

Rapid daily expansion of highly mobile intracardiac calcified tissue: a case report

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Background

A calcified amorphous tumour (CAT) is a non-neoplastic mass lesion arising within the cardiac chamber. CATs are rare but are a common cause of organ embolism. In the present study, we experienced a case of an intracardiac mass with calcification that, in contrast to a typical CAT, suddenly appeared and rapidly expanded without an inflammatory response based on pathological findings.

Case summary

A 58-year-old Japanese man undergoing peritoneal haemodialysis had a high-echoic mobile mass (15 × 6 mm), which was not visible on the transthoracic echocardiography (TTE) approximately a month earlier, in the left ventricular outflow tract noted on TTE performed during a close examination for fever. Although multiple blood cultures were negative, ampicillin/sulbactam and ceftriaxone were initially administered because of suspected blood culture-negative endocarditis. The mass rapidly enlarged (22 × 5 mm) over the following days. A CAT was suspected and resected based on imaging findings with calcification; however, the pathological findings did not indicate inflammation and fibrin that are typically found in CATs. Echocardiography performed 12 months after the resection showed no recurrence.

Discussion

This intracardiac calcified tissue had several similar features to a CAT. However, the initial presentation, enlargement rate, and pathological features of the tissue differed from that of a typical CAT. Although it is unknown whether this mass is a subtype of CAT, when an intracardiac calcified tissue is detected using an imaging test, careful follow-up or early surgical resection should be considered given the possibility of rapid tissue enlargement and embolism caused by the mass.

Keywords

Calcified amorphous tumour • Calcified intracardiac mass • Daily enlargement • Pathology • Case report

ESC Curriculum

2.2 Echocardiography • 2.4 Cardiac computed tomography • 6.8 Cardiac tumours • 7.5 Cardiac surgery

Learning points

- Some intracardiac calcified tissues can undergo rapid daily expansion.
- When intracardiac calcified tissue is observed, careful follow-up or early tissue resection is necessary to prevent embolism.

Introduction

A calcified amorphous tumour (CAT) is a non-neoplastic cardiac disease.¹ CAT can be detected in any cardiac chamber.² CAT is often asymptomatic and discovered incidentally; however, it can present with dyspnoea, syncope, or symptoms of organ embolism. It varies in size and shape and

appears as calcified lesions on echocardiography or computed tomography (CT).³ Resection of a CAT is typically performed to prevent embolization.

Here, we report a case of an intracardiac mobile mass, considered a CAT based on the clinical course and echographic images but showing somewhat different pathological findings and an extremely rapid expansion rate compared to previously reported cases.

