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Case Report

Cardiac calcified amorphous tumor as a potential cause of cerebral infarction: A clinical case report ☆

Hyunjin Kim, MD[a,*], *Shotaro Kanao, MD, PhD*[a], *Satoshi Noma, MD, PhD*[a], *Sumika Yasumura, MD*[b], *Shinji Sumiyoshi, MD, PhD*[c], *Manabu Morishima, MD, PhD*[d], *Takeshi Kubo, MD, PhD*[a]

[a] *Department of Radiology, Tenri Hospital, Nara, Japan*
[b] *Diagnostic Imaging and Nuclear Medicine, Kyoto University, Kyoto, Japan*
[c] *Department of Diagnostic Pathology, Tenri Hospital, Nara, Japan*
[d] *Department of Cardiovascular Surgery, Kitano Hospital, Osaka, Japan*

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ABSTRACT

We report the case of a 62-year-old male on long-term hemodialysis who was admitted to our hospital due to acute cerebral infarction associated with a cardiac calcified amorphous tumor (CAT). The patient presented with recurrent episodes of syncope and retrograde amnesia. Brain MRI identified multiple acute cerebral infarctions, while transthoracic echocardiography (TTE) revealed a 2.5 cm echogenic mobile mass attached to the ventricular side of the posterior mitral leaflet. The patient underwent surgical resection of the mass. Pathological examination confirmed the diagnosis of a CAT. A chest computed tomography (CT), performed incidentally for pneumonia 6 months prior, revealed extensive calcifications in the mass. Postinfarction imaging showed a reduction in calcifications within the mass, suggesting a potential link between the infarction and changes in the cardiac lesion.

Introduction

Cardiac calcified amorphous tumors (CATs) are rare, non-neoplastic masses first described by Reynolds et al. in 1997 [1]. Histologically, they consist of nodular calcifications within an amorphous fibrinous matrix, often accompanied by chronic inflammation and degenerating thrombi. CATs can occur in any cardiac chamber or on any valve and are frequently discovered incidentally during imaging studies [2].

While often asymptomatic, CATs pose a risk for serious embolic events due to their potential to fragment, leading to strokes or systemic embolization [3]. This risk is particularly significant in patients with chronic renal failure undergoing long-term hemodialysis, where abnormal calcium-phosphorus metabolism may contribute to CAT

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* Corresponding author.
E-mail address: panda35493549@gmail.com (H. Kim).

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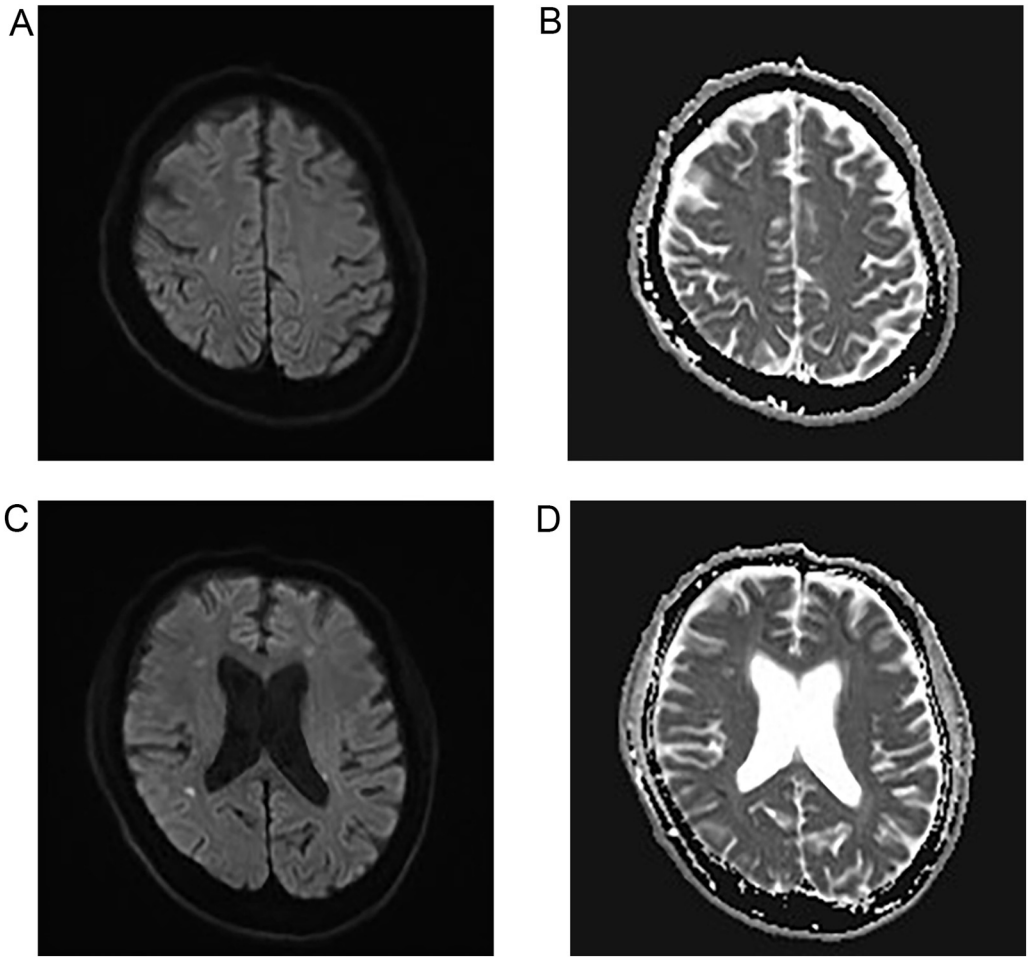

