We present a case of a 38-year-old Turkish patient with a previously diagnosed cardiac hydatidosis, which is a parasitic infection most commonly induced by *Echinococcus granulosus* and with only rare cardiac involvement (0.5%–2.0%). In 1998, the patient survived a cardiogenic shock caused by the rupture of 1 hydatic cyst and concomitant cardiac tamponade. Postsurgery, the patient was treated pharmacologically with albendazole.

In 2006, a cardiac magnetic resonance and a computed tomography examination showed recurrent myocardial cystic formations (Figure 1). Disadvantageously, the largest cyst was located in the inferior wall of the left ventricle in close vicinity to the mitral valvular apparatus. An interdisciplinary conference came to the conclusion to neither treat the patient surgically, because of an extensive myocardial and perivalvular defect, nor pharmacologically with albendazole, because even anthelmintic medical intervention would have borne the risk of weakening the cystic wall and causing wall rupture.

From 2006 to 2012, noninvasive clinical follow-up showed no major complications. In the last evaluation in 2012, the patient was asymptomatic, the ECG showed Q waves in the inferior wall (Figure 2), and the echocardiogram revealed the presence of the lesion, without clear demonstration of its morphology, however (Figure 3). In contrast, cardiac magnetic resonance illustrated morphological features of the cyst in greater detail and gave clear evidence of a cyst transformation from a World Health Organization (Table) type I CE2, an active cyst with multivesicular, multiseptated, rosette-like or honeycomb-like aspect (Figure 4A through 4F), into a CE3B cyst, a transitional cyst with daughter cysts in a solid matrix (Figure 4G through 4J). Rapidly, only within 8 weeks, the lesion showed a marked cyst regression into an almost inactive CE4 form. Moreover, late gadolinium enhancement images showed a fibrous capsule surrounding the lesion, which might represent an inflammatory response contributing to the favorable evolution of the disease (Figure 5).

With this report, we demonstrate a follow-up of a patient with cardiac hydatidosis in whom neither the published previously surgical treatment nor an anthelminthic therapy could be performed, but who nevertheless showed cystic...
regression into an almost inactive form with heterogeneous content and no daughter cysts. We also highlight the value of cardiac magnetic resonance imaging, which allowed a safe follow-up in this case.

Disclosures

None.

References


Figure 2. ECG demonstrated sinus rhythm with a heart rate of 58 bpm, no signs of atrioventricular delay, but small Q waves with corresponding repolarization defects in II, III, and aVF (black arrows) correlating with the anatomic location of the cystic lesion in the inferior wall.

Figure 3. ECG images demonstrating a parasternal long-axis (A) and a midventricular short-axis (B) view showing different aspects of the cystic lesion in the inferior left ventricular (LV) wall (dashed lines). LA indicates left atrium.
<table>
<thead>
<tr>
<th>Type</th>
<th>Description</th>
<th>Stage</th>
</tr>
</thead>
<tbody>
<tr>
<td>CE1</td>
<td>Unilocular, simple cyst with uniform anechoic content</td>
<td>Active</td>
</tr>
<tr>
<td>CE2</td>
<td>Multivesicular with daughter cysts. Rosette-like or honeycomb-like structures</td>
<td>Active</td>
</tr>
<tr>
<td>CE3A</td>
<td>Cyst with detached membranes (water-lily sign)</td>
<td>Transitional</td>
</tr>
<tr>
<td>CE3B</td>
<td>Cyst with daughter cysts in solid matrix</td>
<td>Transitional</td>
</tr>
<tr>
<td>CE4</td>
<td>Cyst with heterogenous hypoechoic/hyperechoic contents. No daughter cysts</td>
<td>Inactive</td>
</tr>
<tr>
<td>CE5</td>
<td>Solid plus calcified wall</td>
<td>Inactive</td>
</tr>
</tbody>
</table>

Figure 4. Steady-state free precession (SSFP) short-axis (A, C, E, G, and I) and 2-chamber (B, D, F, H, and J) views of cardiac magnetic resonance (CMR) examinations in 2006 (A and B), 2007 (C and D), 2009 (E and F), and 2012 (G through J). A lesion with a multivesicular aspect (type CE2 cyst) can be detected in the left ventricular inferior wall (A through F). Note a solid matrix between the cysts in the examination of 2012 showing a change in the lesion stage (type CE3B cyst) according to the World Health Organization (G and H) and an increased solid matrix and reduction of the daughter cysts in subsequent examination (I and J).
Figure 5. Late gadolinium enhancement (LGE) short-axis (A) and long-axis (B) views, from 2012. Note the LGE of the cyst wall, showing the replacement of the myocardium by the cystic lesion and its fibrous capsule.
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