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Timeline

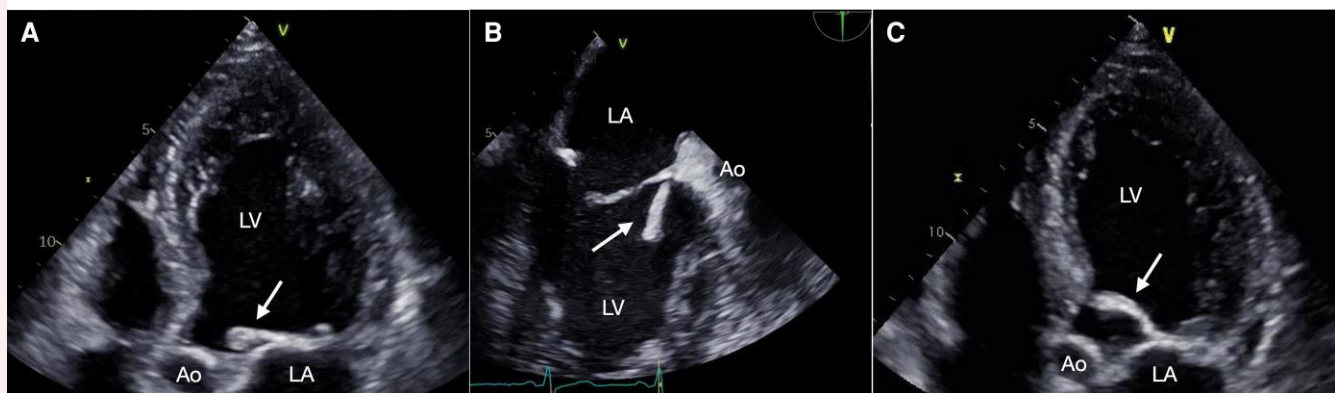
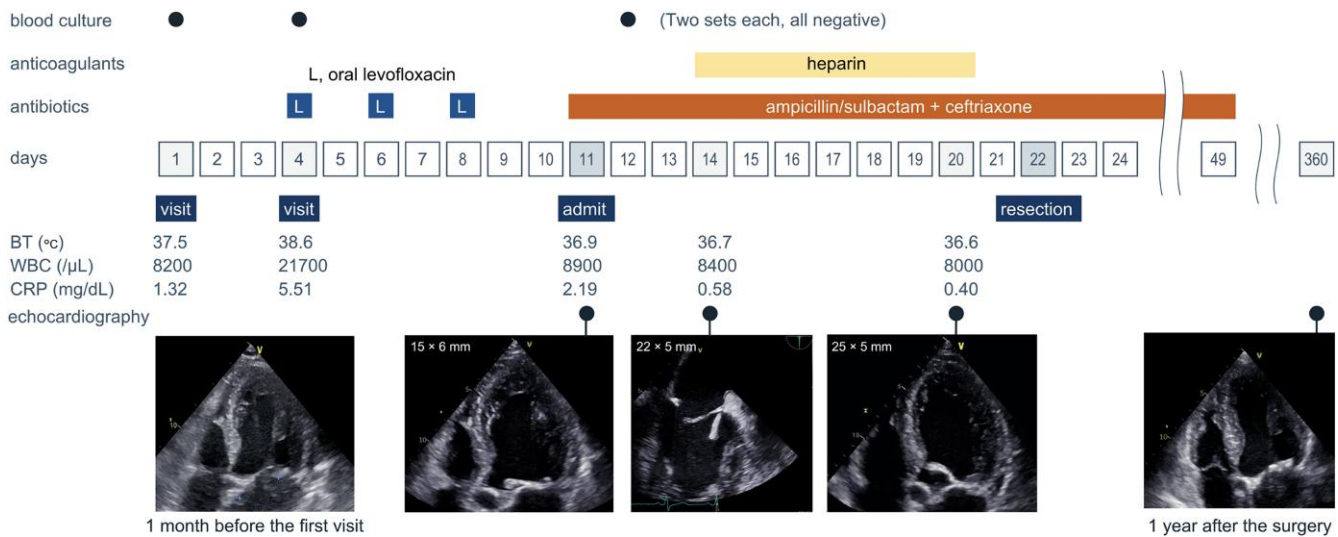


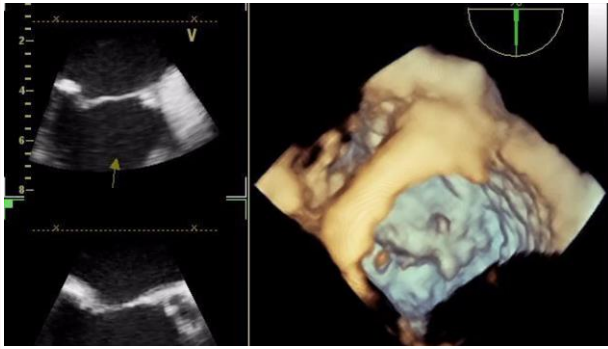
Figure 1 Serial changes of the mass (white arrow) in the left ventricular outflow tract on echocardiography (A,C) are transthoracic images, and (B) is a transesophageal echocardiographic image: (A) day 11 (15 × 6 mm); (B) day 14 (22 × 5 mm); (C) day 20 (25 × 5 mm). Ao, ascending aorta; LA, left atrium; LV, left ventricle.

Case presentation

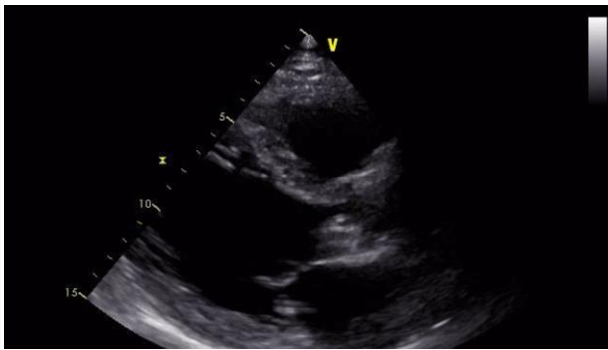
A 58-year-old male who has received peritoneal dialysis for anti-neutrophil cytoplasmic antibody (ANCA)-associated nephritis for 6 years presented to our hospital on day 1 with a complaint of low-grade fever that started 2 weeks ago. No significant physical abnormalities, including heart murmurs or skin rashes, were observed, and there were no clear signs of local infection besides a fever of 37.5°C. Blood tests revealed a slightly elevated white blood cell (WBC) count (8200/ μ L; normal range: 3300–9000/ μ L) with neutrophilia and a slight increase in C-reactive protein (CRP; 1.32 mg/dL; normal range: 0.00–0.30 mg/dL). Two sets of blood cultures and the culture from the peritoneal dialysis exit site were collected. When he revisited the hospital on day 4, his body temperature had risen to 38.6°C, with no significant physical abnormalities. WBC and CRP levels had increased further to 21 700/ μ L and 5.51 mg/dL, respectively. Procalcitonin had slightly increased to 0.32 ng/mL (normal value: \leq 0.05 ng/mL). β -D-Glucan did not increase. A plain CT scan performed on the same day did not reveal any focus of fever, and a COVID-19 antigen test using a nasopharyngeal swab



