Calcific coronary embolization associated with cardiac valve replacement

Necropsy x-ray study

I. Steiner, A. Hlava, and J. Procházka

From the Departments of Pathology, Radiology, and Surgery, Faculty Hospital, Hradec Králové, Czechoslovakia

The hearts of 14 patients dying early after replacement of a calcified aortic or mitral valve were examined for the presence of calcific coronary emboli. The incidence of embolization as shown by x-ray film was 92.9 per cent. The number of emboli in individual cases ranged from 4 to 119. Replacement of an aortic valve was associated with a more severe embolization than replacement of a mitral valve. The mechanisms of calcific embolization are discussed and preventive surgical measures suggested.

A calcified aortic or mitral valve may be a source of systemic calcific embolization. This may occur spontaneously (Moragues, Bawell, and Shrader, 1950; Wigle, 1957; Holley *et al.*, 1963a), or after various intracardiac procedures, particularly operations on such valves (Glotzer, Shaw, and Scannell, 1962; Holley *et al.*, 1963b).

Several necropsy studies of cardiac valve replacement mention the finding of calcific coronary emboli; however, the true incidence and significance of this complication are unknown (Hudson, 1965, 1970; Morales, Fine, and Taber, 1967; Henson *et al.*, 1969; Colapinto and Silver, 1971; Roberts, Bulkley, and Morrow, 1973).

Patients

During the period September 1973 to August 1975 there were 34 deaths following prosthetic heart valve replacement. We have examined the hearts of 14 patients who met the following criteria: (1) severe calcification of the diseased valve; (2) death in the first postoperative week.

Of the 14 patients, 12 were men and 2 were women; their ages ranged from 39 to 60 years (average 47). The aortic valve alone was replaced in 7 patients and the mitral valve alone in another 7. The valve was excised intact in 8 patients, and in several pieces in 6 (5 aortic, 1 mitral). The operations were performed under moderate body hypo-

Received 12 December 1975.

thermia and anoxic heart arrest induced by clamping the ascending aorta. Coronary arterial perfusion was not used.

Methods

The heart was excised from the chest organs and the ventricles sliced at 1 to 1.5 cm intervals, beginning at the apex and stopping 2 to 3 cm from the atrio-ventricular valve rings. Depending on the heart size, 5 to 7 slices were obtained. The coronary arteries on the basal portion of the heart were opened longitudinally.

The formalin-fixed slices were x-rayed by Mammograf (Siemens) on a Mammoray T 1 film at focus size 0.6 mm, 28 kV and 200 to 250 mA, with a focus-film distance of 47 cm.

The site of the calcific particles was recorded on transparent paper, with the ventricular cross-sectional plane divided into 5 segments: anterior, lateral, posterior, the right ventricle, and the septum (Fig. 1). According to the greatest dimension, the particles were classified as small (<0.5 mm), medium-sized (0.5-1 mm), and large (>1 mm).

The nature of most of the particles was checked, either grossly, or histologically.

Results

Gross calcific coronary embolus was found in 2 patients after aortic valve replacement, measuring

FIG. 1 Distribution of the total 513 calcific particles (307 coronary emboli plus 206 intraventricular) in aortic vs. mitral valve replacement.

respectively, 4 mm and 2.5 mm in greatest dimension. In one case the embolus was in the anterior descending artery and in the other in the posterior descending branch of the right coronary artery.

Calcific coronary emboli (Table 1, Fig. 2)

These were present in 13 of the 14 patients (93%). The number of emboli in individual cases ranged between 4 and 119. Of the total 307 emboli, 208 were small, 63 medium-sized, and 36 large. One hundred and twenty (39%) were lodged in the subepicardial arteries, and 187 (61%) in the intramyocardial branches.

There was no obvious difference in the number of emboli between the patients with the valve excised intact and those with the valve removed in several pieces.

Calcific particles in left ventricular cavity (Table 1, Fig. 3)

These were present in 12 patients, adjacent to the endocardium between the papillary muscles and the trabeculae. The number of particles in individual cases ranged between 1 and 47. Of the total 206 intraventricular particles, 168 were small, 29 medium-sized, and 9 large. The majority were on the posterolateral wall, between both papillary muscles. In most instances the number of particles was related to the number of coronary emboli.

