Extensive Calcification of Heart and Coronary Bypass Surgery

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SUMMARY :

Extensive calcification of heart is a rare condition and can be a difficult problem during open heart surgery. We report a patient with this condition who required coronary bypass surgery. The inherent problems during surgery, the final outcome and the possible aetiological factors for this condition are discussed.

INTRODUCTION

Calcification in the heart is often seen in diseased aortic or mitral valves (1, 2). uncommonly it is observed in diseased coronary arteries or in the fibrotic wall of cardiac aneurysms or infarcts (3–5). Extensive cardiac wall calcification is unusual and therefore rarely suspected before cardiac surgery. We report such a case confirmed at surgery describing the problems posed.

CASE HISTORY :

A 66 year old Pakistani, suffered from typical angina pectoris for the last 10 years, which in recent months had deteriorated to class-III (NYHA). Twenty years earlier he had an acute myocardial infarction with an apparently uncomplicated recovery. No additional history of any significant illness in the past was available. The patient had been taking Propranolol, Persantin and Sulfinpyrazone for control of his anginal symptoms.

Cardiac catheterization and angiography was performed which showed a normal sized heart with mildly impaired function and triple vessel coronary artery disease. Left anterior descending coronary artery (LAD) had proximal, critical stenosis, the circumflex artery was totally occluded with narrow marginal branches and the right coronary artery showed multiple critical stenoses. As the films were of relatively low contrast, opacities in the cardiac areas suggestive of calcification were partially masked and their importance underscored.

The patient was referred for coronary revascularization surgery. Routine haematological and blood chemistry were reported to be normal. In particular, blood tests assessing renal and hepatic functions and serum calcium and phosphate levels were normal (values were B. Urea 5.9 mmol /L, S. Creatinine 37 umol/L, S. Bilirubin 1 mmol /L, S. Alkaline Phosphatase 4.2 u/L, S. Protein 70 gm/L, A/G 1.5, S. Fibrinogen 2.5 g/L, Prothrombin time normal. S. Calcium 2.35 mmol/L S. Phosphate 1.18 mmol/L). ECG showed sinus rhythm with a QRS of – 30°, without any abnormality. Chest X-ray was reported as showing a normal sized cardiac shadow, mediastinum and lung fields. There was a rim of opacity in the region of the ventricles which on lateral view was thought likely to be pericardial (fig. I, II).

The patient underwent coronary revascularisation surgery under cardiopulmonary bypass using single aortic and venous cannulae, and employing intermittent ischaemia and electrical fibrillation at a body core temperature of 32°C. At surgery the heart was found to be small, and palpably diseased in the right coronary and left

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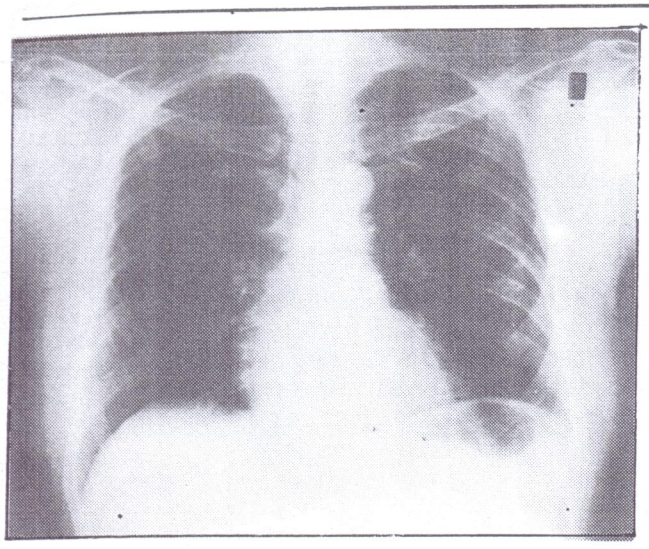

FIGURE - 1.

Chest X-ray in P. A. view showing calcification in the cardiac area.