Video 1 Transthoracic echocardiography on day 11 (day of admission). A highly echogenic 15 × 6 mm mobile mass is observed in the left ventricular outflow tract.



Video 2 Transoesophageal echocardiography on day 14. The mass is now 22 × 5 mm. The clubbed mass is highly mobile, and its stem is attached to the left ventricular side near the anterior commissure of the mitral valve.



Video 3 Transthoracic echocardiography on day 20. The mass is now 25 × 5 mm. It is highly mobile, and its tip jumps through the aortic valve in systole.

was negative. Furthermore, two sets of blood cultures were collected, and oral levofloxacin therapy commenced every other day for 3 days. At hospital visitation on day 11, his fever and inflammatory response had improved. However, transthoracic echocardiography (TTE) showed a relatively high echogenic 15 × 6 mm mobile mass in the left ventricular outflow tract (*Figure 1A, Video 1*), which was not seen on the TTE conducted a month earlier as part of routine tests (see [Supplementary material online, Video S1](#)). No significant valvular diseases were observed. Four sets of blood cultures were collected before administering the antimicrobial agents, and cultures from the peritoneal dialysis exit site were negative. There were no physical findings characteristic of infective endocarditis, elevation of rheumatoid factor, or embolic findings on contrast-enhanced chest and abdominal CT. The patient was admitted with suspected blood culture-negative infective endocarditis (BCNIE) based on the TTE findings and fever. No abnormalities were noted in vital signs, chest radiography, and electrocardiography (ECG) on admission. Empirical intravenous infusions of ampicillin/sulbactam and ceftriaxone were initiated. Two sets of blood cultures collected on day 12 were also negative. There was no fever or re-elevation of the inflammatory response after admission; however, transoesophageal echocardiography (TEE) performed on day 14 showed that the mass had increased to 22 × 5 mm, and its stem was attached to the vicinity of the anterior mitral annulus (*Figure 1B, Video 2*). A CAT was strongly suspected because of the appearance of the mass. Owing to the rapid enlargement over a short period, the mass was also considered a possible thrombus, and heparin infusion was started; however, a TTE performed on day 20 showed that the structure had enlarged further to 25 × 5 mm (*Figure 1C, Video 3*). This calcified mass was also clearly recognized using ECG-gated cardiac CT on day 20, but mitral annular calcification (MAC) was mild, confirmed using CT (*Figure 2*). On day 22, a mass resection was performed for embolization prevention and diagnosis at another hospital. An aortic incision revealed a highly mobile, milky white tissue (25 × 5 mm) attached to the mitral–aortic intervalvular fibrosa (*Figure 3*), which was resected at the base of the stem. The aortic and mitral valves were normal, with no need for reconstruction after mass resection, and the MAC was not prominently visible within the surgical field. Pathologically, the tissue was diffusely calcified, and there was no evidence of neoplasia (*Figure 4*). However, unlike a typical CAT, chronic inflammation and fibrin were not prominent. The presence of bacteria was not clear from Giemsa staining or tissue culture. The postoperative course was uneventful, and TTE performed approximately 12 months after surgery showed no recurrence.

