

A Rare Presentation of Cardiac Hydatid Cyst: Stroke and Acute Aortic Occlusion

Hafize Yaliniz, Acar Tokcan, Tümer Ulus, Bülent Kisacikoglu, Orhan Kemal Salih, Mehmet Sah Topcuoglu, Hakan Poyrazoglu, Cumhuri Alhan

University of Çukurova, Faculty of Medicine, Department of Cardiovascular Surgery, Adana, Turkey



Dr. Yaliniz

ABSTRACT

Cardiac involvement in hydatid disease is uncommon. We report a case of a surgically treated ruptured left ventricular hydatid cyst, which presented with acute stroke and was later complicated by distal aortic embolism due to perioperative dislodgement of the germinative membrane.

INTRODUCTION

Cardiac involvement in hydatid disease is relatively uncommon, occurring in only 0.05% to 2% of all echinococcosis (hydatid) infestations [Ameli 1989, Salih 1998]. In our institution, the proportion of operations for cardiac and pulmonary hydatid cysts was previously reported as 3/418 or 0.7% [Salih 1998].

This article presents a surgically treated ruptured left ventricular hydatid cyst with acute stroke and late distal aortic embolism.

CASE REPORT

A 16-year-old boy without significant medical history was admitted to the neurology department with acute right hemiparesis and aphasia. The heart sounds were normal and all peripheral pulses were present. Chest roentgenograph and routine blood test results were all normal, except for minimal leukocytosis with eosinophilia. Electrocardiography revealed sinus tachycardia and ST-segment elevations in leads II and III and atrial ventricular fibrillation, suggesting subepicardial damage. The cranial computed tomographic (CT) scan performed on admission showed merely moderate brain edema; however, a subsequent brain magnetic resonance image (MR) 24 hours later disclosed an ischemic infarct in the territory of the left middle cerebral artery.

Presented at the 10th Annual CTT Meeting 2004, Miami Beach, Florida, USA, March 10-13, 2004.

Address correspondence and reprint requests to: Hafize Yaliniz, Çukurova University, Faculty of Medicine, Department of Cardiovascular Surgery, 01330, Balcali, Adana, Turkey; 90-322-3386060 3176; fax: 90-322-3386656 (e-mail: hafize101@yahoo.com).

On 2-dimensional echocardiography, a 5 × 5-cm cavitory lesion with a central 3 × 3-cm solid, spheric, free mass in the apicoposterior wall of the left ventricle was seen bulging into the left ventricular chamber (Figure 1). This mass appeared to be floating with each contraction within the cavitory lesion. Abdominal ultrasonography showed multiple cystic lesions in the liver. Indirect hemagglutination test yielded 1/320 dilution.

On the basis of these findings, the presence of a ruptured left ventricular hydatid cyst was diagnosed, and the patient was transferred to our department for urgent surgery on the following day.

The operation was performed through a median sternotomy incision. The pericardium was opened and cardiopulmonary bypass was initiated using bicaval and ascending aortic cannulation. After the heart was arrested, the left ventricle was elevated, and prominent ivory, pericystic fibrous tissue was identified at the left ventricular apicoposterior wall. Following incision of the fibrous pericystic layer the cavity was entered. The absence of the germinative membrane, which was seen on echocardiography on the previous day, was striking. A 1 × 1.5-cm hole at the base of the cavity was seen to be communicating with the left ventricular chamber (Figure 2). The lost germinative membrane was also not in the left ventricle, which was inspected through this hole. This opening was closed with separate pledgeted sutures. The pericystic layer was sutured with pledgeted horizontal mattress sutures to obliterate the cavity. Following termination of cardiopulmonary bypass, the peripheral pulses were examined and bilateral femoral pulses could not be palpated. Doppler investigation on the operating table revealed distal abdominal aortic occlusion from the level of the renal arteries down to the bifurcation. Germinative-membrane embolization was diagnosed and laparotomy was subsequently performed. On inspection, it was seen that the abdominal aorta distal to the renal arteries was spastic and occluded with an elastic material from the level of the inferior mesenteric artery to the bifurcation. Aortotomy was performed, and the germinative membrane was enucleated (Figures 3A and 3B). The aortotomy was closed with running sutures. The hydatid cysts in the liver were left in situ for a second-stage operation. After the operation, all peripheral pulses were present.

The patient's postoperative course was uneventful. Albendazole treatment (10 mg/kg per day) was begun postoperatively. The patient was discharged on the 10th postoperative day.

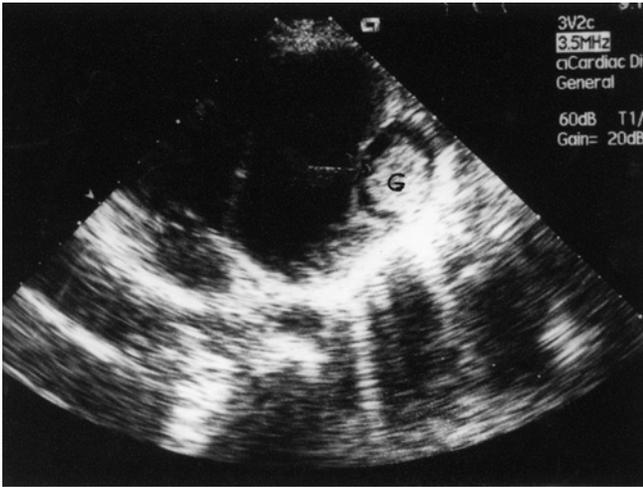


Figure 1. Preoperative 2-dimensional echocardiography of ruptured hydatid cyst located in the left ventricle wall. Arrow indicates the germinative membrane (G).

