Comment on “Acute Echinococcosis: a Case Report”

We read with interest the report by Di Comite et al. (1). We are not aware of previous reports of “acute” manifestations in cystic echinococcosis (CE) in humans or lower animals and we question the validity of this report.

Experimental infections with eggs in animal intermediate hosts have shown that the oncosphere embryo is found in its final site of development (usually the liver) within 3 h postinfection; during the subsequent 10 to 14 days a series of reorganizational events occurs involving cellular proliferation, degeneration of oncospheral hooks, muscular atrophy, vesicularization and central cavity formation, and development of both germinal and laminated layers of the cyst wall (3). Formation of brood capsules and protoscoleces requires a longer time period (10 to 12 months in pigs and 10 months to 4 years in sheep). We have not observed clinical manifestations associated with these early events in experimental infections of lower animals and are not aware of such reports in the experimental literature. In humans, symptoms do not occur and the condition is generally not diagnosed until a year or more postinfection, when the metacestode is well developed and, by virtue of its size and location, begins to produce perceptible discomfort.

Consequently, the absence of experimental evidence of acute manifestations of CE and the lack of confirmatory evidence in this report lead us to doubt that this patient’s symptoms were attributable to CE infection. The authors state that the patient had vacationed in an area of endemicity 3 months before onset of symptoms and that he had been “exposed to sheepdog feces”; however, they provide no details of the exposure. The authors do not indicate the duration of albendazole treatment but report “almost complete resolution of pulmonary nodules, decrease in size of hilar lymph nodes and resolution of eosinophilia” 4 months after discharge. The fact that eosinophilia and pulmonary nodules were reported to be present before treatment with albendazole and subsequently disappeared is not conclusive evidence that these signs were due to CE. The serologic data is also inconclusive. The authors report an indirect hemagglutination test titer of 1:600 without giving the normal range and subsequent values after therapy. However, this test is often nonspecific, and false-positive reactions may be caused by other conditions. Specific immunoglobulin M (IgM) antibodies were not measured, nor was total and specific IgE measured or immunoblotting done. Finally, the authors cite an article by Taylor and others (2) in support of their statement that “a comparison with acute experimental echinococcosis in animals shows a similar response to therapy.” However, Taylor et al. are misquoted here, since their paper regards incomplete cure of echinococcal cysts in gerbils even when treated with high doses of albendazole.

In conclusion, the authors did not perform studies that would confirm their hypothesis. We believe that this case report would have required a more careful and detailed approach for the authors’ claim to be considered.

REFERENCES


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Ed. Note: The authors of the published article declined to respond.