Case Report

Cardiac echinococcosis

Echocardiographic diagnosis with a fatal clinical outcome

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ABSTRACT

Hydatid disease (HD) is an endemic disease found in various regions of the world. The organs mostly affected are the liver and lung. Cardiac involvement in HD is rare. The symptoms are mainly due to the mass effect of the calcific hydatid cyst obstructing the blood or the lymphatic vessels. Other manifestations are secondary infection or cyst rupture in the involved organ. Here, we report a middle aged female patient with no history of medical illness who presented to the emergency room with an unrecordable blood pressure. Echocardiogram showed multiple calcific cysts of echinococcosis granulosa in the left ventricle cavity. In the clinical setting, where there is detection of HD elsewhere in the body, an echocardiogram is mandatory as cardiac involvement is serious and may be fatal.

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Cardiac involvement of hydatid disease (HD) is rare, with a cardiac echinococcosis incidence of 0.2-2%.[1] We often discover cardiac involvement incidentally. Earlier studies report the site of cardiac involvement in the pericardial wall, ventricular free wall, or the septum.[2] In one report, the site of the hydatid cyst was the right ventricle, which ruptured, causing pulmonary embolism and anaphylactic shock.[3] There are no previous reports of involvement of the left ventricle (LV) cavity with multilocular cysts and clinical presentation of fatal shock. We report the unusual cardiac site of HD involvement and illustrate the role of echocardiogram in establishing the diagnosis of a formerly healthy middle aged female, who presented to the emergency room for the first time with unrecordable blood pressure, cold periphery and subsequently went into irreversible shock. The echocardiogram performed showed multiple calcific cysts in the left ventricle cavity.

Case Report. A 43-year-old female presented to the ER with a dry cough and shortness of breath a few days before admission. She had no history of medical or surgical illnesses, and she was not taking any medication. Clinically she had cold and clammy peripheries with peripheral cyanosis and an unrecordable blood pressure, tachycardia of 110 beat per minutes, and temperature of 37.1°C.

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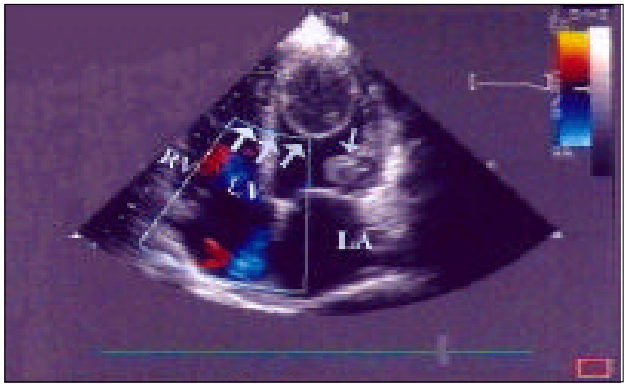

Figure 1 - Four chamber view showing large calcific cystic mass of 7×5 cm located in the apex of the LV (3 large arrows) with daughter cyst inside. Another small cyst of 2×1.5 cm located just above the posterior leaflet of the mitral valve (single small arrow). LV- left ventricle

