

CAPILLARIA HEPATICA INFESTATION IN A DOG.

J. D. SMIT, Onderstepoort Laboratory

Infestation with *Capillaria hepatica* is common in rats and other rodents. In South Africa this parasite has been recorded in rats, mice, rabbits, hares, meerkats, gerbils, hedgehogs and man. No cases in the dog have been described.

According to the literature, the parasite has been observed in mice, rats, squirrels, rabbits, hares, dogs, cats, prairie dogs, beavers, muskats, peccaries, monkeys, pigs and man. Rodents, rats in particular, seem to be the natural hosts. Infestation of domestic animals and man is rare.

Cochrane *et al.* (1957) describe an infestation in 19 out of a random group of 40 rats received by the South African Institute for Medical Research. At Onderstepoort the parasite has occasionally been observed in rats, gerbils and once in a hedgehog. Due to the high incidence of capillariasis in rats it would be expected that dogs, being rat catchers, would become infected. However, in the South African literature no cases in dogs have been described. The case under consideration is the first known infestation in the canine species in South Africa.

The dog was a Bull Mastiff male about eight years old and in good condition. It originated from the Bon Accord area near Pretoria. The animal was off colour and was brought to the clinic for treatment. After being kept under observation for a few days it died. Clinically a tentative diagnosis of Leptospirosis was made.

The most outstanding features at the post-mortem examination were an erosive stomatitis, chronic nephritis, slight icterus, degeneration of myocardium and a generalised tendency towards calcification. The endocardium and the major blood vessels, the aorta in particular, showed a marked degree of calcification.

A histological examination revealed the following: The renal glomeruli were engorged with blood. The epithelium of the tubules of the kidney showed degeneration, with small casts present in the lumina and focal areas of calcification, as well as fairly extensive interstitial connective tissue increase. Numerous small calcified foci occurred in the capsule of the spleen, and a number of the blood vessels showed calcification of the intima. The myocardium showed a slight fatty degeneration and small foci of round cell infiltrations, with the blood capillaries engorged. An extensive calcification of the endocardium was an outstanding feature of the section. In the lung, focal areas of fibroses and calcification with an infiltration, consisting mainly of lymphocytes and active macrophages, occurred. Also present were a marked oedema, emphysema and congestion. The liver showed large, partially encapsulated foci of parasite eggs. Various stages of degeneration, and even disintegration, of the eggs were observed. Calcification was fairly advanced in the latter.

The cell reaction consisted mainly of lymphocytes and macrophages. No eosinophiles were present. The lobulation of the liver was more or less intact, although a certain amount of inter- and intralobular cirrhoses was present. No

Received for publication on 21 April, 1959.—Editor.

CAPILLARIA HEPATICA INFESTATION IN A DOG

indications of the adult parasite could be found. The liver cells showed atrophy and degeneration. A passive congestion almost resembling the picture seen with cyanotic induration, was present.

DISCUSSION

The cause of death in this case was a combination of factors. The chronic nephritis, liver damage and the generalised calcification of the vascular system were contributory factors leading to the final stage, the oedema of the lungs.

The *Capillaria hepatica* infection was of long standing. The absence of any indications of adult worms, the capsule formation and the disintegration and calcification of the eggs, support this contention. The absence of an eosinophilic cell reaction, which normally results from the disintegration of helminth parasites, indicates that the reaction must have subsided, again indicating the chronic nature of the condition.

The question arises as to whether there is any connection between the *Capillaria* infection and the vascular changes observed. Allergic granulomas are believed to represent a form of response of hypersensitive tissue (Churg *et al.*, 1951). According to Beaver *et al.* (1952) and Mercer *et al.* (1950), the sensitizing factor may be one of a variety of substances including parasites, especially round worms. Reimann (1943) describes a case of periarteritis nodosa in a *Trichina* infection. In a human infection with *Capillaria*, Otto *et al.* (1954) observed a slight focal calcification of renal tubular epithelium. In both these cases a definite relationship is shown between the parasitic infection and the vascular system of the host. Brill *et al.* (1953) describe a case of a *Toxocara* infection in a child with a syndrome of eosinophilia and disseminated visceral lesions. Granulomatous lesions of specific structure were found in the heart, liver, kidneys and lungs, often in relation to a severely altered small vein. They concluded that the *Toxocara* infection was responsible for this systemic hypersensitive state in the child.

