Spontaneous calcific coronary embolisation: necropsy x-ray study

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SUMMARY Thirty-one hearts with advanced calcification of one or both left heart valves were investigated for the presence of spontaneous calcific emboli. X-ray (Mammograf) showed coronary emboli in 14 cases, that is, in 45% of the whole series. In seven cases (23%) calcific particles were also present in the left ventricular cavity. A total of 265 calcific emboli (205 intracoronary plus 60 intraventricular) was demonstrated. The embolisation occurred most frequently and most extensively in association with postrheumatic mitral valve disease. Coronary calcific embolisation must be considered in the differential diagnosis of symptoms of ischaemic heart disease in patients suffering from valvular heart lesions.

In a previous paper the incidence and extent of calcific embolisation into coronary arteries after replacement of calcified valves with prostheses were investigated.¹ Slices of heart were x-rayed by Mammograf apparatus.

On histological examination of the x-ray findings we occasionally found emboli with signs of advanced organisation which, therefore, must have been present in the vessel before operation. Thus the following questions arose: whether, how often, and to what extent fragments of calcified tissue are spontaneously dislodged from calcified valves and form emboli in the coronary arterial circulation.

Material and methods

In the period 1975-9, 31 hearts from routine necropsies were examined. The criteria for inclusion in this study were (1) advanced (subjectively evaluated) calcification of one or both left heart valves, and (2) the fact that the patient had been neither operated on nor examined by an invasive technique because of the valve disease. Otherwise it was an unselected series.

The method of examination was the same as in our previous study.¹ The heart was excised from the chest organs and the ventricles were sliced at 1 to 1.5 cm intervals, beginning with the apex and stopping 2 to 3 cm from the atrioventricular valve rings. Depending on the size of the heart, five to seven

slices were obtained. The major epicardial arteries in that part of the heart left after the ventricular slices had been removed were opened longitudinally.

The type of calcific valve disease was evaluated with respect to aetiology² and the presence or absence of ulceration, that is, injury of the valvular endocardium with uncovering of the calcified material.

The formalin-fixed heart 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 five segments: anterior, lateral, posterior, right ventricle, and 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).

When the nature of the calcification in the radiograph was not clear the site was examined histologically.

Results

Of the 31 patients, 16 were men and 15 women; their ages ranged from 35 to 86 (average 64) years. The aortic valve was calcified in 24 cases, the mitral valve in three, and both valves in four cases. In 15 patients (11 men and 4 women, average age 58.7 years) aortic valve disease was based on a congenital lesion—bicuspid or unicuspid aortic valve.

Fig. 1 Distribution of the total 265 spontaneous calcific emboli (205 intracoronary plus 60 intraventricular).

In eight patients (3 men and 5 women, average age 60.0 years) the lesion was rheumatic in origin—aortic and mitral in four, mitral in three, and aortic in one patient. In eight patients (2 men and 6 women, average age 79.4 years) it was a senile type of aortic stenosis.

In 20 cases the valve involved was ulcerated, and crumbly calcific material was uncovered (Fig. 2). In the remaining 11 cases the valvular endocardium was intact.

Macroscopically, calcific emboli were found in two cases. One case was a 39-year-old man with stenosis of a unicuspid aortic valve. The embolus was situated in the anterior descending branch of the left coronary artery. A postinfarction myocardial scar was located in the apical portion of the left ventricular anterior wall. The other was a 67-year-old woman with mitral disease, and the embolus was found in the peripheral portion of the right coronary

Fig. 2 Unicuspid unicommissural stenotic aortic valve from a 39-year-old man viewed from above. Masses of calcium erupt through the valvular endocardium. X-ray of this heart is shown in Fig. 4.

artery. In both cases x-ray examination showed the presence of more emboli.

