Uptake of Tc-99m Diphosphonate in a Massively Calcified Mitral Annulus: Case Report

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In a patient with no recent myocardial insult, uptake of technetium-99m diphosphonate was noted in a region corresponding to massive calcification of the mitral annulus. The similarity of this finding to pathologic myocardial uptake is a potential source of error in the interpretation of myocardial scintigrams.

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Since the initial reports of technetium-99m stannous pyrophosphate uptake in experimentally produced myocardial infarctions in dogs (*1*), as well as in naturally occurring infarctions in patients (*2*), other conditions have been shown to cause similar cardiac uptake. An unexplained accumulation of pyrophosphate was noted in the cardiac region of two patients with malignant tumors unrelated to the heart (*3*). Unstable arteriosclerotic heart disease, myocardiopathies (*4*), and myocardial contusions may show diffuse cardiac-uptake patterns (*5*). More recently, pyrophosphate uptake was reported in cases of left ventricular aneurysm (*6*) and a single uremic individual with secondary hyperparathyroidism showed myocardial accumulation of diphosphonate in a bone scan (*7*). The present report describes the localization of Tc-99m diphosphonate in a massively calcified mitral annulus that has not, to my knowledge, been previously documented.

CASE REPORT

A 65-year-old woman was admitted to the medical center for evaluation of transient left hemiparesis. She had suffered a myocardial infarction complicated by Dressler's postmyocardial infarction syndrome 10 years earlier. Massive calcification of the mitral annulus with extension into the myocardium was noted at that time. There was no history of rheumatic fever.

At the time of the present admission, a grade 2/6 apical systolic murmur was heard and atrial fibrillation with varying ventricular response was noted on electrocardiogram. The serum CPK, SGOT, SGPT, and LDH remained normal throughout the hospital course and there was no electrocardiographic indication of recent myocardial damage. Serum creatinine, BUN, calcium, and alkaline phosphatase were all normal. No signs of sepsis were noted at any time, and the EKG remained unchanged during the hospital stay. The neurologic findings cleared rapidly and were attributed to vascular occlusive disease.

Radiographs of the chest showed no change in the previously noted calcifications (Fig. 1). Scintigrams of the cardiac region in anterior and left lateral projections were obtained on a scintillation camera with high-resolution collimator, 3 hr after injection of 15 mCi of Tc-99m diphosphonate (Fig. 2). They showed uptake corresponding to the calcification in the mitral annulus and its myocardial extension.

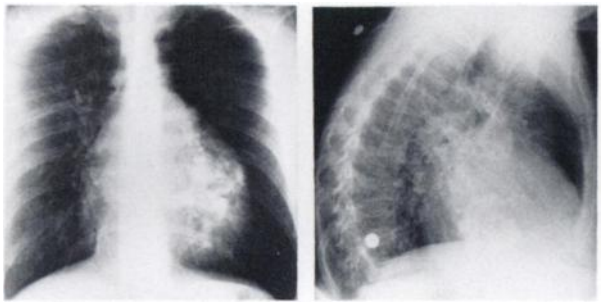

FIG. 1. Chest x-rays show massive calcification of mitral annulus with extension into myocardium.

DISCUSSION

Calcification of the annulus fibrosus of the mitral valve is generally considered a degenerative process unassociated with rheumatic fever, and is found at

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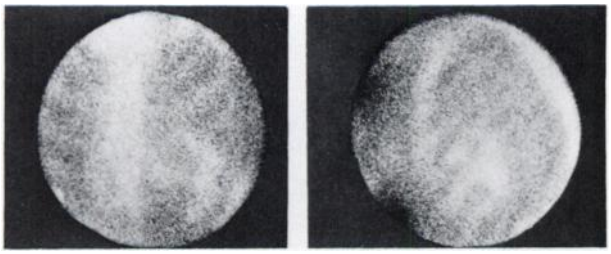

FIG. 2. Anterior and left lateral scintigrams of cardiac region show diphosphonate uptake corresponding to the calcification seen on x-ray.

autopsy in some 10% of patients, though electron-microscopy studies indicate that some calcification is present in the majority of patients over the age of 40 (*8*). Conduction disturbances are frequently associated with this condition and are attributed to the commonly found extension of the calcifications into the septum (*9*). A clinicopathologic study of 14 cases of massive calcification of the mitral annulus, together with an historic review of the subject, has been published by Korn et al. (*8*).

The uptake of bone-seeking radionuclides by damaged myocardium has been ascribed to the cellular influx of calcium with localization in the mitochondria near the hydroxyapatite crystal; thus it would be analogous to the hydroxyapatite adsorption thought to underlie the affinity of these agents for bone (*1*). The belief that a similar mechanism is operative in our case is supported by the finding that calcific foci noted by light microscopy in the mitral annulus at about 40 years of age are identifiable as apatite by x-ray diffraction (*8*). It would appear that despite the lack of apparent change in the appearance of the calcifications in our patient over a 10-yr period, the continued metabolic activity is sufficient to concentrate bone-seeking radionuclides. The potential for the erroneous diagnosis of myocardial infarction in patients with such findings is clear, and may be obviated by the correlation of radiographic and scintigraphic findings.

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