Fig. 1 – Brain MRI shows acute cerebral infarctions scattered throughout the cortex (A-D).

development [4]. Advanced imaging modalities like echocardiography, CT, and magnetic resonance imaging (MRI) are crucial for detecting and characterizing CATs, aiding in differentiation from other cardiac masses [5–7].

We present the case of a 62-year-old man on long-term hemodialysis who experienced acute cerebral infarctions associated with a CAT attached to the posterior mitral leaflet. Notably, imaging showed a reduction in calcifications within the tumor following the cerebral infarction, suggesting embolization of calcified fragments. This case underscores the importance of considering CATs in the differential diagnosis of embolic strokes in patients with chronic renal failure and highlights the role of multimodal imaging in guiding timely surgical intervention to prevent further embolic events.

Case presentation

A 62-year-old male was referred to our hospital after recently reporting multiple episodes of syncope and retrograde amnesia to his previous physician. Upon arrival at our facility, he was asymptomatic, fully alert (Japan Coma Scale 0), and able to communicate effectively. His medical history includes hypertension, type 2 diabetes mellitus, dyslipidemia, peripheral arterial occlusive disease, bilateral internal carotid artery stenosis, and chronic renal failure requiring hemodialysis for the past 11 years. He had undergone coronary artery bypass graft surgery 5 years earlier. The patient's family history revealed no significant findings.

The patient's vital signs were stable, with a blood pressure of 107/80 mmHg, heart rate of 72 beats per minute, and oxygen saturation of 98% on room air. Cardiovascular and respiratory examinations were unremarkable, and neurological examination revealed no focal deficits. His current medications included antiplatelet agents (clopidogrel), antihypertensives (amlodipine, valsartan), a statin (rosuvastatin), and an antidiabetic agent (linagliptin).

Laboratory tests showed mild anemia (hemoglobin 9.6 g/dL, hematocrit 29.0%) and renal dysfunction (blood urea nitrogen 45.5 mg/dL, creatinine 13.9 mg/dL). Other laboratory values, including white blood cell count, platelet count, electrolytes, C-reactive protein, and coagulation studies (prothrombin time 11.3 seconds, international normalized ratio 0.97, activated partial thromboplastin time 30.5 seconds), were within normal limits.

Given the patient's significant medical history and despite being asymptomatic upon admission, a brain MRI was

