The presence of calcification in the left atrium is a relatively common finding in the setting of long-standing rheumatic valve disease, first described by Oppenheimer in 1912 [1]. It is thought to be promoted by the action of chronic strain forces on atrial walls and repeated attacks of endocarditis, but the condition does not always evolve in the same way and the interatrial septum is not usually involved (1, 2).

In many patients, the condition is a not insignificant problem. It increases the difficulty of surgical procedures such as suturing the heavily calcified atrial walls or mobilizing the mitral valve plane. Medically, the characteristics of these patients are rheumatic mitral disease of long duration, the presence of atrial fibrillation and considerable left atrial enlargement, as well as the
prevalence of dyspneic symptoms. According to a previous report, most patients with left atrial wall calcification had undergone previous surgery for mitral valve disease (2). Previous reports did not, however, evaluate CT findings, focusing instead on surgical viewpoints and simple radiographic findings. The aim of this study was to evaluate the CT findings and clinical significance of left atrial wall calcification in patients who had undergone mitral valve replacement.

Materials and Methods

Between February 1998 and December 1999, the unenhanced chest CT scans of 36 consecutive patients (17 males and 19 females) aged from 29 to 74 (mean, 49) years who had undergone mitral valve replacement were retrospectively reviewed. All were admitted for congestive heart failure thought to be the result of preoperative prosthetic valve failure. The original purpose of unenhanced CT was the evaluation of retrosternal adhesion for prior to mitral valve replacement. Chest CT scans were obtained with a Somatom plus-4 (Siemens, Erlangen, Germany) or HiSpeed unit (General Electric Medical Systems, Milwaukee, Wis., U.S.A.) set for the scanning of 7 or 8-mm-thick contiguous axial sections from the thoracic inlet through the lung base. Intravenous contrast material was not used. The CT scans were jointly analyzed in one session by two chest radiologists, who reached their decisions by consensus. Among a total of 36 patients, unenhanced CT revealed left atrial calcification in 15. They were divided into two groups according to whether or not left atrial wall calcification was present. The cause of mitral valve disease, type of previous surgery, and the CT findings were investigated. In addition, the diameter of the left main pulmonary artery at CT, the time interval between previous surgery and CT, and pulmonary arterial pressure were compared between patients with and without left atrial wall calcification. To discover the cause of mitral valve disease, the type of surgery, the time interval between previous surgery and CT scanning, and pulmonary arterial pressure, the patients’ medical records were referred to. CT was used to evaluate the pattern of calcification, the size of the main pulmonary artery, and associated findings such as the presence of a left atrial thrombus.

The pattern of calcification was categorized as focal or diffuse. Focal calcifications was classified as either nodular or linear, according to its shape, the linear pattern being defined as shell-like calcification that lined the inner wall of the left atrium, while diffuse calcification was defined as that which extended over at least one-quarters of the left atrial wall. In 15 patients, systolic pulmonary arterial pressure was determined from the results of cardiac catheterization for the preoperative evaluation of heart function, and in the remaining 21 it was estimated from TR velocity, measured using transthoracic echocardiography (3).

The diameter of the left main pulmonary artery was measured at its widest point perpendicular to its long axis, and also at a right angle to its long axis, lateral to the ascending aorta, and at the level of its bifurcation (4). The pathological findings were reviewed after endoatricectomy of the left atrium.

Differences in systolic pulmonary arterial pressure, the diameter of the left main pulmonary artery, and the time interval between previous surgery and CT scanning between patients with left atrial wall calcification and those without calcification were determined using student’s t test.

Results

The cause of the mitral valve disease was either rheumatic valve disease (n = 35) or Behcet’s disease (n = 1). All patients had undergone previous valvular surgery, namely valvuloplasty (n = 1) or valve replacement (n = 35); mitral valve replacement was performed in 24, and dual replacement in 12 (mitral and aortic valvuloplasty, n = 11; mitral and tricuspid valvuloplasty, n = 1).

Among 15 patients with left atrial calcification, the pattern of this was focal in 12 (linear, n = 7; nodular, n = 5), and diffuse in three (Figs. 1-3). In two of the 15, a left atrial thrombus with subtle amorphous calcification was also found (Figs. 1, 3).

Systolic pulmonary arterial pressure ranged from 37 to 97 (mean, 62) mmHg, and from 26 to 82 (mean, 47) mmHg respectively, in patients with and without left atrial wall calcification. Pressure was significantly higher in patients with left atrial calcification than in those without (p < 0.05).

The diameter of the left main pulmonary artery ranged from 22.5 to 36.3 (mean, 22.8) mm in patients with calcification, and from 17.4 to 30 (mean, 21.6) mm.
A 46-year-old man presented with dyspnea. He had undergone a mitral valve commissurotomy 14 years prior. 

A. B. Posteroanterior (A) and lateral (B) chest radiographs show a curvilinear calcification of dilated left atrium (arrows). 

C. Unenhanced CT scan shows a nodular calcification of the left atrial appendage (arrow). Low attenuated left atrial thrombus is also noted (open arrow). 

