Serial Cardiac Magnetic Resonance Imaging of a Rapidly Progressing Liquefaction Necrosis of Mitral Annulus Calcification Associated With Embolic Stroke

On Chen, MD; Nripen Dontineni, MD; Ghaith Nahlawi, MD; Geetha P. Bhumireddy, MD; Seol Young Han, MD; Yakoub Katri, MD; Iosif M. Gulkarov, MD; Daniel G. Ciaburri, MD; Anthony J. Tortolani, MD; Richard S. Lazzaro, MD; Terrence J. Sacchi, MD; Joshua A. Socolow, MD; John F. Heitner, MD

Mitral annulus calcification (MAC) is a common finding in the elderly. A rare manifestation of MAC is liquefaction necrosis that can be mistaken for a tumor or an abscess. Because its course is most often benign, a correct diagnosis is imperative to avoid unnecessary workup or treatment.

Case
A 76-year-old woman with history of hypertension and dyslipidemia presented with chest pain and elevated cardiac enzymes. A coronary angiogram revealed no significant coronary artery disease.

Echocardiogram (Figure 1) revealed a large, solid mass within the atrioventricular groove and the lateral wall of the left ventricle. There was moderate calcification of the mitral valve annulus. Computed tomography scan of the chest (Figure 2) revealed a soft tissue density inseparable from the region of the mitral valve and the left ventricular wall. Cardiac magnetic resonance (CMR) showed a large mass involving the basal lateral wall near the atrioventricular groove, extending into the left atrium (Figure 3A and 3B). The mass was slightly hyperintense on T1 (Figure 4) and hypointense on T2 imaging (Figure 5). The mass was homogenous on delayed enhancement with a bright ring (Figure 6), the characteristics were not changed with fat saturation, and it was avascular by perfusion (Figure 7). The patient was discharged from the hospital with a scheduled outpatient workup to continue.

The patient returned 2 weeks later, however, with an acute stroke in arterial distribution, and workup for an embolic source commenced. A CMR (Figure 3C and 3D) revealed no left ventricular thrombus; the mass increased in size and changed in consistency, appearing semisolid.

The patient underwent a left thoracotomy for a biopsy, which revealed a thick whitish fluid (Figure 8) thought to be...
pus, leading to initiation of antibiotics. Cytology (Figure 9A) was negative for malignancy but showed macrophages and microcalcification consistent with a calcified cyst. Tissue pathology (Figure 9B and 9C) revealed fragments of fibro-collagenous tissue with calcifications and fragments of normal myocardial tissue. Fluid cultures were negative.

On follow-up CMR (Figure 3E and 3F), the mass appeared to be a fluid-filled cystic cavity with a hypoechoic center. An echocardiogram confirmed these changes.

**Discussion**

MAC is a common finding in the elderly; it is more common in women and patients with chronic kidney disease. The morbidity due to MAC is mostly associated with the hemodynamic effects on the mitral valve. A less common manifestation of MAC is liquefaction necrosis. Its prevalence is unclear and estimated to be 0.067% among all patients and 0.63% among patients with MAC. The exact mechanism causing this disease is not clear, but it appears to be associated with a surge in serum calcium. Echocardiographic follow-up revealed MAC to be a dynamic process with some cases transforming from a solid mass to liquefaction and regression or even complete resolution. In our case, serial CMR studies support the theory of a dynamic spectrum of this disease. In our patient, the mass exhibited rapid progression with changes in consistency over 1 month starting as a homogeneous mass on the first scan, with progressive changes over 1 month to a fluid-filled cavity. The use of CMR in the workup of liquefaction necrosis of MAC has been reported in only a few cases thus far; however, with the advantage of being able to determine tissue characterization, CMR is an ideal imaging modality. The findings in these previous reports were consistent with the findings in our patient. Liquefaction necrosis has been
described as a well-demarcated structure involving the mitral annulus, with a hyperintense center on T1-weighted images, no change on fat saturation sequences, and absence of enhancement on first pass perfusion. Delayed enhancement imaging reveals a hyperenhanced ring and absence of enhancement in the center.3

T1-weighted images cause tissue that has a short T1 relaxation time to be hyperintense, such as fat, sebaceous material, hemorrhagic products, proteinaceous fluid, and gadolinium-based contrast. T2-weighted images cause tissue to be hyperintense that have a long T2 relaxation time which generally occurs with water or edema (ie, as seen with many tumors). Calcium generally will have a hypointense signal on both T1- and T2-weighted images. This mass had similar CMR characteristics on T1- and T2-weighted imaging as previously described. The slightly hyperintense signal within the core of the mass on T1-weighted imaging is likely due to the high proteinaceous fluid content within the calcified wall. Because of the low water content and high protein content, the center of the mass was hypointense on T2-weighted imaging. The hyperenhanced ring on delayed enhanced imaging can be attributable to fibrotic myocardium surrounding the cystic mass that is likely a by-product of the high content of activated macrophages and lymphocytes and their respective cytokine damage of surrounding myocardium.

When biopsied, a caseous fluid consisting of calcium, cholesterol, and fatty acids was obtained leaving a calcified envelope.1,2 In our case, a similar fluid was obtained and was initially thought to be pus by gross inspection; however, cultures were negative.

Conclusion
The clinical course of MAC is usually benign and rarely requires intervention. The recognition of this disease and its distinct imaging features is essential to avoid unnecessary workup, surgical procedures, or treatment. However, an association with embolic stroke and mitral valve dysfunction has been described; surgical replacement of the mitral valve should be considered in those cases.4

CMR is an excellent imaging modality for the diagnosis of liquefaction necrosis because of the ability for tissue characterization via T1- and T2-weighted sequences, perfusion, fat saturation, and postcontrast sequences, and for follow-up of
progression and assessment of the effect on mitral valve function, as well.

Disclosures

None.

References


Figure 8. Surgical biopsy. Images taken during thoracotomy and biopsy; the white arrow indicates caseous fluid obtained after biopsy needle was removed.

Figure 9. Cytology and pathology. A, Light microscopy examination of fluid obtained during biopsy, a thin preparation using the Papa-nicolau stain. Asterisk indicates source fluid containing calcification (blue); black arrows, red blood cells. B and C, pathology, light microscopy examination of tissue obtained on biopsy. Cell block treated with hematoxylin and eosin. Black arrow indicates fibrocollagenous tissue with calcification; asterisk, normal myocardial cells.
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