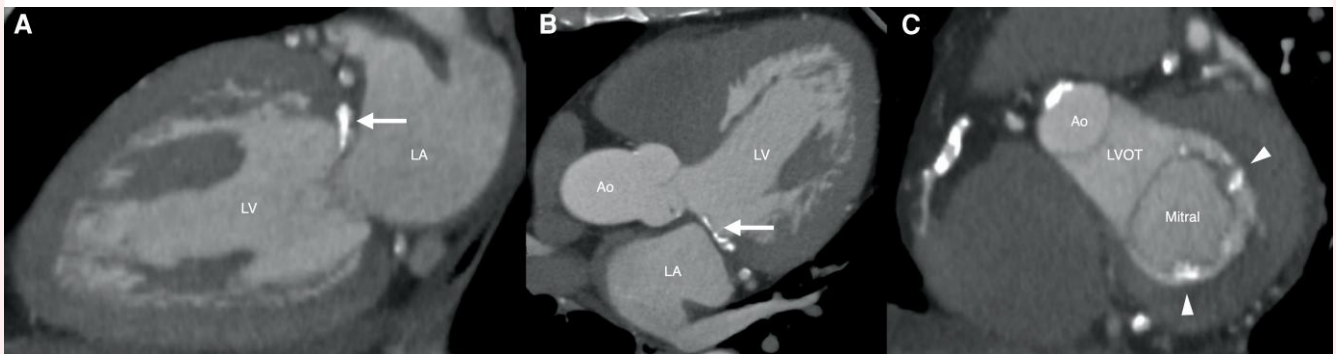


Figure 2 Contrast-enhanced electrocardiography-gated cardiac computed tomography image at day 20. (A,B) A clubbed mass (white arrow) with calcification attached to the left ventricular side near the anterior mitral annulus. (C) Mild calcification of the posterior mitral annulus (white arrowhead). Ao, ascending aorta; ECG, electrocardiogram; LA, left atrium; LV, left ventricle; LVOT, left ventricular outflow tract.

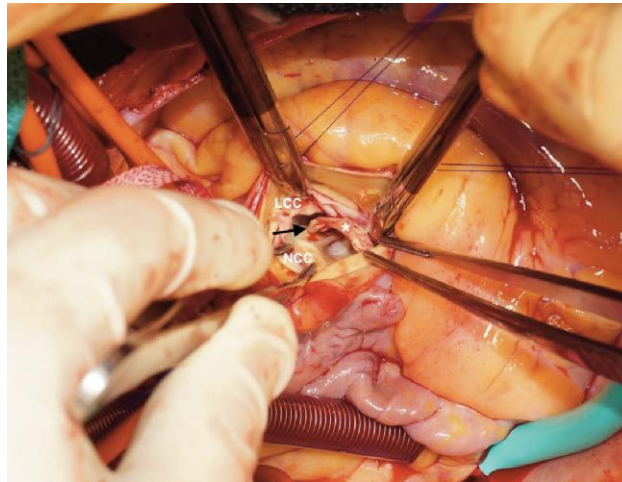


Figure 3 Intraoperative image showing a mass (*) with a stalk (black arrow) just below the border between the non-coronary cusp and left coronary cusp. NCC, non-coronary cusp; LCC, left coronary cusp.