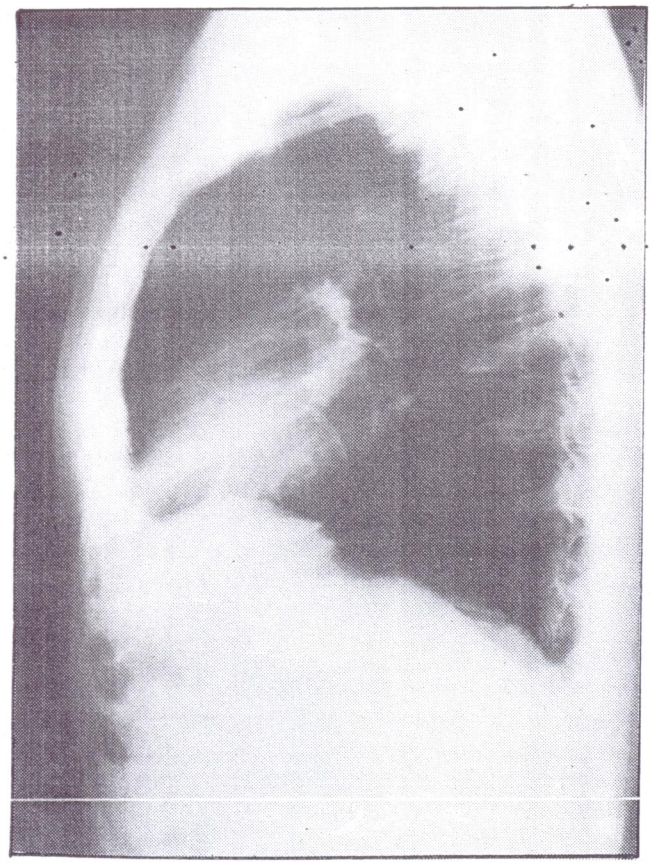

FIGURE – 2.

Chest X-ray in lateral view showing cardiac calification.

anterior descending arteries. The pericardium was free from calcification. On bypass, gentle upward lifting of the heart (which was difficult and tended to occlude venous return) the whole of the postero-basal area of the heart, mainly on the left ventricular apsect, was non-indentable and rock hard. Saphenous vein bypass grafts were easily constructed to the right and the left anterior descending coronary arteries but was not possible to the circumflex system. An attempt to expose the obtuse marginal artery was abandoned as it was tunnelling deep through the chunk of calcific mass. At the end of the procedure the patient was taken off bypass with mild inotropic support. The post operative course was satisfactory and free of complication. The follow up to date (9 months) shows the patient to have a symptom-free life without antianginal drugs.

DISCUSSION

Calcification in the heart was first described by Morgagni (6) and noted radiologically by Scholz (7). Many pathological studies exist describing the nature of calcific lesions in the heart (8–11). Degenerate cardiac valves and diseased coronary arteries are commonly responsible for radiologically observed localised cardiac calcifications. In contrast to the myocardium, it appears that the fibrous skeleton of the heart has increased susceptibility to calcium deposition (12). Fibrotic ventricular aneurysms and old myocardial infarct scars are also recognised sites of cardiac calcification (3,5). Rarely calcification has been observed in mural thrombi and in cardiac myxomas (13). Extensive cardiac calcification is rare, and most reports are necropsy studies or chance findings (14, 16).

Clasically cardiac calcification has been subdivided into two broad categories – metastatic and dystrophic (3). A third idiopathic group has also been included (17–18). Dystrophic cardiac calcification is by far the commonest group, and as opposed to the metastatic variety, occurs in the absence of any disturbance in the calcium metabolism. The patient described in this report, most likely is an example of extensive, dystrophic calcification occurring in an old myocardial infarct. The long gap of 20 years between the acute episode and the surgery could possibly explain the severity of calcification, though this is purely speculative. In the absence of obvious features of diseases producing hypercalcemic

states, it is difficult to explain such extensive lesions. Brean (19) on the basis of study of 14 cases, concluded that calcification was most likely after a large infarct, in patients who survived six years or more.

Review of literature indicates many aetiological factors, that can accelerate or modify the process of calcification. Local tissue alkalinity, citric acid — calcium binding (20), alkaline phosphates (21), acid mucopolysaccharides (22), have all been ascribed important roles. Systemic factors like hypokalaemia (23), renal failure and haemodialysis (24, 25), toxic or septic factors (26) cardiac surgery (27), and radiation therapy (28) have all been incriminated. Exact aetiology however is often difficult to determine.

From the surgical point-of-view, grafting of coronary arteries embedded in calcific mass may be impossible. Such patients should be categorised in the high risk group.

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