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CLINICOPATHOLOGICAL FEATURES AND HISTOPATHOLOGY OF EXPERIMENTAL HEPATIC CAPILLARIASIS IN MUSKRATS (ONDATRA ZIBETHICUS)

Joanna D. Borucinska,[1] Herbert J. Van Kruiningen,[1] Janine N. Caira,[2] and Antonio E. Garmendia[1]

[1] Northeastern Research Center for Wildlife Diseases, Department of Pathobiology, University of Connecticut, Storrs, Connecticut 06269-0089, USA

[2] Department of Ecology and Evolutionary Biology, University of Connecticut, Storrs, Connecticut 06269-3043, USA

ABSTRACT: Ten muskrats (*Ondatra zibethicus*) each were infected with 17,000 eggs (long-term study) and eight muskrats each were infected with 8,000 eggs (short-term study) of *Capillaria hepatica* (Nematoda). Food intake, body weight, and selected clinicopathological parameters were measured every 2 days for 28 days in the short-term study and every 14 days for 184 days in the long-term study. Muskrats in the short-term study had moderate to severe necrotizing granulomatous hepatitis associated with mild anorexia and weight loss, varying degrees of leukocytosis with eosinophilia and elevation of serum alanine and aspartate aminotransferases. No significant changes in packed cell volume, hemoglobin, total plasma protein, albumin, blood urea nitrogen, bilirubin, lactate dehydrogenase or alkaline phosphatase were found among animals from the short-term study. Muskrats in the long-term study had severe necrotizing granulomatous hepatitis associated with marked anorexia, weight loss and 60% mortality over 39 days post-inoculation (PI); animals that survived for 184 days did not return to pre-inoculation body weights despite returning to normal food intake. Hepatic lesions at 184 days PI consisted of minimal to severe liver replacement by *C. hepatica* eggs. No statistically significant differences in values of clinical parameters between inoculated animals and a non-inoculated control group from the long term study were found.

Key words: Muskrat, *Ondatra zibethicus*, *Capillaria hepatica*, hepatic capillariasis, clinical pathology, reservoir host.

INTRODUCTION

Capillaria hepatica (Bancroft 1893) is a nematode from the Order Trichurata, Family Capillaridae, that was originally described from the livers of rats by Bancroft (1893). Hepatic capillariasis has been recorded worldwide in over 20 mammalian species including humans (Choe et al., 1993). The first report of hepatic capillariasis in muskrats was in Canada (Price, 1931). Since then, high prevalences (up to 87%) of hepatic capillariasis have been recorded in some muskrat populations in the United States (Penn, 1952; Borucinska and Nielsen, 1993).

The disease in natural human cases or experimental infections in animals, is consistently characterized by elevated liver enzymes and eosinophilia (Zahner et al., 1981). Although hepatic capillariasis can be a significant mortality factor in laboratory and wild rodents (Vollerthun, 1972; Singleton and Spratt, 1986; Singleton and McCallum, 1990), nothing is known about it's effect on the mortality of muskrats, one of its natural hosts. Our objective was to describe the effect of hepatic capillariasis in muskrats.

MATERIALS AND METHODS

The project was conducted in two separate studies with two different groups of muskrats (Animal Protocol No. 9731102 approved by the Animal Care and Use Committee, University of Connecticut, Storrs). We used 30 animals between October 1994 and August 1995: 22 wild-trapped animals, including 10 from Wayne County, Pennsylvania (USA) (41°30′N, 75°30′W), 12 from Storrs, Connecticut (USA) (41°48′N, 72°15′W), and eight animals born to wild-caught parents maintained in a breeding colony established at the University of Connecticut, Storrs. Animals used in the experiments were housed individually in 0.5 × 2 m, wire-mesh cages on concrete floors, with metal nesting boxes and 100 × 100 × 10 cm metal water basins. Wood shavings were provided as nesting material. The light was natural. The temperature was kept between 12 and 16 C. A constant dose of a commercial laboratory ro-

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dent diet (Laboratory Rodent Diet 5001, PMI Feeds Inc., St. Louis, Missouri, USA) was fed daily. Animals were maintained under these conditions for 3 to 6 mo prior to the beginning of the study.