X-ray showed calcific emboli in 14 cases, that is, in 45% of the whole series (Figs 3 and 4). The numbers of coronary emboli in individual hearts were 37, 33, 32, 30, 24, 13, 10, 10, 6, 3, 3, 2, 1, and 1, respectively, totalling 205. Of this number, 121 were small, 53 medium-sized, and 31 large. The distribution of the emboli in the ventricular walls is shown in Figure 1. Sixty-six emboli (32.2%) were found in the posterior wall, 60 (29.3%) in the lateral wall, and 39 (19.0%) in the anterior wall of the left ventricle, 20 (9.8%) in the right ventricular wall, and 20 (9.8%) in the ventricular septum. It is probable,

Fig. 3 X-ray of a midventricular slice from a case of calcific aortic stenosis. In the posterior and lateral walls of the left ventricle calcific coronary emboli are seen both subepicardially and intramurally.

Fig. 4 X-ray of a midventricular slice from a case of calcific aortic stenosis (the valve of this case is shown in Fig. 2). There is a gross calcific embolus in the anterior interventricular coronary branch. In addition, two large particles are seen between the trabeculae on the endocardium of the left ventricular posterior wall.

therefore, that the emboli occur with roughly equal frequency in the left and right coronary ostia.

In seven cases (5 aortic, one mitral, one aorto-mitral) calcific particles were also seen in the left ventricular cavity between trabeculae on the endo-

cardium (Fig. 4). The numbers of these intraventricular particles were, in individual cases, 19, 14, 9, 8, 8, 1, and 1, respectively, totalling 60. Forty-six were small, eight medium-sized, and six large.

Thus 265 calcific emboli (205 intracoronary plus 60 intraventricular) were demonstrated radiologically.

On assessing the findings of 265 emboli in 14 cases with regard to the valve involved, embolisation was present in nine of 24 cases of aortic valve disease (37%). The total number of emboli was 160, an average of 18 per case. In mitral disease, embolisation was present in all three cases (100%), and the total number of emboli was 48, an average of 16 per case. Emboli were found in two of four cases of aorto-mitral disease (50%); the total number of emboli was 57, an average of 28 per case.

Embolisation occurred in 47% of cases with congenital aortic disease, in 75% with rheumatic disease, and in one case only of senile aortic stenosis.

Ulceration of the valve was found in all 14 cases with embolisation and also in six cases without embolisation.

Histological examination of emboli trapped in the coronary artery or on the wall of the endocardium of the left ventricle showed that the calcific particle was covered by a layer of collagen and elastic fibres as a result of intimal or endocardial reaction, and thus its surface was smooth and endothelialised (Figs 5 to 8).

Fig. 5 A small intramyocardial coronary artery is occluded by a calcium embolus. Multi-nucleated giant cells, as a foreign-body reaction, are seen on the surface. Haematoxylin and eosin $\times 200$.

Fig. 6 *A particle of calcium is trapped between the trabeculae in the left ventricular cavity. H and E $\times 100$.*

Fig. 7 *A large coronary artery contains a calcific embolus, which has caused a deep defect of the arterial wall (on the left side). The entire embolus is covered by a fibrous layer. Elastica $\times 20$.*

Fig. 8 An organised calcific coronary embolus is covered by a thick layer of collagen and elastic fibres. *Elastica* $\times 100$.

Fig. 9 Calcific embolus in the perivascular space encapsulated by fibrous tissue. *H and E* $\times 200$.

The trapped embolus can cause an arterial wall defect of varying depth. Occasionally, the embolus penetrates through the artery wall and is found perivascularly, encapsulated by fibrous tissue (Fig. 9).

Discussion

Spontaneous calcific embolisation is a rare condition. It may involve the coronary arteries,³⁻⁷ cerebral arteries,⁸ retinal arteries,⁹ kidneys,⁵ spleen,⁵ or the lower extremities.⁵ The source of calcium is usually a calcified heart valve but sometimes mitral annular sclerosis.⁸

However, this phenomenon has only been investigated in a prospective and quantitative survey by Holley *et al.*,¹⁰ who examined 165 cases of calcific aortic stenosis. In each case they examined the coronary arteries macroscopically and studied the histology of 10 samples of the left ventricular wall and ventricular septum. In 28 cases (17%) they found 32 calcific emboli in coronary arteries; 10 of these were visible macroscopically in large branches and 22 were seen on microscopy. The authors admit that, using this method, they examine only 2-3% of the mass of the heart and that they can study approximately 0.5% of the tissue from each histological block.