In the dog under consideration no eosinophilic cell reaction was present, but this could be explained by the chronic nature of the lesions. At the time of the disintegration of the worms there must have been a severe allergic reaction. This assumption is warranted in view of the generally accepted contention that the release of foreign protein by the disintegrating parasites is responsible for an allergic reaction and a resultant increase in the number of eosinophiles. This allergic reaction was most probably responsible for the vascular changes, and ultimately for the combination of factors responsible for death. The general tendency towards calcification of the intima is of even greater significance if one considers the rare occurrence of arteriosclerosis and calcification in dogs. It is therefore not unlikely that the chronic *Capillaria* infection in this case predisposed the general tendency towards calcification, as a reaction of the body to a state of hypersensitivity produced by the disintegrating worms.

SUMMARY

A review of the literature on *Capillaria hepatica* infestation is given. The first case of *Capillaria hepatica* in the dog in South Africa is described. The main gross and histopathological changes are described. The relationship between the *Capillaria* infection and the vascular changes are discussed. The opinion is expressed that the disintegrating worms were responsible for an allergic state in the body, leading to generalised calcification of the intima of the blood vessels.

REFERENCES

- BEAVER, P. C., SNYDER, C. H., CARRERA, C. M., DENT, J. H. & LATTERTY, J. W. (1952). Chronic eosinophilia due to visceral larva migrans. *Pediatrics*, Vol. 9, pp. 7-19.
- BRILL, R., CHURG, J. & BEAVER, P. C. (1953). Allergic granulomatosis associated with visceral larva migrans. *Am. J. Clin. Path.*, Vol. 23, p. 1208.
- CHURG, J. & STRAUSS, L. (1951). Allergic granulomatosis, allergic angiitis and periarteritis nodosa. *Am. J. Path.*, Vol. 27, pp. 277-294.
- COCHRANE, J., SAGORIN, L. & WILCOCKS, M. G. (1957). *Capillaria hepatica* infection in man. *S. Afr. Med. J.*, 16-21 Sept.
- FOSTER, A. A. & JOHNSON, C. M. (1939). An explanation for the occurrence of *Capillaria hepatica* ova in human faeces suggested by the finding of three new hosts used as food. *Tr. Roy. Soc. Trop. Med. and Hyg.*, Vol. 32, pp. 639-644.
- LUTTERMOSER, G. W. (1936). A helminthological survey of Baltimore house rats (*Rattus norvegicus*). *Am. J. Hyg.*, Vol. 24, pp. 350-360.
- MERCER, R. D., LUND, H. Z., BLOOMFIELD, R. A. & CALDWELL, F. E. (1950). Larval ascariases as a cause of chronic eosinophilia with visceral manifestations. *Am. J. Dis. Child.*, Vol. 80, pp. 46-58.
- MÖNNIG, H. O. (1941). *Veterinary Helminthology and Entomology*. Baltimore, Tindall and Cox, London.
- ORTLEPP, R. J. (1958). Personal communication.
- OTTO, G. F., BERTHRONG, M., APPLEBY, R. E., RAWLINS, J. C. & WILBUR, O. (1959). Eosinophilia and hepatomegaly due to *Capillaria hepatica* infection. *Bulletin of the John Hopkins Hospital*, Vol. 94, No. 6, pp. 319-336.
- REIMANN, H. A., PRICE, A. H. & HERBERT, D. A. (1943). Trichinoses and periarteritis nodosa. *J. Am. Med. Ass.*, Vol. 122, p. 274.