Eggs of *C. hepatica* used for inoculation were obtained from fresh muskrat carcasses submitted by local trappers. Infected livers were homogenized in a tissue blender and the liberated eggs were cleaned and embryonated following the method of Solomon and Soulsby (1973). Muskrats were inoculated with the eggs during laparotomy. The laparotomy was done under anesthesia with 1 to 2 mg/kg of xylazine hydrochloride (Rompun, Miles Inc., Shawnee Mission, Kansas, USA) followed by 10 mg/kg of ketamine hydrochloride (Ketaset, Fort Dodge Laboratories, Inc. Fort Dodge, Iowa, USA), both given intramuscularly. Eggs were injected through a 23 gauge needle into the anterior portion of the stomach. This laparotomy was used to establish that the wild-caught animals were free of liver disease prior to inoculation.

Blood samples were taken from each muskrat according to the schedules given for each study. Blood was always collected in the morning before feeding time. Animals were anesthetized and 2 ml of blood were collected from the jugular vein. Two blood smears, 0.1 ml of serum and 1 ml of blood preserved with sodium ethylenediaminetetraacetate (EDTA) were sent to a veterinary diagnostic laboratory (Cenvet Laboratory, Woodside, New York, USA) for analysis of the following parameters; total protein, albumin, blood urea nitrogen (BUN), total bilirubin, direct and indirect bilirubin, lactic dehydrogenase (LDH), alanine aminotransferase (ALT), aspartate aminotransferase (AST), alkaline phosphatase (AP), total white blood cell count (WBC), and differential WBC count, hematocrit (HTC) and hemoglobin (Hb). The measurments were done using an automated system with commercial reagents (Alliance 550 Express, CIBA Corning Diagnostic Corporation, Oberlin, Ohio, USA), according to the manufacturer's instructions. Blood cell counts were done from blood smears stained with the Wright stain (Sheehan and Hrapchak, 1980). The set of clinical parameters chosen included indicators of hepatic (ALT, AST, AP, LDH, bilirubins, albumin and total serum protein) and renal (BUN) functions, as well as indicators of nonspecific inflammatory processes (WBC count and differential count).

In the first experiment (long-term study), we evaluated the clinical and clinicopathological features in chronically infected (up to 184 days), wild-trapped, adult muskrats. To calculate the infectious dose of *C. hepatica* eggs for the chronic study, doses reported from experimental infections in mice and rats (Luttermoser, 1938) were extrapolated to average body weight in adult muskrats; this resulted in a calculated dose of 17,000 eggs per animal.

In the second experiment (short-term study) we evaluated early (up to 28 days) clinicopathologic changes in infected adult muskrats. The inoculation dose in the short term study was reduced to 8,000 eggs per animal because of high mortality encountered in muskrats given 17,000 eggs in the first experiment.

Twenty wild-trapped animals were used in the long-term study. These were divided into 10 control animals, consisting of six males weighing from 871 to 1,306 g and four females weighing from 812 to 1,029 g, and 10 experimental animals, consisting of six males weighing from 875 to 1,348 g and four females weighing from 958 to 1,390 g. The experimental animals were inoculated with 17,000 eggs each, and the control animals with water. After inoculation all animals were monitored three times a day, and after the two first deaths occurred in the experimental group, five times a day, to detect moribund individuals. At 30 days post-inoculation (PI), a second laparotomy was done to obtain a needle biopsy of liver lesions in the experimental group and to obtain a liver sample from the control group. All 20 animals were bled at the time of inoculation at day 0, at 30 days PI, and then every 14 days until the termination of the study at 184 days PI, at which point all animals were euthanized by intravenous injection of pentobarbital sodium and phenytoin sodium (Beuthanasia-D Special, Schering-Plough Animal Health Corp., Kenilworth, New Jersey, USA). Differences in the mean values of blood parameters between the experimental and control animals were evaluated using a *t*-test (StatWorks™, Apple Computer Inc., Cupertino, California, USA); $P \leq 0.05$ was considered significant.

