Calcified amorphous tumor (CAT) is a rare non-neoplastic cardiac mass characterized by nodular calcification in an amorphous background of fibrin with focal inflammation and degenerating thrombus.\textsuperscript{1,2} Although some cases of successful surgical excision have been reported,\textsuperscript{1} little is known about the precise mechanism of disease progression. Histology indicated the presence of chronic inflammation in the CAT in some studies,\textsuperscript{2} but the time course of changes in

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**Figure 1.** (A) Computed tomography or (B) \(^{18}\)F-fluoro-2-deoxyglucose (FDG) positron emission tomography (PET) of a calcified mass of the left ventricle. (C, D) Changes in \(^{18}\)F-FDG PET (sagittal section) between (C) baseline and (D) 6 months later. Standardized uptake values (SUV) are changed from (C) to (D) as follows: max/average SUV, 8.32/4.32 to 6.50/3.74 (apex), 5.90/3.70 to 4.27/2.97 (lateral wall) and 3.93/3.04 to 3.19/2.72 (septal wall), respectively.
chronic inflammation and even in morphology in CAT, remain unknown. Here, we evaluated the time course changes of the inflammatory findings on 18F-fluoro-2-deoxyglucose (FDG) positron emission tomography (PET). We also performed histological analysis of CAT to evaluate the pro-fibrotic changes of the tumor. Thus, we investigated the disease mechanism of CAT in the clinical setting.

A 67-year-old woman presented to hospital with shortness of breath. She had a 10-year history of exertional dyspnea. A mobile tumor (approximate diameter, 10 mm) with calcification in the left ventricle (LV) had been identified 5 years previously at a health check-up. To avoid embolism, the patient underwent resection of the mobile cardiac mass. Two years after the surgery, she visited our hospital because of exertional dyspnea. Blood pressure, heart rate, and body temperature were 122/82 mmHg, 76 beats/min, and 36.6°C, respectively. No murmur was detected on physical examination. Echocardiography showed reduced left ventricular systolic function with an ejection fraction of 40% and normal diastolic pattern. On laboratory analysis, serum parathyroid hormone, calcium, prothrombin time, active partial thromboplastin time, creatinine, and C-reactive protein were within normal limits. Elevated plasma brain natriuretic peptide (BNP) was observed (317.5 pg/ml). Computed tomography (CT) showed an amorphous calcified mass on the endocardium of the LV (Figure 1A). We compared these findings with those on CT obtained 3 years previously at the other hospital, and found no obvious change in size of the cardiac mass. FDG-PET showed a higher intensity region corresponding to the region of the calcified mass identified on CT (Figure 1B) using heparin loading to reduce the physiological 18F-FDG uptake by the normal myocardium. Coronary angiography showed no significant abnormalities in the coronary arteries. Biopsy of the right ventricle (RV) was performed to exclude secondary cardiomyopathies, and pathology indicated moderate hypertrophy of cardiomyocytes and perivascular fibrosis, but no calcification or inflammation in the endocardium of the RV (Figures 2A, B). Figure 2C shows the excised tissue from the mobile mass. Hematoxylin and eosin staining showed moderate hypertrophy of cardiac myocytes and fine granular staining around the large nodule, indicating calcification in the excised tissue. (E) Osteopontin-positive cells around the organized nodule. (F) Accumulation of CD68-positive cells around the calcified nodule.

Figure 2. (A) Hematoxylin and eosin (HE) and (B) Masson trichrome staining showing moderate hypertrophy of cardiomyocytes and perivascular fibrosis, but no calcification or inflammation in the endocardium of the right ventricle. (C) Excised tissue from the mobile mass. (D) HE staining showed moderate hypertrophy of cardiac myocytes and fine granular staining around the large nodule, indicating calcification in the excised tissue. (E) Osteopontin-positive cells around the organized nodule. (F) Accumulation of CD68-positive cells around the calcified nodule.
18F-FDG PET. Intriguingly, 18F-FDG PET showed inflammation in the myocardium around the calcified mass. We also confirmed that there were no OPN-positive cells in the RV. A possible explanation for this is that the calcified tumor may have resulted from organized change of an extrinsic mass from the myocardium, such as old thrombus, with subsequent development of mild-moderate inflammation in the mass and the myocardium around the tumor. Similarly, a 43-year-old male patient was reported to have hypertrophic cardiomyopathy with a calcified LV thrombus,

6 and a 58-year-old woman had an inflammatory myofibroblastic tumor. On the basis of pathology and CT/PET, we diagnosed the present patient with CAT in the LV. We recommended surgery to remove the tumor from the LV because of systolic dysfunction and high levels of plasma BNP. Follow-up 18 F-FDG PET was done 6 months later. Sagittal because of systolic dysfunction and high levels of plasma BNP. We also started treatment using β-blockers because of systolic dysfunction and high levels of plasma BNP. Follow-up 18 F-FDG PET was done 6 months later. Sagittal sections showed the decreasing FDG uptake (Figure 1C, baseline; Figure 1D, 6 months later). On quantitative analysis the intensity was decreased after treatment (Figures 1C,D), suggesting that the inflammation had improved in parallel with β-blocker treatment. There was no elevated intensity in the other organs and no remarkable inflammatory changes were detected on routine blood examination, indicating that inflammation was cardiac specific. The mechanism of the changes on 18F-FDG PET were not completely determined. A previous experimental study showed that carvedilol suppressed leukocytosis in cardiac interstitial fluid in a canine chronic ischemic cardiomyopathy model, suggesting the anti-inflammatory effects of carvedilol. Another human study also suggested the anti-oxidative effects of carvedilol. On the basis of these reports, we suggest that carvedilol treatment can contribute to the improvement of cardiac-specific inflammation, reflected by the decreasing FDG uptake.

We observed the size of the cardiac mass for 2.5 years with no obvious changes, and, to the best of our knowledge, this is the first study on the time course changes of cardiac-specific inflammation, using both 18F-FDG PET and histology, in a patient with CAT. We also observed pro-fibrotic changes in the CAT, suggesting that chronic inflammation and pro-fibrotic processes might play an important role in disease progression. Further studies are necessary to confirm the relationship between inflammation and disease progression in CAT. Consequently, the underlying mechanism behind the progression of CAT will be clarified.

References

Supplementary Files

Movie S1. Transesophageal echocardiography showing calcified amorphous tumor in the left ventricle.

Movie S2. Transesophageal echocardiography showing calcified amorphous tumor in the left ventricle.

Please find supplementary file(s); http://dx.doi.org/10.1253/circj.CJ-15-0136