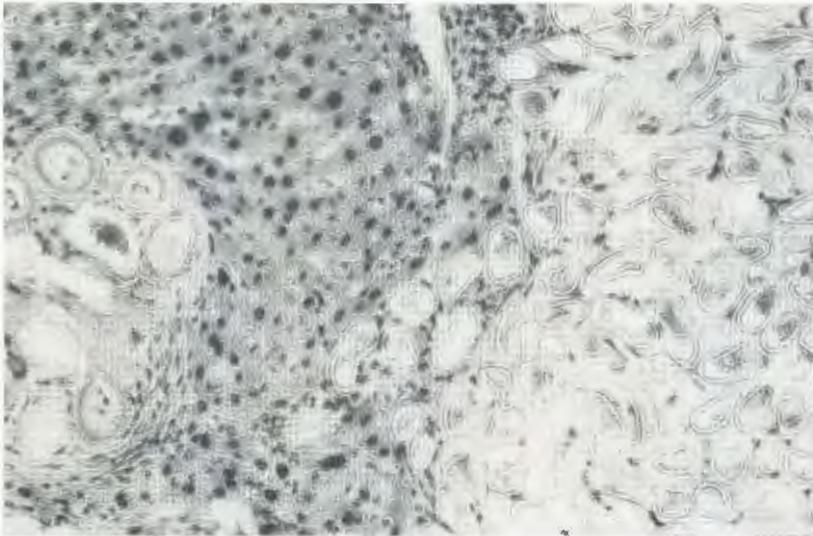


FIG. 1.—Centres of necrotic foci containing large numbers of eggs, almost complete absence of cell reaction. Rat liver. X. 48.

CAPILLARIA HEPATICA INFESTATION IN A DOG



FIG. 2.—Well preserved eggs; note typical barrel-shape with discernible plugs in each end.
Rat liver. X. 1000

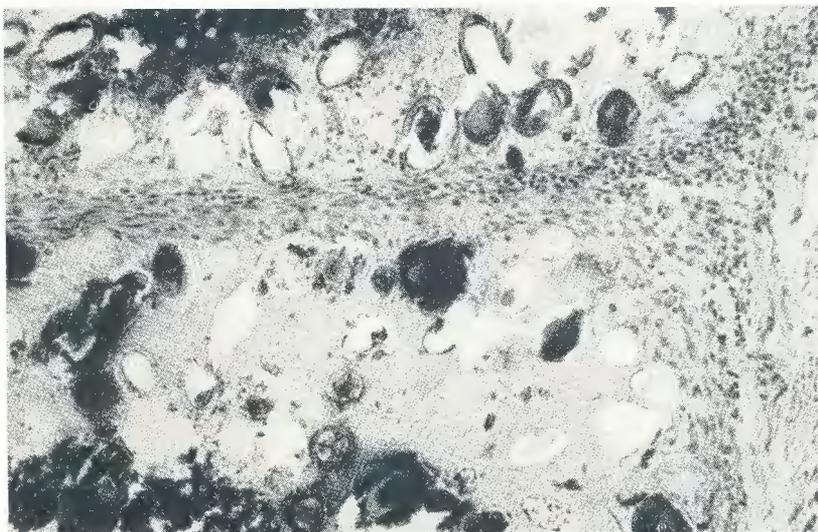


FIG. 3.—Necrotic centres containing eggs; note disintegration and calcification towards centre of foci, cell reaction mainly lymphocytes and macrophages, no eosinophiles.
Dog liver. X. 48.