The short-term study was conducted using eight animals born in captivity. These animals consisted of four males ranging in weight from 843 to 1,092 g, and four females weighing from 895 to 1,182 g. Each animal was inoculated with 8,000 eggs of *C. hepatica* as described. After inoculation, blood was collected from each animal every 48 hr for the following 28 days PI at which point the study was terminated and all animals were euthanized. Post-inoculation values of blood and serum parameters were compared to a pre-inoculation range established for each animal prior to inoculation with *C. hepatica* eggs. The pre-inoculation ranges were based on values from blood samples taken 9, 7, 4 and 2 days prior to inoculation; the ranges were calculated as the mean ± 2SD. Post-inoculation values that fell outside

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the control range, were considered abnormal (elevated or lowered).

In both studies, body weights of animals were taken at each bleeding time. Food intake was recorded daily for each animal. Samples from the brain, eyes, skeletal muscle, heart, adrenal, thyroid, thymus, pancreas, spleen, lymph nodes, lung, stomach, small and large intestines, kidney, urinary bladder, gonads and two sections of liver were collected from each animal during necropsy. All samples were fixed in 10% buffered formalin, embedded in paraffin, sectioned at 5 μm, stained with hematoxylin and eosin (H&E), and examined with light microscopy. Sections with lesions seen on H&E stained slides were selectively stained with any of the following stains: tissue Gram, Grocott, periodic acid-Schiff (PAS), trichrome, Voerhoff, Steiner's and Von Kossa as described in Sheehan and Hrapchak (1980).

RESULTS

Except for absence of swimming and diminished defecation during the day following surgery, no changes in behavior were observed in animals from the short term study throughout the length of the experiment. The group (mean ± SD intake units) pre-inoculation food intake (20 ± 0 intake units, considered 100%) declined to 80% (16 ± 4.6 intake units) during the first 5 days after inoculation; intake units were standard pellets. Intake returned to 100% at 10 days PI and then declined gradually to reach 72% (14 ± 7.1 intake units) of the pre-inoculation intake by the end of the study at 28 days. The group (mean ± SD) pre-inoculation body weight (1031 ± 17 g, considered 100%) declined steadily throughout the 28 days of the study, reaching 93% (955 ± 113 g) at 28 days PI.

Total WBC counts (mean ± 2SD) were elevated in six animals from 7 to 28 days PI, (highest mean 15,300 ± 1,000/mm^3 at 28 days PI). This elevation was predominantly due to eosinophilia present in all animals between 10 and 28 days PI (highest mean 991 ± 644/mm^3 at 28 days PI), lymphocytosis in four animals on day 28 PI (mean 5,822 ± 2,944/mm^3), and erratic neutrophilia in seven animals (highest mean 8,000 ± 3,402/mm^3 at 28 days PI). The ALT value was elevated in seven animals from 10 to 28 days PI (highest mean 292 ± 101 International Units, IU, at 26 days PI). The AST value was elevated in seven animals from 7 to 28 days PI (highest group mean 442 ± 174 IU at 21 days PI). Values of albumin, total protein, Hb, AP, LDH, BUN and bilirubin were within the control ranges established for each animal prior to inoculation.

All infected animals in the short term study survived until the termination of the study at 28 days PI. Gross lesions found at necropsy included, uniformly distributed, multifocal to coalescing white pinpoint to serpingeous foci under the capsule and within the liver (Fig. 1), moderate body wasting, and mild, diffuse cloudiness of the mesentery. Histologic lesions within liver were similar in all animals. Adult worms, eggs and inflammatory lesions had replaced over 50% of hepatic parenchyma (Fig. 2). The inflammatory response around worms or eggs ranged from none to severe, necrotizing, granulomatous hepatitis. The granulomas consisted of eosinophils, giant and epithelioid cells surrounding worms or eggs within necrotic debris and an amorphous PAS-positive material. Plasma cells and lymphocytes were usually present at the periphery of granulomas. No bacteria or fungi were seen in these lesions after staining with Gram and Grocott stains. With trichrome stain, we observed minimal collagen deposits immediately surrounding individual eggs and worms. With Von Kossa stain for calcium, we observed a mineralized layer (two to three cells thick) around, or small foci of mineralization within, most granulomatous lesions.

