HOW TO MANAGE ASYMPTOMATIC LIVER HYDATIDS

Farrokh Saidi MD

Cystic echinococcal disease disappeared many decades ago from industrial countries. Only rarely now, and primarily because of increased international travel, liver hydatids surface as clinical curiosities at medical conferences in western countries. The situation is usually that of a liver hydatid having ruptured into the biliary system causing bile duct obstruction. Invariably the consensus among discussants is that the patient should have been operated upon much earlier to avoid the cyst becoming complicated. Such a verdict, however, is inappropriate and inapplicable in endemic areas of developing countries where echinococcal disease is still rampant. In these regions so many cases of asymptomatic cases of liver hydatids are detected in the course of ultrasound investigation for other conditions, that it would impossible to handle all of them surgically.

The fact that many liver hydatids are first encountered as irregularly calcified and clinically silent lesions, suggests that most liver hydatids undergo a slow process of spontaneous abortion. They can, therefore, be left alone. An equally important reason for a conservative approach to incidentally discovered liver hydatids in endemic areas is an economic one. To manage all asymptomatic liver hydatids prophylactically to avert possible complications, would overwhelm available health resources in endemic areas. To operate on the many, fearing complications in the few, cannot be justified in that setting.

Can incidentally discovered asymptomatic liver cysts be treated medically when risk taking, even for the few, is unacceptable? About forty years ago benzimidazole carbamate derivatives, mebendazole first and then albendazole, were reported from Europe to have deleterious effects on the larval stage of the parasite Echinococcus granulosus in man. Mebendazole has been used for many years in India as an effective vermifuge with low absorption rate. Enough experience has accumulated to show that clinical efficacy of these drugs against hydatid cyst is no more than about 50%, even after prolonged administration. Fortunately their toxicity over long periods of time is also very low. It would be reasonable, therefore, to empirically administer these drugs to patients in endemic areas who are accidentally found to have a benign cystic lesion of the liver suspected to be echinococcal. Drug treatment should be continuous and not interrupted, with liver function and the blood picture checked at intervals, and pregnancy avoided in the interim. Both the physician and the patient should fully understand that success is neither guaranteed nor apparent until at least a full year of drug treatment. Involution of the cyst may have been the fate of the cyst and not the effect of the medicine. Given the fact that pharmaceutical companies of industrial countries are not likely to invest money in discovering better drugs for Echinococcosis, a disease seen only in endemic areas of the world, one should not expect a truly effective drug for this parasitic condition becoming available soon. The problem of how and when asymptomatic liver hydatids should be treated, remains unknown.

Another approach to asymptomatic liver hydatids, also originating in the West, is percutaneous needle aspiration of the cyst followed by instillation of hypertonic saline and 90% alcohol. For a number of reasons this particular form of treatment, while in line with the now fashionable mode of minimal access cannot be recommended, for two reasons:

a) While univesicular liver cysts can be needle aspirated, only centrally located multivesicular hydatids are likely to rupture into major bile ducts and precipitate acute biliary obstruction. Hitting each and every small daughter cyst in these multivesiular lesions is technically impossible and
fraught with unacceptable risks.

b) Notwithstanding many reports of presumed success, 20% hypertonic saline does not have the hoped for scolicidal potency, nor is it innocuous.6 Lastly, entry of these agents into the biliary tree is likely to result in lethal sclerosing cholangitis months or years later.

The basic question remains as to which incidentally discovered liver cysts are likely to become complicated, justifying their prophylactic treatment. The natural course of the disease, in particular the relative frequency of a given cyst becoming calcified is not known. Clearly, the needed information will not become available if a preemptive surgical intervention to avoid the risks of complication, no matter how remote, is the dominant treatment policy. The fact that hydatid cysts grow at a very leisurely pace makes it difficult to convince patients with asymptomatic cysts to wait for months if not years to see if they are or are not at risk. It is true that in the end either the host or parasite must die. But the odds are heavily stacked against the latter, even though liver cysts take a long time before showing which way they will go.

What, then, is a clinically logical and economically justifiable approach to the large number of incidentally discovered asymptomatic liver hydatids encountered in endemic areas? For one thing, vague pain and nonspecific complaints localized to the upper abdomen, usually noticed by patients when their liver cyst has been discovered, can be safely ignored. There is no anatomic or pathologic reason for an uncomplicated liver hydatid cyst to be symptomatic, regardless of its size or location. They expand much too slowly to be able to stretch Glisson’s capsule. It might be difficult to assure patients that their vague aches and pains do not portend imminent rupture into major bile ducts. The value of immunodiagnosis, resting on the antigenicity of the hydatid fluid has been well summarized.7, 8 My personal opinion is that a positive serology can be dismissed as useless in terms of indicating viability and hence the likelihood of impending complication.

There can be no question, of course, that a liver hydatid must be viable and grow in size to impinge and cause pressure necrosis of the wall of an adjacent large bile duct to bring about a serious complication. This does not mean, however, that expansion in size is relentless, nor inevitably lead to a complication. For unknown reasons, some cyst may stop growing at any time while some cysts reach an enormous size and remain clinically silent. Still, if serial sonographic studies at, say, six monthly intervals show definite enlargement of a particular cyst, that particular cyst bears watching and perhaps elective surgical removal.

The aim in this compact overview of treating asymptomatic liver hydatids has been to declare those management policies originating or applied in industrial West, where the disease no longer exists, as not necessarily applicable or appropriate to endemic East, where the disease still abounds. However, one treatment policy that emanated in the industrial West more than a century and a half ago, should be put into action in all parts of the world endemic to echinococcal disease, as it broke the life cycle of the parasite: rigid enforcement of public health measures, to prevent access of stray dogs to offal at slaughterhouses. That this self-evident step has not been followed or perhaps cannot be followed in endemic areas of the world, underlines one particular aspect of this parasitic condition: the effective control of this disease as well as the public health problems it creates, is more sociocultural than medicosurgical.

References