery was pulsating but the distal artery was pulseless.
Recurrent Calcium Emboli

and obviously contained a thrombus. This thrombus extended into the anterior tibial artery. The artery was opened and found to contain fresh clot, which was removed. Following this, a firm mass, whitish in hue and friable in nature was removed from the anterior tibial artery and tibial-peroneal trunk (Fig 2). There was no evidence that this material was attached to the intima. A firm, trailing, well-defined thrombus extended from this gray friable material into the origin of the anterior tibial artery for a distance of approximately 4 mm. A long fresh thrombus was removed from both the anterior tibial and posterior tibial arteries by passing a No. 3 and 4 Fogarty embolectomy catheter through them. After removal of the cast and thrombus, there was excellent retrograde bleeding. Postoperatively, pulses were restored in the foot.

Second Visit

Ten months later, the patient returned with the same symptoms as on his first visit; however, their onset was slower. About a week prior the pain had begun in his left leg on walking and progressed to the point that he experienced severe pain in his left calf after taking only a few steps. Both limbs appeared to be of normal color and were similar, except that the dorsalis pedis pulse on the left was absent. The posterior tibial artery was palpable. Using a handheld Doppler, the posterior tibial artery could be heard very well, but the dorsalis pedis and anterior tibial arteries produced no signals.

The patient was admitted to the hospital and an arteriogram was obtained. It showed a typical, localized, bullet-shaped mass in the popliteal artery and the anterior tibial artery at the site where the anterior tibial is normally visualized. A duplex scan was obtained. Calcium could be observed in the popliteal artery but not clearly enough to enable the diagnosis of embolus to be made without the corroborating arteriogram.

The operative findings were as follows: The old incision was reopened and the popliteal artery exposed. On opening the artery, the findings were identical to those at the earlier operation. There was definite atheromatous material and fibrin which formed a plug at the origin of the anterior tibial artery (Fig 3). The mass was easily dissected free. When this was removed, the remainder of the artery appeared smooth, glistening and normal. A No. 4 Fogarty embolectomy catheter was passed distally in the posterior tibial artery.

Because the artery had been narrowed, a saphenous vein patch was inserted for a distance of 3 to 4 cm from the popliteal artery to the tibial peroneal trunk. An arteriogram was obtained and showed a patent arterial tree. The patient did well postoperatively.

After this second episode, aortic valve replacement was recommended. Prior to valve replacement, coronary arteriography was performed. It was elected not to attempt to cross the aortic valve as the patient was considered to be at risk for embolization from the diseased aortic valve.4

At operation, the coronary arteries were normal. The aortic valve was replaced by a Medtronic Hall prosthesis. The postoperative course was complicated by a large pericardial effusion requiring drainage. After resolution of the effusion the patient did well. Pathologic examination of the patient's aortic valve revealed a fibrotic and calcified cardiac valve with foci of myxomatous degeneration. The valve was severely fragmented and the number of raphes could not be determined.

CONCLUSION

The incidence of calcium emboli from calcified aortic and mitral valves is not known with certainty, although autopsy studies suggest that they may be more common than previously appreciated. They are most commonly associated with rheumatic valvular disease.4 Patients are at risk during catheterization4 and for this reason, we elected not to cross the aortic valve. Emboli are also common at the time of surgery,4 although there was no evidence that this occurred in the present case.

Our patient had a second major embolus ten months after his first. This suggests that patients with a first calcium embolus are at risk for recurrence. Autopsy studies also demonstrate that embolic events tend to be recurrent.1,2 Furthermore, while working in experimental pathology in 1955, Whisnant7 demonstrated streamlining of flow such that stainless steel bearings, injected into the carotid arterial system of the dog, repeatedly lodged in the same branch of the left middle cerebral artery.7

We suggest that patients with major calcium emboli be considered for valve replacement even if the degree of stenosis would not otherwise warrant valve replacement.

REFERENCES


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