The periportal areas were edematous and infiltrated with small numbers of mast cells, eosinophils, lymphocytes, and plasma cells. In some portal triads lymphatic dilatation was present (Fig. 3a). A distinct change was found in the hepatic arterioles within portal tracts. This consisted of moderate separation, fragmentation, and hyalinization of fibers in the media as well as endothelial hypertrophy (Fig. 3b). Using

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FIGURE 1. Experimental *Capillaria hepatica* infection in a muskrat. White foci and tracts in the liver at 28 days PI. Bar = 2 cm.

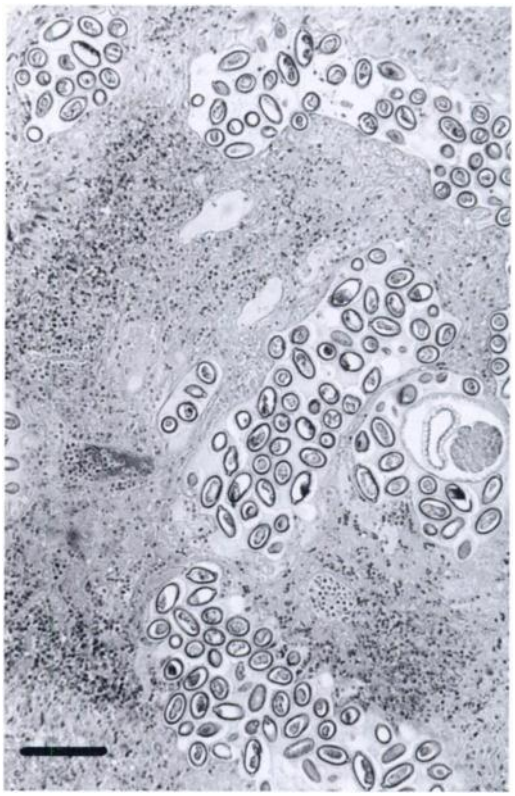


FIGURE 2. Experimental *Capillaria hepatica* infection in a muskrat at 28 days PI. The parasites and granulomatous inflammation replace most of the normal liver parenchyma. H&E. Bar = 150 μm.

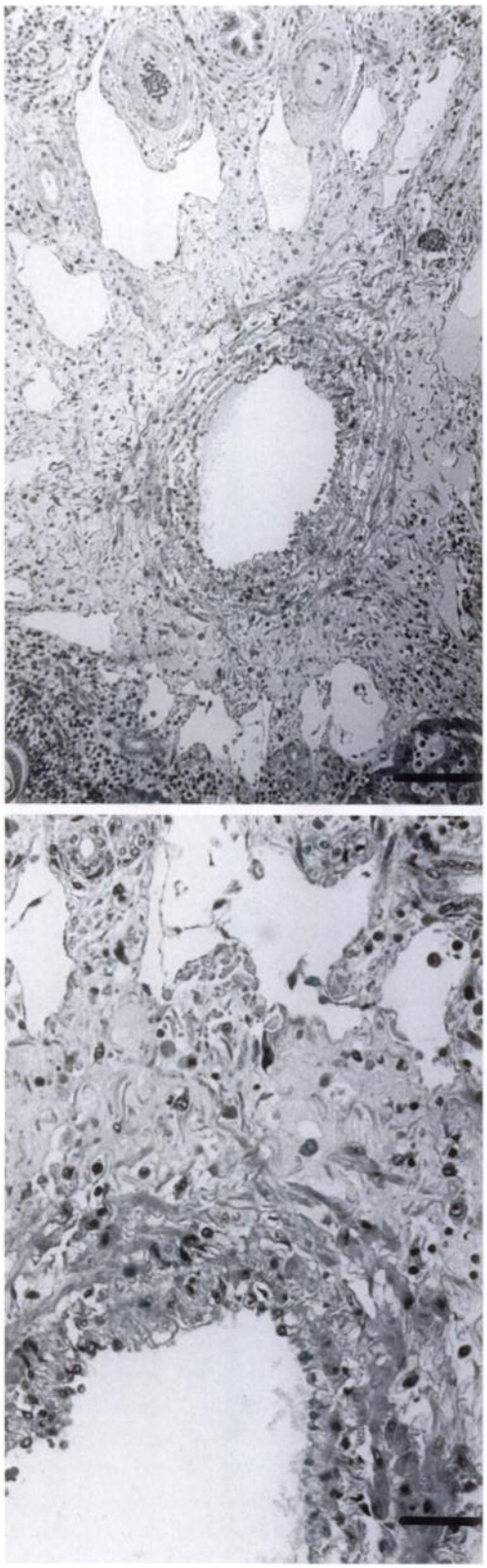
PAS and Congo red stains, we did not detect mucopolysaccharides or amyloid within walls of these vessels. Features of hepatocellular regeneration in the form of karyo- and cytomegaly, mitoses, and multinucleated hepatocytes, were seen occasionally in the liver. No lesions were found in the biliary system. The extrahepatic lesions found included pulmonary emboli containing *C. hepatica* eggs, chronic, multifocal peritonitis, and mild membranous glomerulonephropathy.