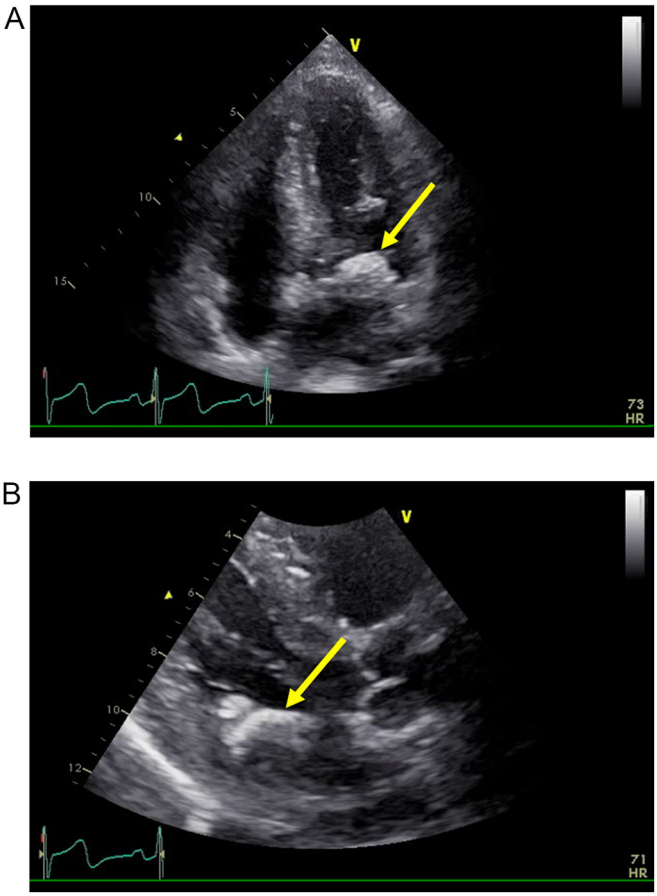

Fig. 2 – TTE shows a mobile mass measuring 2.5 cm in diameter attached to the mitral valve.

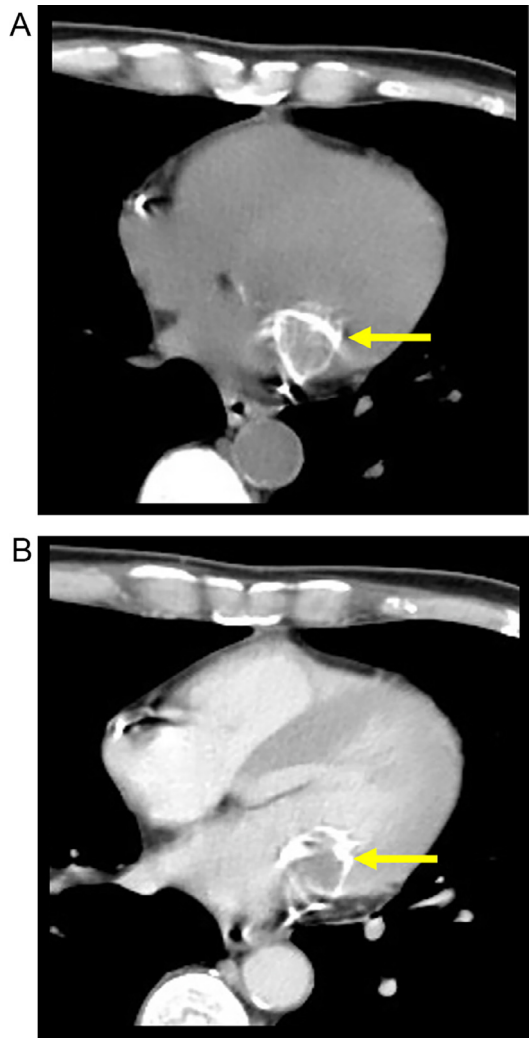

Fig. 3 – Preoperative contrast CT shows a mass attached to the mitral valve with marginal calcification (A), with no significant contrast enhancement (B).

conducted to exclude a cerebrovascular event. The MRI revealed multiple small subcortical infarctions in both cerebral hemispheres on diffusion-weighted imaging (DWI) and corresponding hypointense areas on apparent diffusion coefficient (ADC) maps, consistent with acute cerebral infarctions (Fig. 1A-D).

To assess for a potential cardiac source of emboli, an ECG and a TTE were performed. The ECG showed normal sinus rhythm with no ST segment changes, indicating no arrhythmias. The TTE identified a 2.5 cm highly echogenic mass attached to the ventricular side of the posterior mitral leaflet, moving synchronously with it (Fig. 2). The characteristics of the mass raised suspicion of a CAT, and the cardiovascular surgery team was consulted.

Given the recent cerebral infarctions and the embolic potential of the mass, especially due to its mobility and valvular attachment, surgical intervention was recommended to prevent further embolic events. Over the subsequent 9 days, detailed imaging studies, including CT and MRI, were conducted as part of the preoperative evaluation. During this period, clopidogrel was discontinued, and anticoagulation was managed with heparin to optimize surgical conditions, adjusted 1 week prior to surgery. Meanwhile, the patient remained under close monitoring in the neurology ward throughout the entire preoperative phase.

