HYDATID CYST EMBOLIZATION TO THE LEFT VENTRICLE: A CASE REPORT

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We report a 29-year old female with pulmonary hypertension who died suddenly. Se had multiple hepatic hydatid cysts, and a cystic embolus was found in the post-mortem study in the left ventricle, obstructing the mitral valve orifice. Possible mechanism include: a—cyst opening into the collector veins through the inferior vena

cava with subsequent pulmonary embolism; b—passage of the cyst through a patent foremen ovale (paradoxical embolism), due to increased rightsided pressures and c—passage into the left ventricle with obstruction of the mitral valve orifice causing inadequate left ventricular filling.

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Hepatic hydatid cysts are frequent in medical practice in Argentina¹, and vascular embolization to the lungs is not unusual. Cardiac localization of hydatid cysts is rare. Di Bello² found 350 instances published. We report the case of a 29 year old female patient who died, an hydatid cyst being found obstructing the mitral valve orifice.

CASE REPORT

A 29 year old female was admitted on April 15, 1975, because of progressive effort dyspnea, cyanosis, chest pain and jaundice. She had been diagnosed as having bronchial asthma in her childhood. On admission, heart rate was 88 bpm, with isolated premature contractions, and blood pressure was 100/60. S2 was loud and paradoxically split. There were signs of systemic venous congestion, and marked hepatomegaly. Laboratory tests revealed polycythemia, hyperbilirubinemia and hypoproteinemia. Casoni's test was negative. Chest roentgenogram showed a dilated pulmonary artery and clear lung fields (fig. 1). The ECG showed clockwise rotation, right ventricular hypertrophy, mild right bundle branch block and Sodi-Pallares' signs³ (fig. 2).

Four days after admission she died. Post-mortem study revealed multiple hepatic hydatidosis, with a significant decrease in right lung volume due to extrinsic hepatic compression. After opening the left atrium, gentle squeezing of the ventricular wall did not move the mitral cusps because of the presence of a cyst

(1 cm diameter) obstructing the mitral valve orifice (fig. 3). Histologic signs of chronic pulmonary hypertension were evident, as well as bilateral acute pulmonary thromboembolism and a patent foremen ovate.

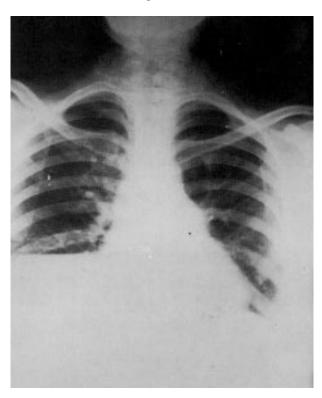


Fig. 1—Chest roentgenogram. Dilated pulmonary arteries and clear lungs. The elevation of the right hemidiaphragm Points out a marked hepatomegaly due to multiple hydatid cysts as shown at autopsy.

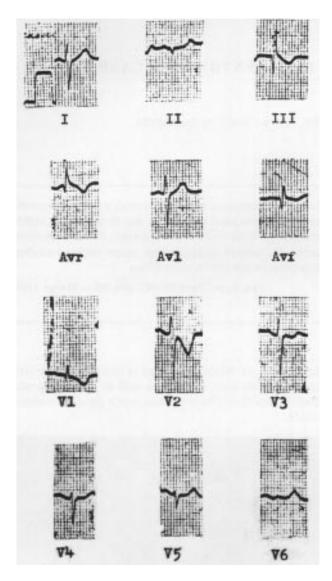


Fig. 2—Right ventricular hypertrophy, mild right bundle branch block and Sodi-Pallares' sign (small q wave in VI lead, strongly suggesting right atrial dilacation)

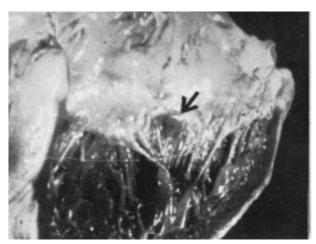


Fig. 3—Hydatid cyst in the left ventricle (arrow).

DISCUSSION

As no pulmonary echinococcal cysts could be demonstrated, a paradoxical embolization through the foramen ovale was the most likely mechanism for the cyst embolus.

Several cases have been reported of passage of hepatic cysts into the inferior vena cava, due to its close contact with the liver¹. Cyst opening may also occur into the collector veins, since these thin-walled structures are surrounded by scarce amounts of connective tissue¹.

This passage does not always lead to death of the patient, since, when the fissure is narrow the cyst may get without major damage. If it reaches "vis a tergo" in the right atrium, it can lead to death due to pulmonary embolism or secondary pulmonary echinococcosis. Or, as in our patient, it may reach the heart through a patent foremen ovate due to increased right-sided pressures.

The hydatid cyst could have reached the left ventricle by means of: a) passage from the inferior vena cava into the pulmonary artery, pulmonary anastomosis, and pulmonary veins. Nevertheless, this seems unlikely due to the size of the anastomosis (200 microns); b) passage of parasites through the same circuit and later cystic transformation in the capillaries or the pulmonary anastomosis. This is rather difficult because of the lack of secondary pulmonary cysts, and of the slow cyst growing rate (1mm/month); c) a paradoxical embolus through an atrial septal defect or a patent foramen ovale⁴. Sometimes, if the pulmonary embolus is not the first episode, pulmonary hypertension and right ventricular failure could ensue, increasing a previous right-to-left shunt at the atrial level^{5,6}. In addition, the increase in right atrial pressure (evidenced by indirect ECG signs)⁵, leads to right atrial dilatation and a greater patency of the foramen ovale

We believe the hydatid cyst reached the left heart through an anatomically patent foramen ovale, favored by right-to-left shunt which could account for the marked cyanosis appearing one hour before death. Probably the cyst remained trapped between the major and secondary orifices of the mitral valve, among the interchordal spaces, disturbing left ventricular filling. In any event, the patient had many other possible causes for sudden death (acute pulmonary embolism and/or acute right heart failure in the setting of recurrent pulmonary embolism and pulmonary hypertension) other than hypothetical mitral valve obstruction. In any case, other aethiologies of mitral valve obstruction such as atrial myxoma or pedunculated tumors have to be ruled out. In these cases, a careful echocardiographic and cineangiographic assessment may prove helpful to obtain an accurate diagnosis and decide about surgical treatment.

RESUMO

Paciente de 29 anos de idade do sexo feminino, com hipertensão arterial pulmonar faleceu subitamente.

À necrópsia foram demonstrados múltiplos cistos hidáticos pulmonares e um êmbolo cístico obstruindo o orifício da valva mitral. Entre os mecanismos possíveis para explicar esse achado, incluem-se: a) abertura do cisto em veias que são coletoras da veia cava e embolia pulmonar subseqüente; b) embolia paradoxal através de foramen oval permeável, na presença de hipertensão pulmonar; c) migração para o ventrículo esquerdo causando obstrução da valva mitral.

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