In the long-term study, diminished swimming and defecation was present in the control animals for 1 to 2 days following each surgery. Four animals in the control group died prior to termination of the study at days 132, 139, 140, and 166 PI, respectively; none of them exhibited detectable signs of debilitation prior to death.

The experimental animals from the long term study had diminished swimming and defecation for 1 to 6 days following each surgery and progressive weight loss throughout the study. Six animals in the experimental group died between 25 and 39 days PI (five animals overnight and one animal during anesthesia), these deaths were not preceeded by a visible moribund condition; the remaining four muskrats were euthanized at the end of the study, 184 days PI.

During the 30 days following inoculation in the long term study, food con-

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sumption (mean ±SD of intake units), as compared to group mean pre-inoculation values (20 ± 0 intake units considered as 100%) dropped to 93% (18.3 ± 0.6 intake units) in the control, and to 63% (12.6 ± 4.5 intake units) in the experimental group. Fourteen days after the second surgery (44 days PI) food intake dropped to 83% (16.6 ± 3.4 intake units) in the control group but increased to 73% (14.6 ± 5.6 intake units) in the experimental group. From 44 days PI both groups had a gradual increase in food intake such that by 72 days PI both groups had returned to their pre-inoculation levels of intake. Food consumption remained at this level until the end of the study.

Body weight (mean ± SD), as compared to the group mean pre-inoculation value (969 ± 134 g, considered as 100%), decreased in the control group to 98% (921 ± 90 g) in the first 30 days PI and then remained at 120% (1,075 ± 60 g) of the pre-inoculation value throughout the study. In the experimental group, the mean pre-inoculation group body weight (mean 1,179 ± 202 g, considered as 100%) declined to 83% (1,060 ± 220 g) during the first 44 days PI, then increased slowly to reach 101% at 128 days PI, but then declined progressively to be 91% (1,073 ± 189 g) of the pre-inoculation value at 184 days PI. The differences in body weights between the two groups from 58 to 184 days PI were significant ($P \leq 0.05$).

Although higher eosinophil counts and serum ALT and AST levels were present in the experimental group at 30, 44 and 58 days PI of the study, no statistically significant differences were found among the clinicopathological parameters between

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FIGURE 3. Experimental *Capillaria hepatica* infection in a muskrat at 28 days PI: (a) Periportal tract with edema, inflammation and lymphangiectasia. H&E. Bar = 150 μm. (b) Portal arteriole with medial disorganization, hyalinization, edema and endothelial hypertrophy. Plasma cells, lymphocytes and mast cells are present. H&E. Bar = 100 μm.

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the control and experimental groups. Within the higher values, the maximum (mean ± 1SD) eosinophil count, 1,200 ± 1,000/mm^3, was present 44 days PI, the highest ALT level, mean 628 ± 448 IU was present 30 days PI, and the highest AST level, mean 572 ± 337 IU was also present at 30 days PI.

Gross lesions in the four control animals that died during the experiment, included a widespread sarcomatous tumor involving most of the visceral organs and a brain abscess, in one animal each. Data from these two muskrats were excluded from the study. The two other animals died during anesthesia and had severe pulmonary congestion and edema. No significant gross or histologic lesions were present in the remaining six control animals at the time of euthanasia.