As far as we know, nobody has solved the problem of calcific embolisation systematically and quantitatively using x-ray examination. By our method we are able to detect calcific particles as small as 0.1 to 0.15 mm.¹

When assessing the radiographs, it is necessary to distinguish intracoronary or intraventricular calcific emboli from calcifications of a different nature. The embolic origin of the calcification could be determined unambiguously in most cases according to its location, size, density, and 'frayed' borders. When the x-ray finding was not clear, the site was examined histologically. Thus calcified coronary plaques of atheroma could be excluded, as well as calcification in subendocardial scars of the left ventricular myocardium, in the apices of papillary muscles, or in pericardial adhesions.

Calcific embolisation was seen in 14 of 31 patients (45%), and in all cases it came from a calcified and ulcerated valve. On studying the incidence and extent of embolisation, we found that it occurs slightly more frequently in association with mitral than with aortic disease. This is probably due to the fact that a calcified mitral valve is liable to become ulcerated.¹¹ With regard to aetiology, embolisation is found, most often, in post-rheumatic valve disease, less frequently in aortic disease with congenitally malformed valves, and, exceptionally and only to a small extent, in the senile type of aortic stenosis.

Surprisingly enough, intraventricular emboli were found in 23% of cases. The presence of calcific particles in the left ventricular cavity is understandable in mitral disease. In aortic disease, however, it can be explained only by regurgitation of blood flow due to an incompetent aortic valve. Intraventricular emboli were always associated with many intracoronary emboli.

As to the consequences of coronary occlusion with calcific material, we assume that it will cause myocardial ischaemia. However, it is not easy to prove this histologically. Scars, and occasionally also necroses of the left ventricular myocardium—mostly in a subendocardial location, were encountered in almost all hearts, irrespective of the presence of embolisation. The main role in the pathogenesis of these ischaemic lesions is played by coronary insufficiency in the heart with valvular disease and cardiomegaly rather than by calcific emboli. A quantitative analysis of the extent of scarring was not the purpose of this study. However, coronary calcific embolisation must be considered in the differential diagnosis of symptoms of ischaemic heart disease in patients suffering from valvular heart lesions.

References

- Steiner I, Hlava A, Procházka J. Calcific coronary embolization associated with cardiac valve replacement. Necropsy x-ray study. *Brit Heart J* 1976;**38**:816-20.
- Steiner I. Aortic valve stenosis with calcification. *Čs Patol* 1978;**14**:1-10 (in Czech).
- Hudson REB. *Cardiovascular Pathology*. London: Arnold, 1965; Vol 1:643-8; 1970; Vol 3:408-9.
- Moragues V, Bawell MB, Shrader EL. Coronary embolism: review of the literature and report of a unique case. *Circulation* 1950;**2**:434-7.
- Soulié P, Caramanian M, Morin B, Acar J, Forman J. Embolies calcaires du rétrécissement aortique calcifié. *Arch Mal Coeur* 1964;**57**:633-71.
- Soulié P, Caramanian M, Soulié J. Rétrécissement aortique calcifié; anatomie pathologique. *Arch Mal Coeur* 1969;**62**:1096-118.
- Wigle ED. Myocardial fibrosis and calcareous emboli in valvular heart disease. *Brit Heart J* 1957;**19**:539-49.
- Ridolfi RL, Hutchins GM. Spontaneous calcific emboli from calcific mitral annulus fibrosus. *Arch Pathol Lab Med* 1976;**100**:117-20.
- Pantely GA, Housman LB, DeMots H, Rahimtoola SH. Monocular blindness secondary to calcific embolization. An unusual presentation of rheumatic mitral valvular disease. *Chest* 1976;**69**:555-6.
- Holley KE, Bahn RC, McGoon DC, Mankin HT. Spontaneous calcific embolization associated with calcific aortic stenosis. *Circulation* 1963;**27**:197-202.
- Woolley ChF, Baba N, Kilman JW, Ryan JM. Thrombotic calcific mitral stenosis: morphology of the calcific mitral valve. *Circulation* 1974;**49**:1167-74.

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