Specifically, contrast-enhanced CT and cardiac MRI were performed to further characterize the mass and rule out additional abnormalities. The contrast-enhanced CT showed a mass with marginal calcification attached to the mitral valve without significant contrast enhancement (Fig. 3A and B). Cardiac MRI revealed the mass to be isointense relative to the myocardium on T1-weighted images and fat-suppressed T2-weighted images (Fig. 4). On gradient-echo cine sequences, the mass displayed firm attachment to the ventricular side of the posterior mitral leaflet with mobility (Fig. 5), and no other abnormalities were identified.

Surgical resection of the mitral valve mass was performed via median sternotomy 9 days after discovery of the mass. Intraoperatively, a 3 cm mass was found on the ventricular side of the posterior mitral leaflet, appearing either attached to or integrated with the leaflet surface. Upon incising the posterior leaflet, grayish milky white fluid flowed from the mass, which was promptly aspirated, causing the mass to collapse. The posterior leaflet and the mass were excised en bloc

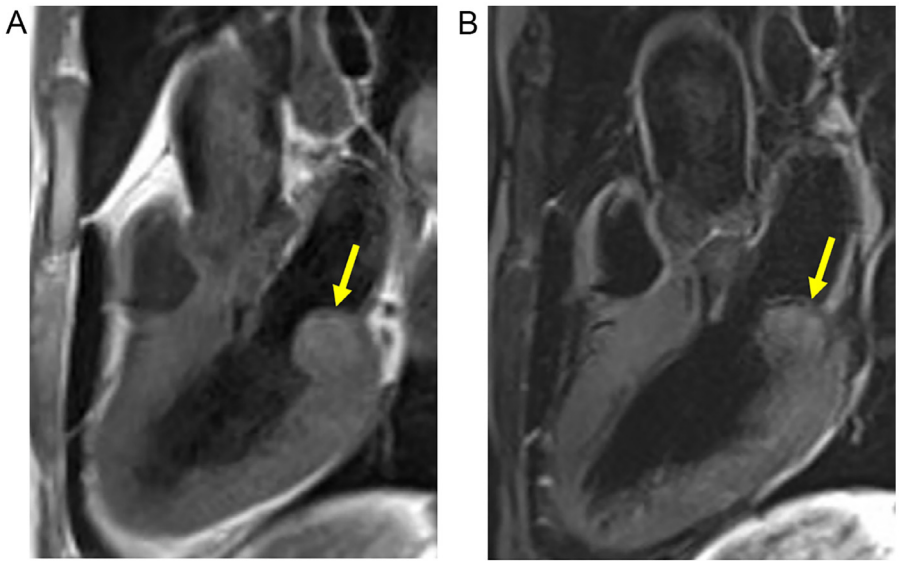

Fig. 4 – Cardiac MRI reveals that the mass has an isointense signal compared to the myocardium on T1-weighted sequences and fat-suppressed T2-weighted sequences.

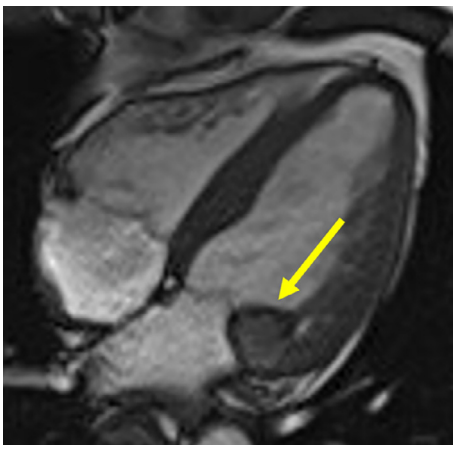

Fig. 5 – On gradient-echo cine sequences, the mass shows firm attachment to the mitral valve with mobility.

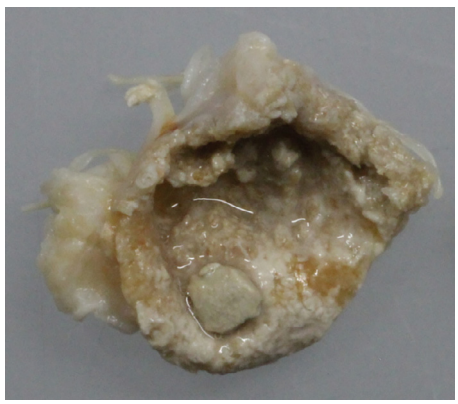

Fig. 6 – Gross appearances of the resected masses.