Gross lesions in all ten experimental animals included severe body wasting, marked hepatosplenomegaly, and mild peritonitis at the time of death or euthanasia. Histologic lesions present in the livers of the six animals that died between 25 and 39 days PI were similar to lesions found in experimental animals from the short-term study, but were more severe. The four animals euthanized on day 184 had firm livers with uneven surfaces; on histologic examination of these livers, there were small to large aggregates of *C. hepatica* eggs within otherwise normal liver architecture (Fig. 4). These eggs appeared mostly intact, although shrinking and basophilia of the embryo were conspicuous in some. With trichrome staining, we observed mild fibrosis around eggs. In the one animal with few eggs, these were partially mineralized, and phagocytized by giant cells within discrete granulomas (Fig. 5). The periportal lesions at 184 days PI were as in the animals from the short-term study (Fig. 5). Extrahepatic lesions were similar to the ones found in the short term study.

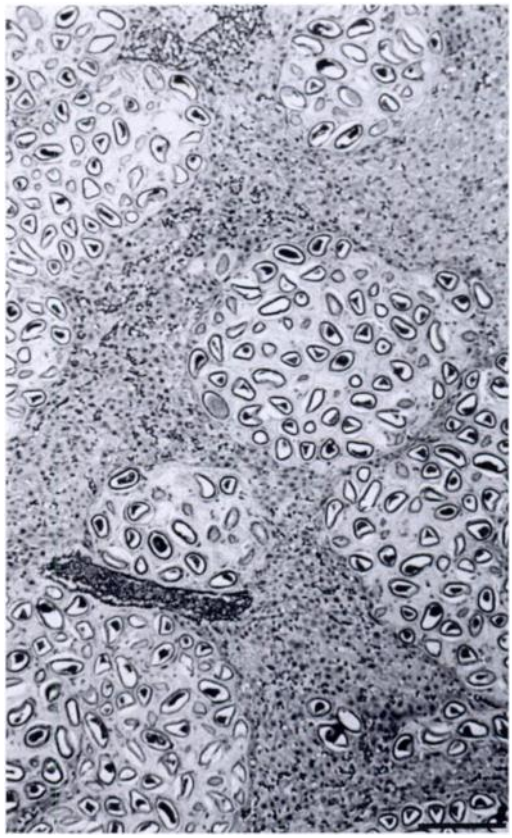


FIGURE 4. Experimental *Capillaria hepatica* infection in a muskrat at 184 days PI. Large numbers of eggs occur with no inflammation. There is extensive regeneration of liver. H&E. Bar = 150 μm.

DISCUSSION

The high mortality present in muskrats inoculated with a dose of 17,000 eggs was a surprising findings, as higher inoculation doses per kg/body weight have been reported in the literature (Vollerthun et al., 1974). Thus muskrats are more sensitive to infection than rats or mice, or the intragastric inoculation used in our study is more efficient than the per os inoculation used by other investigators.

Zahner et al. (1981) found a time-frame of 30 days PI of maximum mortality, elevated liver enzymes, and leukocytosis in *Mastomys natalensis* infected experimentally with *C. hepatica* similar to the reported here; they concluded that this acute mortality was caused by hepatic injury induced by parasite metabolites, parasite migration and oviposition, and release of inflammatory mediators by host cells. Similar clinicopathological features

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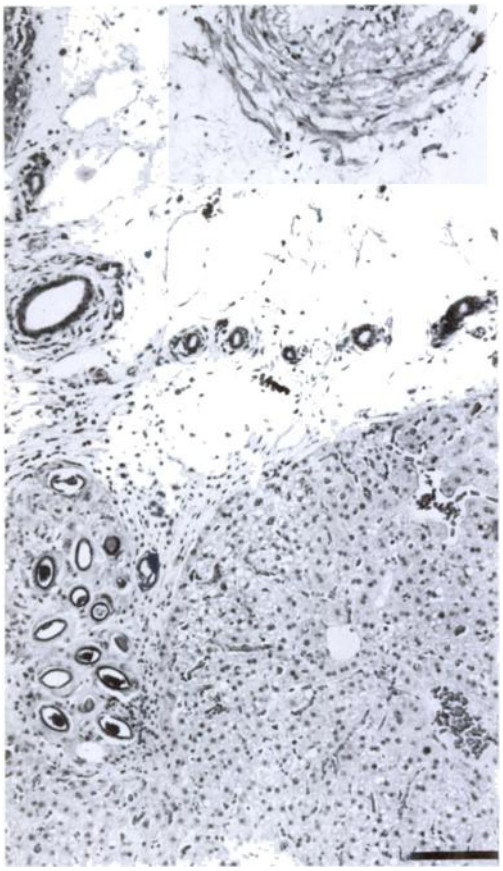