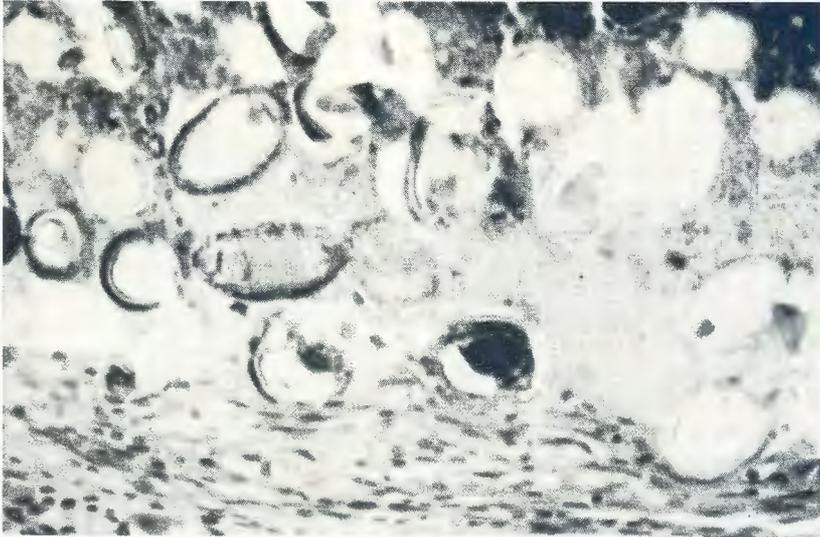


FIG. 4.—Necrotic centres containing typical eggs; note capsule formation. Dog liver. X. 125.

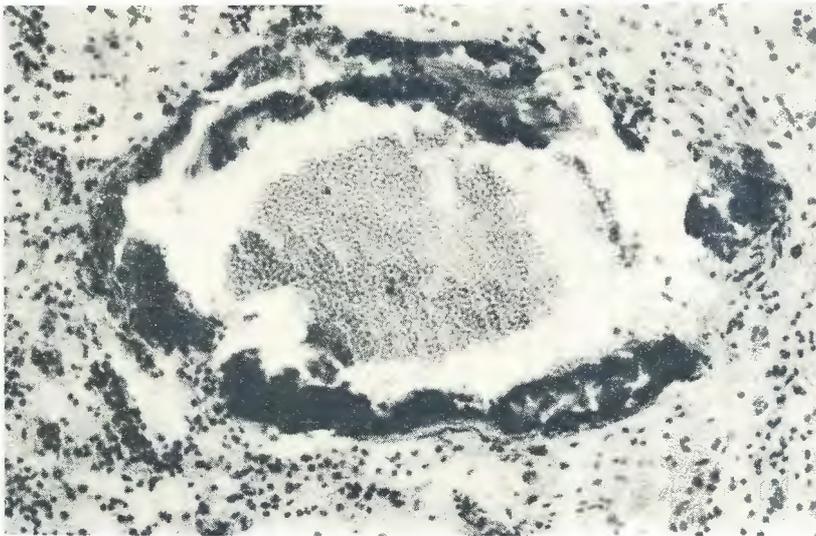


FIG. 5.—Artery in spleen, showing marked calcification of wall. Dog spleen. X. 48.

CAPILLARIA HEPATICA INFESTATION IN A DOG

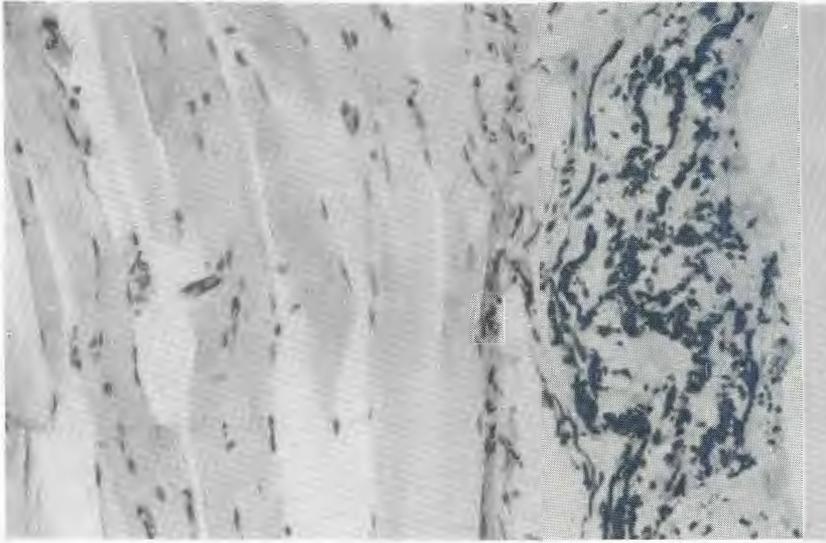


FIG. 6.—Heart, showing extensive calcification of endocardium. Dog endocardium. X. 48.