Aortic vs. mitral valve replacement (Table 2, Fig. 1)

Both the emboli and the intraventricular particles were much more common in the aortic valve replacement, as shown in Table 2. There was also a different distribution of the emboli (Fig. 1); in

TABLE 1 Calcific coronary emboli associated with cardiac valve replacement in 14 necropsy patients

Case No.	Valve replaced	Calcific coronary emboli				Calcific particles in LV cavity			
		Small	Medium-sized	Large	Total	Small	Medium-sized	Large	Total
1	Aortic	25	13	2	40	20	5	0	25
2	Mitral	3	0	1	4	0	0	0	0
3	Aortic	22	6	3	31	31	5	2	38
4	Aortic	19	1	0	20	20	3	1	24
5	Mitral	7	0	0	7	5	4	0	9
6	Mitral	7	3	2	12	3	0	0	3
7	Aortic	74	27	18	119	17	0	0	17
8	Aortic	3	1	0	4	33	8	6	47
9	Mitral	7	2	1	10	7	2	0	9
10	Mitral	0	0	0	0	0	0	0	0
11	Mitral	3	1	0	4	4	0	0	4
12	Aortic	32	7	8	47	23	2	0	25
13	Mitral	3	2	0	5	1	0	0	1
14	Aortic	3	0	1	4	4	0	0	4
Total		208	63	36	307	168	29	9	206

FIG. 2 An x-ray picture of a midventricular slice of the heart of Case 7 shows numerous calcific coronary emboli both subepicardially and intramurally. The calcified aortic valve was removed in one piece 7 days before death. The postoperative course was dominated by features of low cardiac output syndrome with ischaemic changes on the electrocardiogram ($\times 1.3$).

aortic valve replacement the left coronary artery appears to be more severely affected and the right coronary artery less so, when compared with the mitral valve.

Histological examination of the myocardium showed focal necroses in the left ventricle and the ventricular septum in each of the 10 patients who survived more than 1 day. These were extensive in 3 patients, all of whom had aortic valve replacement. In the one case with gross embolus in the posterior descending coronary branch a small myocardial infarction was found distal to the site of occlusion.

Discussion

This study has shown that replacement of a calcified heart valve, and particularly of the aortic valve, is frequently associated with calcific coronary embolization. The aortic valve, compared with the mitral valve, is usually more heavily calcified and the calcium more often extends into the adjacent

heart structures. During resection of such a valve, fragments of calcium fall into the left ventricular cavity. Those which get entrapped between the trabeculae may not be removed by the surgical toilet. After resumption of blood flow some of them are washed out to become systemic emboli. This is probably the basic mechanism of calcific coronary embolization. Thus, the pattern of embolization in mitral valve replacement (Fig. 1), in which this basic mechanism operates alone, may be regarded as reflecting the average distribution of the coronary blood flow.

In aortic valve replacement an additional mechanism is suggested by the more severe involvement of the anterior and lateral heart segments which are supplied by the left coronary artery (Fig. 1); during manipulation with the valve, fragments of calcium may fall directly into the coronary ostia, mainly into the left, which is widely exposed.

The x-ray method used in this study proved capable of detecting calcific particles as small as

TABLE 2 *Calcific coronary emboli associated with aortic vs. mitral valve replacement in 14 necropsy patients*

		<i>Valve replaced</i>	
		<i>Aortic (7 cases)</i>	<i>Mitral (7 cases)</i>
Calcific coronary emboli	No. of patients affected	7	6
	Total no. of emboli	265 (86.3%)	42 (13.7%)
	No. of emboli in individual patients	4-119 (av. 38)	0-12 (av. 6)
Calcific particles in LV cavity	No. of patients affected	7	5
	Total no. of particles	180 (87.4%)	26 (12.6%)
	No. of particles in individual patients	4-47 (av. 26)	0-9 (av. 4)

0.1 to 0.15 mm. It also allowed easy distinction of the fragments of calcium from the shell-like calcifications of atheromatous lesions.

Particles smaller than 0.1 mm are unlikely to be detected during histological examination except by chance. Nevertheless, in our previous study of 68 necropsy patients (Steiner, Křivková, and Procházka, 1975), the histological incidence of calcific coronary emboli from the calcific valves only was 24 per cent with isolated mitral valve replacement, 53 per cent with isolated aortic valve

replacement, and 75 per cent with mitral plus aortic valve replacement. The true incidence and extent of calcific microembolization must, however, be much higher, as only 2 to 3 per cent of the total heart mass is taken for histology and from this the paraffin sections form approximately 1/200 to 1/300.

