Man with a Right Thigh Mass

(See page 417 for the Photo Quiz)

Figure 1. MRI of the patient’s right thigh showing intramuscular cysts (A, B) and CT of the patient’s lungs showing a cavity with peripheral calcifications in right lung (C) and a densely calcified lesion in right lobe of the liver (D).

Diagnosis: Multiorgan cystic echinococcosis (Hydatid cyst).

The clue to the diagnosis was the presence of cystic thigh lesions and concomitant calcified hepatic and pulmonary lesions in a patient who was continually exposed to an area in which echinococcosis is endemic. A unique feature of the current case is the radiographic demonstration of different stages of infestation: a calcified cyst, a cavitary lesion with rim calcification, and regular cysts (figure 1). Virtually any anatomic site can be affected by Echinococcus species. It most frequently affects the liver (in 50%–70% of cases) and lungs (20%–30%), but it can also affect other organs, such as the heart, the brain, and the bones (<10%) [1]. Involvement of thigh muscles is rare. The diagnosis was confirmed by an echinococcus IgG ELISA titer of 3.05 (normal value, ≤0.88). The absence of eosinophilia is typical, because this finding is usually present only when there is leakage of antigenic cyst fluid [2]. Our patient was PPD negative. Fungal serologic tests as well as fungal and mycobacterial stains and cultures had negative results.

Echinococcosis is a zoonosis caused by larval stages (metacestodes) of cestodes that belong to the genus Echinococcus, family Taeniidae. Cystic echinococcosis is caused by Echinococcus granulosus. Although knowledge of its global distribution is incomplete, it is present on all continents and is endemic in Mediterranean countries and in parts of the former Soviet Union, China, Northern and Eastern Africa, Australia, and South America [3]. In the United States, immigrants from areas of
endemicity constitute an important risk group [4]. Dogs and other canids are the definitive hosts of *Echinococcus* species, whereas other mammals (typically ungulates, such as sheep) serve as intermediate hosts. Humans are not part of the natural cycle, but they can become aberrant or incidental hosts via ingestion of *E. granulosus* eggs. The clinical course of cystic echinococcosis is variable, ranging from rapid expansion with formation of daughter cysts and rupture into the biliary or bronchial tree to slow expansion, spontaneous collapse, and dense calcification of dead cysts [5]. *Echinococcus multilocularis* is the cause of alveolar echinococcosis. It is acquired from wild canids and is characteristically fulminant with a high mortality rate. Polycystic echinococcosis, caused by *Echinococcus vogeli* or *Echinococcus oligarthrus*, is restricted to South and Central America and is only rarely associated with human infection [6].

The diagnosis of echinococcosis is typically first suspected as the result of radiologic findings of cystic lesions, but a combination of serologic tests is needed for confirmation. Primary serologic tests, such as ELISA, indirect hemagglutination, latex agglutination, and immunoelctrophoresis, have high sensitivities but unsatisfactory specificities. For example, one of the most commonly used tests (ELISA) has sensitivities of 83%–96%, depending on the involved organ and number of cysts [7], whereas its specificity is lower because of cross reactivity with *Taenia solium* and other helminths. Thus, an adjunctive secondary test with higher specificity for *Echinococcus* species, such as immunodiffusion (arc 5) or immunoblot assays [8, 9], is recommended by the World Health Organization [10], although these tests are generally unavailable in the United States. In patients with equivocal serological test results, fine-needle aspiration may be considered for diagnosis, but this has to be balanced against the risk of anaphylaxis if there is spillage of cyst contents [11].

Cystic echinococcosis may not require treatment unless the patient is symptomatic or the function of adjacent tissue is at risk. Treatment options include surgical excision in toto or puncture, aspiration, injection of a proctoscolicidal agent (e.g., 95% ethanol, hypertonic saline, or cetrimide), and reaspiration with or without pre- and postinterventional pharmacotherapy with a benzimidazole (albendazole or mebendazole). Pharmacotherapy alone may be used in selected cases. Albendazole has superior efficacy to mebendazole [12], and the highest response rates are seen with young cysts that are <8 cm in diameter [13, 14]. The combination of albendazole plus praziquantel appears to be superior to albendazole alone [15].

The extent of our patient’s thigh involvement precluded treatment with local proctoscolicides, and he opted for systemic drug therapy after considering the potential morbidity from complete surgical excision. He tolerated oral albendazole (400 mg twice daily) and a follow-up MRI after 4 months of treatment showed some reduction in the size of the thigh cysts. The calcified hepatic and pulmonary lesions remained unchanged.

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