Hemiparesis and aphasia improved significantly approximately a month after his discharge, and no neurologic sequelae were detected during follow-up on the fifth postoperative month.

DISCUSSION

Echinococcosis is a human parasitic disease caused by the larval stage of *Echinococcus granulosus*, which is a cestode tapeworm. Echinococcal infestation is endemic in sheep-raising areas of the world, notably in the Mediterranean countries, the Middle East, South America, Australia, and New Zealand. Humans serve as incidental intermediate hosts for the larval stage of the parasite; infection is transmitted by ingestion of ova from contaminated dog feces. The hexacant embryos released from the ingested ova penetrate the intestinal mucosa and enter the portal circulation, from which they are carried to the liver. Embryos escaping from the vascular beds of liver and lungs, which serve as natural filters, may be

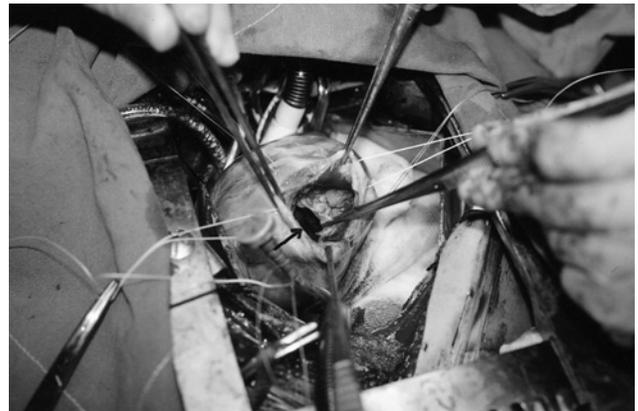


Figure 2. Cystic cavity in the left ventricular free wall. Arrow indicates the hole connecting the cavity and left ventricular chamber.

trapped in the heart. In 60% of the cases, they are located within the left ventricle [Ameli 1989, Salih 1998].

It is estimated that only approximately 10% of patients with cardiac hydatid cysts have clinical manifestation of the disease. Symptoms are variable and directly related to the location, size, and integrity of the cyst. Patients with cardiac hydatid cysts may often remain asymptomatic for many years, but there is always a threat of possible rupture. Rupture may occur into the pericardial space or, as in the present case, into the cardiac chambers. The latter is a life-threatening complication that occurs in approximately 40% of patients with untreated cardiac hydatid cysts. Liberation of hydatid fluid into the circulation may produce a profound, potentially fatal circulatory reaction [Ameli 1989, Salih 1998]. Germinative-membrane embolism secondary to intracardiac rupture of the cyst may lead to pulmonary [Bayazit 1991] and systemic embolization [Rosenberg 1993, Ceyran 2002, Unlu 2002]. Acute stroke as a presenting symptom of cardiac hydatid disease is exceptionally rare, and there are only several cases reported in the literature [Benomar 1994]. A few cases of limb ischemia secondary to peripheral arterial embolism of a

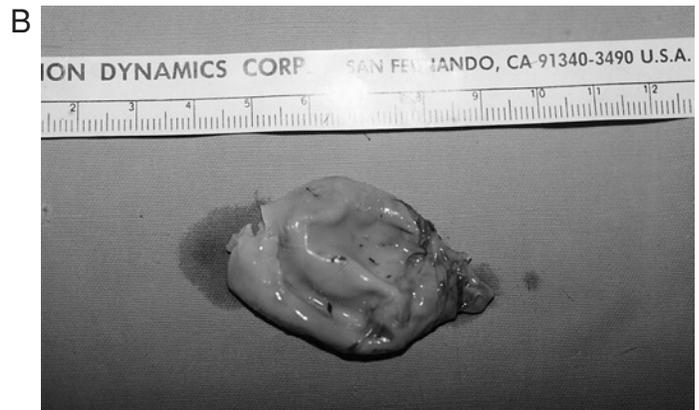


Figure 3. A, The germinative membrane is enucleated from the abdominal aorta (arrow); B, germinative membrane.

ruptured cardiac hydatid cyst have been reported [Ceyran 2002, Unlu 2002]. If occlusion involves the terminal aorta, bilateral lower-limb ischemia occurs. This was previously reported by Rosenberg et al [1993]. Our case is of particular interest because embolization of the germinative membrane to distal aorta possibly occurred perioperatively.

As in the present case, echocardiographic study is very useful in investigation of the cause of cerebral embolism. Although CT and MR scans may aid in the detection and localization of heart cysts, echocardiography is the diagnostic method of choice as a noninvasive, practical, and highly sensitive tool. Because of life-threatening complications, surgical excision is the treatment of choice even for asymptomatic cardiac hydatid cysts. Supplemental medical therapy with the benzimidazole derivatives mebendazole and albendazole has been recommended to reduce the risk of recurrence [Ameli 1989, Salih 1998] and is especially indicated in the event of intracardiac rupture.

In conclusion, the possibility of ruptured cardiac hydatid cysts should be considered in young patients with stroke or peripheral or pulmonary embolisms. Patients with ruptured cardiac hydatid cysts should undergo periodic follow-up for possibly disseminated secondary cysts.

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