Cardiovascular examination revealed a soft systolic murmur of 2/6 at the apex and lungs with coarse crackles in the right base, jugular venous pressure was not elevated, there were no palpable lymph nodes, the liver and spleen were not palpable with no other organomegaly and no leg swelling. She was intubated and transferred to the intensive care unit, where she received intravenous crystalloid, colloids, dopamine, and hydrocortisone. Laboratory results showed oxygen saturation in room air of 85%, white blood count of 12 × 10^9 (normal range [NR]: 3.6-9.6 × 10^9), hemoglobin of 10.5 gm/dl (NR: 12-14.5 gm/dL), platelet count 186 × 10^9 (NR: 140-440 × 10^9/ L), differential white blood cell showed neutrophil count of 75%, basophilic count of 1%, lymphocyte of 12%, eosinophil of 10%, monocyte of 2%, erythrocyte sedimentation rate (ESR) of 55 mm/h (NR: 0-20 mm/h), alanine transaminase (ALT) 56 U/l (NR: 5-35 U/l), aspartate transaminase (AST) 70 U/l (NR: 5-35 U/l), alkaline phosphatase 466 U/l (NR: 30-300 U/l), bilirubin 17 umol/l (NR: <18), serum amylase 100 U/l (NR: 0-180 U/l), urea 5.8 mmol/l (NR: 2.5-6.7 mmol/l), creatinine 100 umol/l (NR: 62-140 umol/l), potassium 4.6 mmol/l (NR: 3.5-5 mmol/l), sodium 140 mmol/l (NR: 137-148 mmol/l), calcium 2.1 mmol/l (NR: 2.12-2.65 mmol/l), phosphate 1.1 mmol/l (NR: 0.8-8.5 mmol/l), creatine kinase (CK) 200 U/l (NR: 25-195 U/l), creatine kinase isoenzyme (CKMB) of 4 U/l (NR: <10 U/L), troponin level 0.02 ugm/l (NR: 0.005-0.2 ugm/l), immunoglobulin G 2 g/l (NR: 5-13 g/l), immunoglobulin E 0.5 g/l (NR: <0.005 g/l), immunoglobulin M 10 g/l (NR: 0.4-2.5 g/l), immunoglobulin A 2 gm/l (NR: 0.5-4 g/l), uric acid 300 mol/l (NR: 140-390 mol/l), prothrombin time 12 seconds (NR: 10-17 seconds), activated partial thromboplastin 32 seconds (NR: 30-40 seconds), international normalized ratio (INR) 1.1 (NR: <1), total protein of 69 gm/l (NR: 60-80 g/l), and albumin of 27 gm/l (NR: 35-50 g/l). A 12-lead (electrocardiogram) showed sinus rhythm with left bundle branch block. Chest x-ray showed mild cardiomegaly and small pulmonary opacity in the right lower lobe, no lung congestion. A chest and abdominal CT showed fatty liver, 2 cystic structures in the LV cavity with no pericardial effusion. There was no mass or cystic structure in the abdomen. An echocardiogram showed global hypokinesia with LV end systolic cavity dimension of 4.9 cm and LV end diastolic dimension of 6.4 cm and LV ejection fraction of 30%. The left atrium with a dimension of 4.1 cm and with moderate mitral and tricuspid regurgitation, with a large calcific cystic mass of 7×5 cm located in the apex of the LV, with a daughter cyst inside and another small cyst of 2×1.5 cm just above the posterior leaflet of the mitral valve (**Figure 1**). She remained anuric, with no response to maximum inotropic support and later arrested with unsuccessful resuscitation.

Discussion. Echinococcosis is a troublesome disease, particularly common in sheep-raising communities caused by the cestode of *echinococcus granulosus*. In two-thirds of the cases, it mostly affects the liver, with the lung affected in nearly one third.[4,5] It can involve other organs such as the heart, brain, muscle, kidney, bone and pancreas in a smaller proportion.[5] Cardiac involvement is rare, and may cause severe complications such as cyst rupture, anaphylactic shock and sudden death.[6] In our case, the patient presented with peripheral hypoperfusion with the presence of multiple calcific cysts (hydatid) in the LV cavity. She was asymptomatic before admission, and the CT of the lung and abdomen showed no pulmonary or lung involvement. The presence of calcium in the wall of the cyst requires more than 10 years to develop, and total calcification indicates that the cyst was mostly non-viable.[7] The myocardial depression and the dilation of the LV may be a primary disease, or secondary to the toxic effect of the released toxins and the metabolic effect of the state of low coronary perfusion. The state of shock, high eosinophilic count and the high level of immunoglobulin E suggest the rupture of one cyst in the myocardial cavity with wide spread systemic dissemination and immunological reaction, resulting in her fatal condition.[8] The definitive evidence of HD in this case was the presence of the calcium in the wall and the small daughter cyst in the large cyst in the LV. Therefore, in the clinical setting where we detect HD elsewhere in the body, it is mandatory that

patients have an echocardiogram as the cardiac involvement is serious and possibly fatal. Furthermore, we should include hydatid cyst in the differential diagnosis of any cystic mass in the myocardium as this may alter the surgical management.

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