Examination of the hearts of patients dying at longer intervals after the operation (not included in this study) allowed us to follow the natural history of the emboli. Even several years after operation

FIG. 3 *An x-ray picture of a midventricular slice of the heart of Case 8 shows calcific particles in the left ventricular cavity. Most particles are on the endocardium of the posterolateral wall between the papillary muscles. The calcified aortic valve was removed in 5 pieces. The patient died from diffuse hypoxic damage of the brain 5 days after operation. ($\times 1.5$.)*

the calcific particles can be seen on x-ray film, both as coronary emboli and intraventricularly. Microscopically, they are completely covered by collagen, as a result of the intimal, or endocardial reaction. The embolus may occasionally be extruded from the artery and is found perivascularly.

It is difficult to assess the clinical importance of calcific coronary embolization as similar ischaemic myocardial lesions may also be seen with replacement of non-calcified valves. It was thought to be the prime cause of death in 2 patients and its role was seriously considered in an additional 3 who died with low cardiac output syndrome, which was otherwise unexplained by the necropsy.

As the coronary circulation receives approximately 4 per cent of the cardiac output, it may be presumed that a considerable number of the calcific particles from the left ventricle embolize to other organs, e.g. the brain and the kidneys.

The results of this study warrant the recommendation of certain preventive surgical measures:

- (1) Occlusion of the coronary ostia during manipulation of the aortic valve.
- (2) Removal of the calcium extending into the valve ring and the surrounding structures.
- (3) A thorough cleansing of the left ventricle and the aortic root before the circulation through the heart is restored.

We are grateful to Mrs. J. Zdražilová for her technical help.

References

- Colapinto, N., and Silver, M. D. (1971). Prosthetic heart valve replacement. Causes of early postoperative death. *Journal of Thoracic and Cardiovascular Surgery*, **61**, 938.
- Glutzer, D. J., Shaw, R. S., and Scannell, J. G. (1962). Calcific coronary emboli following open valvuloplasty for aortic stenosis. *Journal of Thoracic and Cardiovascular Surgery*, **43**, 434.
- Henson, D. E., Najafi, H., Callaghan, R., Coogan, P., Julian, O. C., and Eisenstein, R. (1969). Myocardial lesions following open heart surgery. *Archives of Pathology*, **88**, 423.
- Holley, K. E., Bahn, R. C., McGoon, D. C., and Mankin, H. T. (1963a). Spontaneous calcific embolization associated with calcific aortic stenosis. *Circulation*, **27**, 197.
- Holley, K. E., Bahn, R. C., McGoon, D. C., and Mankin, H. T. (1963b). Calcific embolization associated with valvotomy for calcific aortic stenosis. *Circulation*, **28**, 175.
- Hudson, R. E. B. (1965). *Cardiovascular Pathology*, Vol. 1, p. 643. Edward Arnold, London.
- Hudson, R. E. B. (1970). *Cardiovascular Pathology*, Vol. 3, p. 408. Edward Arnold, London.
- Moragues, V., Bawell, M. B., and Shrader, E. L. (1950). Coronary embolism: review of the literature and report of a unique case. *Circulation*, **2**, 434.
- Morales, A. R., Fine, G., and Taber, R. E. (1967). Cardiac surgery and myocardial necrosis. *Archives of Pathology*, **83**, 71.
- Roberts, W. C., Bulkley, Bernadine, H., and Morrow, A. G. (1973). Pathologic anatomy of cardiac valve replacement: a study of 224 necropsy patients. *Progress in Cardiovascular Diseases*, **15**, 539.
- Steiner, I., Krivková, I., and Procházka, J. (1975). Pathology of cardiac valve prosthetic replacement: A study of 68 necropsy patients. *Sborník vědeckých prací LFKU Hradec Králové*, **18**, 159.
- Wigle, E. D. (1957). Myocardial fibrosis and calcareous emboli in valvular heart disease. *British Heart Journal*, **19**, 539.

Requests for reprints to Dr. I. Steiner, Department of Pathology, District Hospital, 532 03 Pardubice, Czechoslovakia.