D. Curvilinear calcification is observed in the free border of the left atrium (arrows). Note the left atrial thrombus (open arrows). 

E. Note the left atrial thrombus with a focal high attenuated area which is suggestive of calcification (open arrows). Curvilinear calcification is seen (arrows).
in those without. The mean interval between previous surgery and CT scanning was 16 and 14 years respectively, in patients with and without left atrial calcification. For neither arterial diameter nor mean interval was the difference between patients with and without calcification significant.

Six patients underwent endoatriectomy. Pathologic examination revealed plate-like dystrophic calcification in all six and associated bland mural thrombi in two.

**Discussion**

A number of cases of left atrial calcification, even at uncommon site such as the atrial appendage, have been described in the literature (5). Most have been reported as unusual conditions or incidental radiographic findings.

Calcification of the left atrium is an uncommon com-
plication of longstanding valvular rheumatic disease and calcification of the atrial endocardium is even more uncommon. It has been suggested that calcification of the left atrium occurs in response to the chronic strain forces present in mitral disease. Gedgaudas et al. (6) suggested that overstretching of the left atrial wall secondary to severe mitral stenosis is the primary cause of the lesion, but in some patients there is no overstretching, as in those with a small calcified left atrium and a giant right atrium. Roberts et al. (7) reported that calcific deposits prevented the left atrium from dilating, decreased its compliance, and caused the elevated left atrial pressure to be transmitted to the pulmonary vessels and right ventricle, leading to tricuspid insufficiency. Another theory suggests that repeated endocarditis and ulceration of the atrial wall lead to calcification: one of the principal factors may be the turbulence resulting from valvular alteration (1, 2).

According to an earlier report (2), most patients with left atrial calcification had undergone previous surgery for their mitral valvular disease. As the number of patients undergoing cardiac valve replacement has grown, subsequent valve surgery has become increasingly frequent. The newer generations of mechanical valves are far more efficient and less prone to structural failure than older ones, but other valve and non-valve-related complications still constitute a major cause of morbidity and mortality. Bioprostheses, on the other hand, implanted in large numbers in the 1970’s and early 1980’s, have now entered their second decade of life since implantation, and biodegradation is becoming more frequent (8).

In this study, all patients were admitted due to congestive heart failure thought to be a result of prosthetic valve failure prior to further surgery involving the prosthetic valve, and all showed significantly increased systolic pulmonary arterial pressure. This indicates that although pulmonary arterial pressure was lower during short-term follow-up after valve replacement, hemodynamic change leading to increased pressure does occur.

Fig. 3. A 58-year-old man who had undergone mitral valve replacement due to mitral stenosisinsufficiency.
A. Lateral image shows a curvilinear calcification of a dilated left atrium [arrows].
B. Unenhanced chest CT shows a nodular calcification of the left atrial appendage [arrow]. Note that the subtle low attenuated thrombus contains a high-attenuated area [open arrows].
C. Curvilinear calcification is observed along the free border of the left atrium [arrows].
Factors which relate to hemodynamic change may include the surgical technique employed, postoperative treatment, biodegradation of the bioprosthesis, or defective mechanical valves. In this study, systolic pulmonary arterial pressure in patients with left atrial calcification was significantly higher than that in those without.

The time interval between initial and subsequent prosthetic valve surgery may also be an important factor associated with increased pulmonary arterial pressure and the formation of left atrial calcification (2). However, this study demonstrated no significant difference between the two groups.

Calcification of the left atrial wall is frequently associated with adherent mural thrombi, which on occasion may also calcify. The two types of calcification may be differentiated by the fact that intramural calcification is linear, nonlaminated, and marginal in distribution, whereas calcified thrombi are nonlinear and laminated [9]. Vickers et al. [10] suggested that calcification of the left atrial appendage may be confused with a calcified mural thrombus, and doubted that X-rays can differentiate between the two. Gedgaudas et al. [6] expressed similar doubt and reported that a histologically proven calcified thrombus is unusual (3 of 26 in their series). Van de Sande et al. [11] advocated the use of angiocardiography in searching for intra-atrial thrombi prior to surgery; left atrial thrombi, containing calcification and easily detected at CT, were observed in two of our patients. Mural thrombi, on the other hand, were depicted by CT as low-attenuating lesions containing an area of high attenuation.

Since calcification of the left atrial wall will usually indicate thickening, fibrosis, and difficulty in entering the left atrium, it is important that the surgeon is aware of its presence prior to surgery. Hemorrhage from friable tissues and the likelihood of mural thrombi are also problems that a surgeon should be alerted to if left atrial calcification is noted.

In conclusion, left atrial wall calcification is a common finding in patients who have undergone mitral valve replacement, particularly in those with high pulmonary arterial pressure.

References

1. \[ \text{연구 방법:} \]

   1.1. \[ \text{연구 대상:} \]

   1.2. \[ \text{연구 방법:} \]

   2. \[ \text{결과:} \]

   3. \[ \text{의의:} \]

   4. \[ \text{결론:} \]

   5. \[ \text{자료 및 방법:} \]

   6. \[ \text{결과:} \]