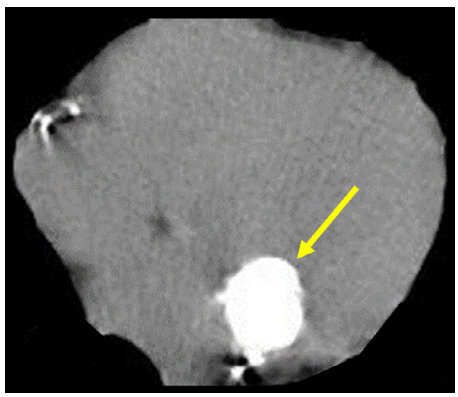

Fig. 7 – Retrospective review of a noncontrast CT scan performed 6 months earlier for pneumonia (7A) reveals prominent internal calcifications within the mass, compared to a follow-up CT scan after the cerebral infarction (7B).

(Fig. 6). Postoperative management included anticoagulation with warfarin and antiplatelet therapy with aspirin to prevent thromboembolic events. The patient recovered uneventfully and was discharged after 1 week with instructions for close outpatient follow-up.

Histopathological analysis of the excised mass revealed fibrous tissue with granular calcifications throughout the specimen, without malignant cells, confirming the diagnosis of a CAT.

A retrospective review of a noncontrast CT scan performed 6 months earlier for pneumonia revealed that the mass was already present at that time, measuring approximately the same size (Fig. 7). The lesion exhibited extensive internal calcifications, appearing as areas of high attenuation. However, in the imaging studies following the cerebral infarction, these

internal calcifications were notably absent, despite the overall size of the mass remaining unchanged (Fig. 3A).

At 6 months post-surgery, follow-up brain MRI showed no new ischemic events, and at twelve months, echocardiography revealed no evidence of CAT recurrence. Throughout the follow-up period, the patient remained free of neurological or cardiac symptoms.

Discussion

Cardiac calcified amorphous tumors (CATs) were first reported in 1997 as cardiac masses characterized by nodular calcification in an amorphous background of fibrin, accompanied by focal inflammation and a degenerating thrombus [1]. Some studies suggest that CATs represent late-phase changes in a thrombus, with abnormal calcium metabolism due to renal dysfunction and inflammation associated with hemodialysis contributing to rapid growth and pathological changes [4].

On echocardiography, CATs typically present as calcified endocavitary masses located in any cardiac chamber, on any valves, or along valvular annuli, varying in size from small punctate lesions to large masses [2]. On CT, CATs appear as partially or diffusely calcified hypodense masses with prominent calcification foci [5]. MRI shows them as homogeneous masses with low signal intensity on precontrast T1- and T2-weighted images and no enhancement post-gadolinium [6]. They may be mobile or immobile on gradient-echo cine sequences, appearing firmly attached to the ventricle [7]. The configuration and shape of cardiac CATs vary widely. In the differential diagnosis cardiac CATs, several entities should be considered, including calcified thrombi, fibromas, myxomas, primary cardiac osteosarcomas, valvular vegetation, and caseous calcification of the mitral annulus (CCMA) [8].

Patients with CATs are mostly asymptomatic at the time of diagnosis, with CATs often being incidentally discovered, particularly in older individuals [9]. While surgery was performed in the majority of cases, it is possible that asymptomatic patients managed without surgery might be underreported due to the lack of histopathological confirmation [5].

Generally, CATs are known to carry a potential risk of stroke or embolism [3]. Our case highlights this risk, as the patient experienced cerebral infarctions likely due to embolization from the CAT. The disappearance of internal calcifications in the mitral valve mass following the patient's cerebral infarction suggests that calcified fragments may have embolized, contributing to the acute cerebral infarctions. The temporal correlation between the loss of calcifications and the onset of neurological symptoms strengthens the hypothesis that the CAT served as the source of emboli leading to the cerebral events. This finding underscores the potential unstable nature of CATs and the importance of careful follow-up, particularly regarding embolic complications [10].

In conclusion, CATs are rare cardiac masses with potential for embolic events, particularly in patients with chronic renal failure on long-term hemodialysis. Our case demonstrates that changes in calcification within a CAT can correlate with embolic cerebral infarctions. This underscores the importance of vigilant monitoring and timely surgical intervention. Advanced imaging modalities play a vital role in detecting and characterizing these tumors, guiding appropriate management to mitigate risks such as stroke or other embolic events.

Patient consent

Written informed consent was obtained from the patient.

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