FIGURE 5. Experimental *Capillaria hepatica* infection in a muskrat at 184 days PI. Small numbers of eggs occur within granulomas. Periportal lesions are as in the acute phase of infection. H&E. Bar = 150 μm.

of hepatic capillariasis were previously reported, also from *Mastomys natalensis*, by Vollerthun et al. (1974) and from specific pathogen-free rabbits by Winkelmann (1974). In these two studies, maximum eosinophilia, AST, and ALT values occurred between 21 and 28 days PI, as they did in our study. In our long-term study, although ALT and AST levels and eosinophil counts were higher in the inoculated than in the control animals from 30 to 72 days PI, the differences were not statistically significant due to high SD values probably resulting from subclinical incidental infections found in these animals (data not shown). During the remainder of the long-term study, the values of all clinical parameters were similar in both the control and inoculated groups, and the values fluctuated around normal values reported for muskrats by Nagel and Kemble (1974). Based on the high variability of values of clinical parameters in animals from the long-term study, we conclude that laboratory evaluation of hepatic functions and WBC differential counts would not suffice to make a diagnosis of hepatic capillariasis in wild populations. Thus *C. hepatica* may be an underdiagnosed cause of several parasitic syndromes of humans and animals throughout the world including: visceral larva migrans (Kumar et al., 1985), tropical eosinophilia (Gupta and Ranhawa, 1960), and helminthic anaphylactic syndrome (Odunjo, 1970).

In both our studies studies, we failed to observe changes in levels of the remaining clinical parameters including total protein, albumin, BUN, total bilirubin, direct and indirect bilirubin, LDH, AP, HTC and Hb; this is in agreement with results reported previously for laboratory animals infected experimentally with *C. hepatica* (Vollerthun, 1972; Winkelmann, 1974).

Most of the hepatic lesions found in our study have been described in other animal species infected with *C. hepatica* (Winkelmann, 1974; Zahner et al., 1976). An immune basis for these lesions has been documented by Raybourne and Solomon (1984). The periportal inflammatory lesions described in this study for the first time in association with *C. hepatica*, are also consistent with persistent immune phenomena.

Based on the character of the hepatic lesions, muskrats, just like rats and mice, may be well adapted to infection with *C. hepatica*. In contrast, in humans, chimpanzees (*Pan satyrus*), and rabbits (*Oryctolagus cuniculus*), all of which are considered to be incidental hosts, the parasite eggs undergo mineralization and digestion by giant cells during chronic disease, and there is marked, parasite-induced hepatic fibrosis or cirrhosis (Lämmler et al., 1974; Pereira, 1983).

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A membranous glomerulonephropathy, similar to that observed in our study, was previously diagnosed as an immunoglobulin A nephropathy in one human case of hepatic capillariasis by Choe et al. (1993). At this time, the causative relationship of this lesion with hepatic capillariasis in muskrats is unknown.

Although the severity and character of hepatic lesions found in the experimental animals in our studies were similar to those reported in natural infections of muskrats, the milder infection was the most frequently observed in nature (Borucinska and Nielsen, 1993). From this we conclude that hepatic capillariasis does not by itself contribute significantly to mortality within a muskrat population but may predispose clinically sick muskrats to predation.

Because most parasite eggs within the liver of muskrats appeared viable at the time of acute mortality or chronic infection in our studies and in natural cases, their release would lead to environmental contamination with eggs of *C. hepatica*. Thus natural infection would spread and persist among muskrats, forming a reservoir of *C. hepatica*.

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