HEPATIC CAPILLARIASIS MAY SIMULATE THE SYNDROME OF VISCERAL LARVA MIGRANS, AN ANALYSIS

by

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The adults of *Capillaria hepatica*, typically a tissue dwelling nematode, are found in the hepatic parenchyma of a wide variety of vertebrate hosts including man. The reservoir hosts of the parasite include rats, mice, squirrels, rabbits, hare, dogs, cats, beaver and pigs (4). The nematode has a limited life span of five to six weeks in the liver of definitive host surviving only so long as is biologically necessary for propagation. Mature worms copulate and finally disintegrate releasing large number of eggs in the liver tissue.

The eggs of *C. hepatica* play a major role in the pathology of the infection eliciting granulomatous lesions and necrosis in the liver (11). The eggs of the nematode are entrapped in the hepatic tissue and access to exterior occurs when the host dies and the liver disintegrates. The eggs are then dispersed, complete their embryonation and attain infectivity. A further mode of egg dispersion is through predatory hosts and cannibalism in rodents. Human hepatic capillariasis is characterised mainly by hepatomegaly, eosinophilia and gammaglobulinaemia (1, 4, 17).

A majority of the two dozen authentic cases of human hepatic capillariasis so far recorded have been diagnosed at necropsy after fatal termination of the disease. The occult nature of the infection presents difficulties in recognition in that the condition is not amenable to ante-mortem diagnosis except perhaps by finding parasite eggs in liver punch biopsy material. In genuine cases of hepatic capillariasis no eggs are found in stool samples. Intestinal capillariasis in humans due to *C. philippinensis* is a better known infection in Far-East countries because the condition can be diagnosed easily by coproexamination. However, in few other instances the eggs of *C. hepatica* appearing in human stool samples consequent to accidental ingestion of eggs of this nematode contained in infected liver constitute a spurious infection.

The syndrome of hepatic capillariasis, viz., hepatomegaly, eosinophilia and gammaglobulinaemia has also been known to be pathognomic for visceral larva migrans (VLM). VLM is induced in humans by non-human ascarid larvae, involves mainly the liver and constitutes a public health problem of considerable magnitude. Accounts of toxocariasis and the pathogenesis and clinical effects of VLM are given by Glickman *et al.* (8) and Soulsby (19).
Since the precise aetiopathology of hepatomegaly in children in developing countries is not always well understood or determined, especially when facilities for specific ante-mortem diagnosis are not available, an outline is given below which suggests that at least some of the hepatomegaly in developing countries may be connected with C. hepatica infection.

During the course of a routine enquiry into the prevalence of a hepatic metacestode in house rats in the northern part of India in the early sixties the author (V.K.) noticed about one-fourth of these also harboured eggs of C. hepatica. There are other reports claiming a higher infection rate of C. hepatica in rodent population of tropical areas (4, 9). The house rats abound in typical village houses and granaries of these areas. Fairly large number of pariah dogs are also present in these situations as scavengers and also they prey on the house rats. In the predation process, the dogs ingest rat livers harbouring C. hepatica eggs and the unembryonated eggs pass through the digestive tract of the predator and are found in faeces; intact and undamaged (6). Through this mechanism the eggs are disseminated in the environment for embryonation. Cats also may contribute to this dissemination process in a similar way. The dogs and cats defecate in the open and often in places close to children's playground. Many of the children of younger age group in developing countries show pica and dirt eating habits and the embryonated eggs of C. hepatica may be accidently ingested in this way by the children provoking hepatic capillariasis. This condition has quite frequently been reported from children up to seven years of age (2, 4, 5, 10, 13, 14, 17, 21).

The question of hepatic capillariasis complicating the diagnosis of VLM would surface because of the extreme similarity of syndromes of the two disease conditions although the former is only occasionally encountered in man. Until the first half of the present century only a solitary case of human hepatic capillariasis was on record (12). Twenty-three more authentic case records have since been added to this growing list to-date. This increase in number of reported cases in a relatively short time span is probably because of an increasing awareness of the disease condition rather than an actual increase in its incidence.

It is remarkable that a vast majority of the cases of human hepatic capillariasis are reported from the developed world while the incidence of infection in the reservoir hosts and the prevailing ecosystem in the tropical countries would suggest an enhanced chance of human infections in these areas. Three cases of human hepatic capillariasis are on record from South Africa (4, 10, 17) and a case of C. hepatica infection in a woman is recorded from Nigeria (1). In other areas of the African continent this infection has not been identified in humans. An English soldier had picked up C. hepatica infection while serving in India and subsequently died because of the infection (12). Until now no other indigenous case of human infection is reported in Asia. Gupta & Randhawa (9) have, nevertheless, warned that the cases of hepatomegaly and eosinophilia among children in India need closer examination since cases of hepatic capillariasis may escape detection. Unfortunately, the isolated instances of case reports of hepatic capillariasis in humans do not provide the true epidemiological status of the disease in human on global basis. Because of the highly insidious nature of the disease, the specific aetiology in a majority
of human cases remain undiagnosed in the tropical developing or underdeveloped countries.

What may happen in cases of extremely low grade infection burden in humans where the patients may fail to present the classical clinical disease is a matter of speculation. Slais (18) has reported on nine human cases in what he considers the disease caused by ingestion of a single embryonated egg of C. hepatica. The diagnosis is based on the histopathological features of the liver nodules and worms were present in seven of these cases.

Silverman et al. (17) had pointed to the fact that their 17 month old child patient had a sand eating habit and that the terrain where she lived had numerous rats. These two factors, which may be ubiquitous in the tropical world, would certainly favour the build-up of C. hepatica infection among children especially where this parasite is commonly encountered in rodents. Straightforward evidences in support of some of these contentions should be forthcoming. This may be resolved by differential diagnosis through serological methods employing culture derived antigens.

Acknowledgement — The authors are thankful to Prof. E.J.L. Soulsby, University of Cambridge, U.K. for going through the manuscript and offering comments.

Received for publication on September 20, 1984.

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