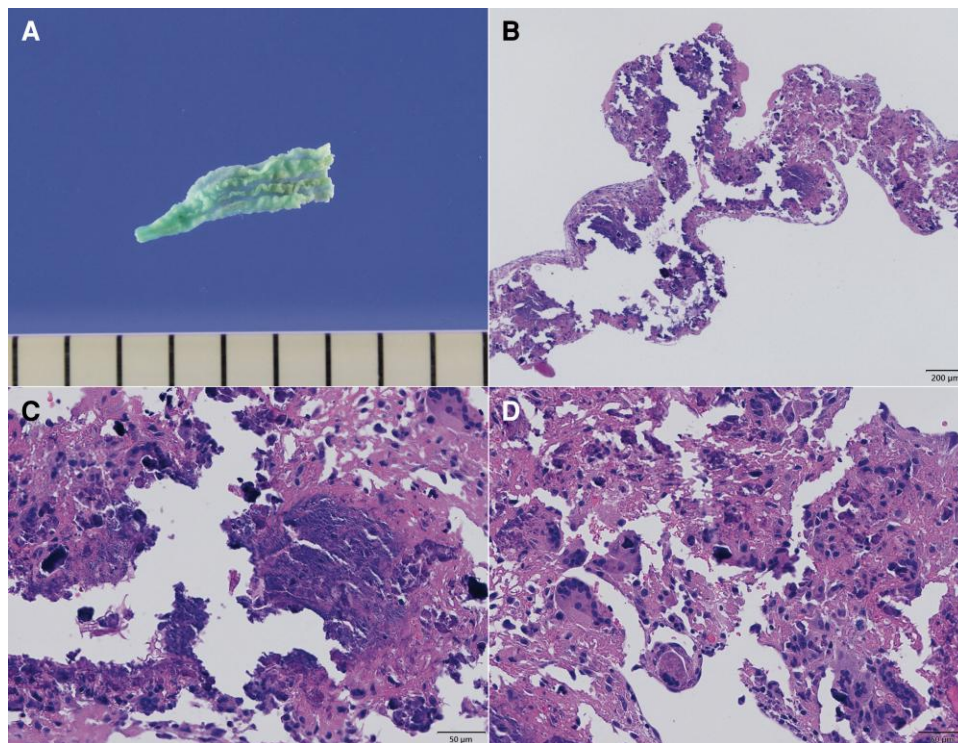


Figure 4 Examination of resection specimens. (A) Part of the resected mass (the right-hand edge had been torn off by 5 mm). The overall size of the mass is 25 × 5 mm, and the stem is on the left. The one scale on the ruler seen below indicates 5 mm. (B–D) Haematoxylin and eosin stain of the resected specimen. Calcification is diffusely present. Neutrophils, fibrin, fibroblasts, and collagenous tissue are almost absent. There is no evidence of neoplasia.

Discussion

Here, we report a unique type of intracardiac tissue, which appears to be a type of CAT based on its calcification-rich pathology; however, it exhibited three characteristics that somewhat differ from those typically reported in CAT, as described below.

First, the tissue was discovered following a fever. TTE performed a month earlier showed no mass; however, shortly afterwards, the patient became febrile, and TTE showed a new mobile mass in the left ventricular outflow tract. Although four sets of blood cultures were negative, BCNIE was suspected because the fever and inflammatory response decreased after the commencement of antibacterial treatment. It is unclear whether the fever was related to the mass because it continued to rapidly expand after the resolution of the fever, the pathological features of the resected tissue did not indicate inflammation, and tissue cultures were negative.

Second, the tissue suddenly appeared and rapidly grew. This tissue was not seen on TTE 1 month earlier and increased by 7 mm (about 1.5-fold) on TEE just 3 days after it was first found on TTE. Even a comparison between TTEs showed a 10 mm (approximately 1.7-fold) enlargement of the tissue in 9 days. There are reports of CATs expanding over 1–2 months but no reports of ‘daily’ expansion.^{4,5} There has been a case report of a CAT showing rapid enlargement due to partial detachment of the MAC;⁶ however, in the present case, the MAC was barely seen on the preoperative CT. Moreover, the possibility of the increase of thrombus attached to the MAC was inconsistent with the pathological findings, suggesting the tissue itself increased in size over a short period, which seems to be an important feature that is not reported in most CATs.

Finally, the tissue showed diffuse calcification on histopathology, unaccompanied by inflammatory findings. A CAT is histopathologically characterized by the presence of calcified nodules on an amorphous background of fibrin with degeneration and localized chronic inflammation, together with calcification.¹ Contrarily, the tissue in the present case showed diffuse calcification but lacked fibrin and inflammatory findings. It is unclear how the ‘mass of calcification’ grows.

Meanwhile, the present mass exhibited some features similar to a CAT. For instance, CATs tend to cause complications in patients with end-stage renal failure² and can be seen as a hyper-echoic tissue that ‘swings’.^{4,6} A limitation of this study is that the timing and frequency of echocardiography may have contributed to ‘the speed’ of mass expansion. In our case, there was sufficient opportunity to observe the daily enlargement of the mass. In contrast, in many previous reports on CATs, resection was performed immediately after the discovery of the mass. In reality, CATs may also be able to expand just as rapidly as observed in this case.

Conclusively, we present a case report of an intracardiac tissue with similar characteristics to a typical CAT based on imaging but with somewhat different pathology. Careful follow-up or early resection is important for embolization prevention and diagnosis of an intracardiac calcified mass detected on echocardiography or other imaging modalities because of the possibility of rapid expansion.

Lead author biography



Yuta Sudo was born in 1985, in Tokyo, Japan. He studied cardiovascular medicine at the Kameda Medical Centre and Yokosuka Kyosai Hospital. He specializes in cardiovascular care and has extensive knowledge on heart failure and cardiac implantable electronic devices. Apart from his medical interests, he loves football passionately.

Supplementary material

Supplementary material is available at *European Heart Journal—Case Reports*.

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Consent: The authors confirm that written consent for submission and publication of this case report, including images and associated text, was obtained from the patient in line with the Committee on Publication Ethics (COPE) guidance.

Conflict of interest: None declared.

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Data availability

The data underlying this article are available in the article and in its online supplementary material.

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