

Uncommon Isolated Unilocular Myocardial Cyst in a Dog-Friendly Young Female Patient

— Multimodality Imaging —

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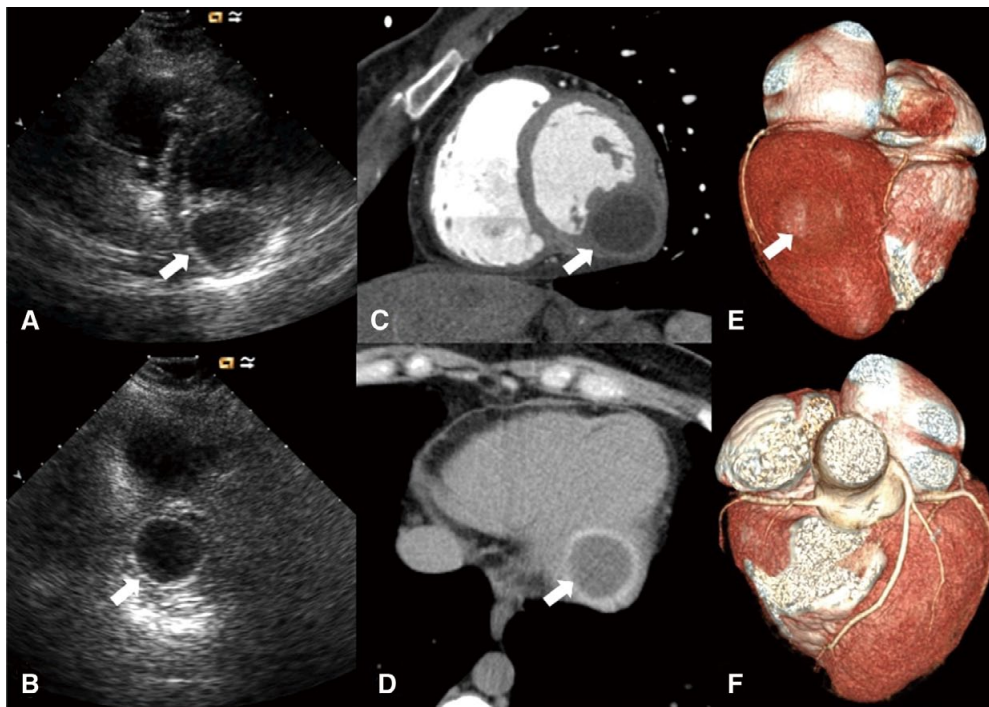


Figure 1. (A,B) Transthoracic echocardiography and (C,D) coronary computed tomography angiography (CCTA) showing a well-defined cystic mass localized within the infero-lateral left ventricle wall. The lesion's wall is hyperechoic on (A,B) echocardiography and hyperdense on (D) unenhanced CT. (E,F) 3-D volume rendering CCTA showing normal epicardial coronary arteries.

A 25-year-old woman was admitted to the emergency department with chest pain, dyspnea and fever (38.5°C). At admission, cardiac troponin I was <0.015 ng/mL; C-reactive protein, 5.54 mg/dL (normal,

0.0–0.50 mg/dL); white blood cells (WBC), $18.83 \times 10^3/\mu\text{L}$ (normal, $4.00\text{--}10.00 \times 10^3/\mu\text{L}$); neutrophils, $8.02 \times 10^3/\mu\text{L}$ (normal, $2.80\text{--}5.25 \times 10^3/\mu\text{L}$); eosinophils, $0.47 \times 10^3/\mu\text{L}$ (normal, $0.07\text{--}0.42 \times 10^3/\mu\text{L}$); and *Echinococcus* IgG antibodies,

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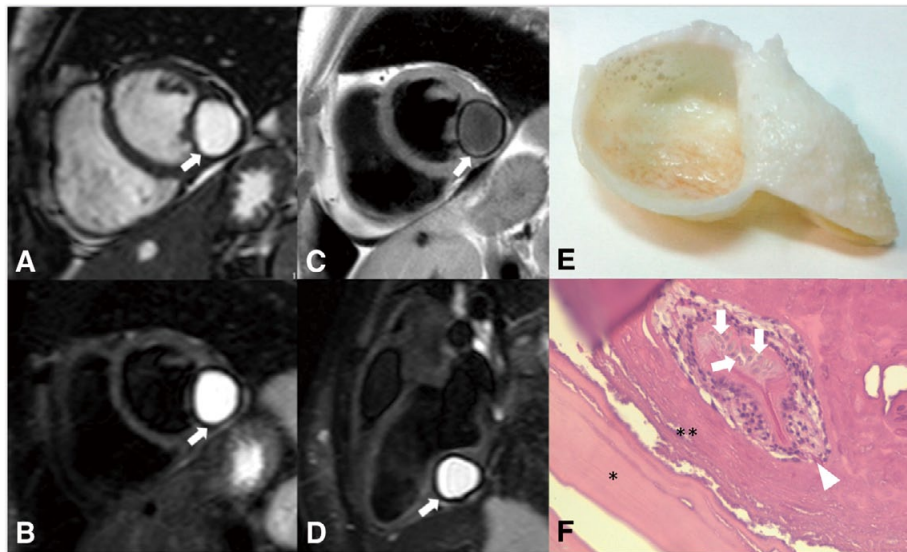


Figure 2. (A) Short-axis (SA) steady-state free precession, (B,D) SA and left ventricular outflow tract T2-weighted (T2W) short tau inversion recovery imaging confirm the presence of a large cystic lesion characterized by a homogenous high signal intensity with no septae or solid portions. The lesion is delimited by a thin hypointense outer rim (pericyst) on (C) T1W and (D) T2W imaging, which is rich in collagen and generated by the host. (E) Excised surgical specimen of the left ventricular cystic mass. (F) Histopathology of the left ventricle resection tissue. Close-up of a fragment stained with hematoxylin and eosin showing fibrosis generated by the host (asterisk), the pericyst (double asterisk) made of proteinaceous material incorporating a scolex (arrowhead), the presence of which can be inferred by some hooklets (arrows).

32 NTU (normal, <11 NTU). Transthoracic echocardiography showed a single, large, well-defined cystic mass in the infero-lateral left ventricle (LV) wall characterized by hyperechoic contour (Figure 1A,B). The patient first underwent coronary computed tomography angiography (CCTA) to exclude coronary artery compression and to clearly visualize the cyst and the surrounding tissues. CCTA showed normal epicardial coronary arteries (Figure 1E,F) and confirmed the presence of a 30×28-mm round lesion with homogeneous fluid content within the LV infero-lateral wall delimited by a thin, slightly calcified, high-attenuation wall (Figure 1C,D). On cardiovascular magnetic resonance imaging (CMR) the cystic nature of the mass was confirmed, characterized by a homogenous very low signal intensity on T1-weighted imaging (T1WI), and homogenous high signal intensity on T2-weighted imaging (T2WI). The mass was delimited by a thin hypointense outer rim on T1WI and T2WI (Figure 2A–D). This CT and CMR pattern (“rim sign”) represents the pericyst, a dense fibrous capsule from the reactive host tissue, which strongly suggests a diagnosis of type 1 hydatid cyst (HC; unilocular HC with walls).¹ Given, however, the high WBC, in order to differentiate HC from cardiac abscess, 3 series of axial single-shot spin-echo echo-planar diffusion-weighted imaging were acquired using the following b values: 0, 600 and 1,200 s/mm², to calculate the apparent diffusion coefficient (ADC). ADC characterizes the biological tissues according to diffusion properties of the water content. The mean ADC for type 1 HC and for abscesses is $2.84 \pm 0.38 \times 10^{-3} \text{ mm}^2/\text{s}$ and $1.09 \pm 0.32 \times 10^{-3} \text{ mm}^2/\text{s}$, respectively.² On ADC mapping the present lesion had high signal intensity (Figure S1B), and ADC was $2.59 \times 10^{-3} \text{ mm}^2/\text{s}$ – very close to that reported in the literature. The ADC of abscesses is significantly lower

than that of HC because abscesses contain inflammatory cells, bacteria, necrotic tissues, and highly cellular and viscous proteinaceous exudate, which restrict diffusion, resulting in low ADC.² The patient disclosed that she had regular close contact with domestic dogs. She also noted that she regularly brought home and cared for stray dogs. Serologic immunofluorescent antibody testing was positive for *E. granulosus*. After surgical excision of the cyst, pathology confirmed the diagnosis of HC both macroscopically (Figure 2E) and microscopically (Figure 2F), and the presence of a typical laminated cyst wall lined by germinal layer and scolices.

Hydatid disease is a parasitosis caused by *E. granulosus*, and is endemic in European, Middle Eastern, Mediterranean, South American and African countries. Humans become infected after contact with a definitive host, namely canines, or via consumption of contaminated water or vegetation. Ultrasonography (US) is the tool of choice for diagnosis, although CT, CMR and serology are also frequently used to corroborate the diagnosis. HC can involve all organs, although it preferentially affects the liver (50–70%) and lungs (20–30%). Cardiac HC have been described in 0.5–2% of echinococcosis cases and are usually univesicular. Isolated cardiac HC, without liver involvement, is very uncommon.^{3,4} Cardiac involvement occurs via invasion of the myocardium, first through the coronary artery circulation. The clinical presentation is usually insidious (chest pain, arrhythmias, myocardial infarction) and directly related to the location and the size of the HC. It can cause displacement of coronary vessels, rhythm disturbances and mechanical interference with the atrioventricular valves and ventricular function. The growth of HC is usually slow and asymptomatic but there is always the

lethal hazard of cyst perforation, which may become life-threatening, causing cardiac tamponade, purulent pericarditis and sudden cardiac death.³ Early diagnosis and an integrated treatment strategy are crucial. The resolution of CT is excellent in the evaluation of HC and, in particular, of cyst wall calcification, but the multiplanar and multiparameter imaging facilities and the non-invasiveness of CMR allow comprehensive evaluation of the disease.

Disclosures

The authors declare no conflict of interest.

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Supplementary Files

Supplementary File 1

Figure S1. (A) The lesion does not enhance on short-axis first-pass perfusion.

Please find supplementary file(s);
<http://dx.doi.org/10.1253/circj.CJ-16-1215>