VASCULAR DAMAGE CAUSED BY Cruorifilaria tuberocauda IN THE CAPYBARA (Hydrochoerus hydrochaeris)

Authors: MORALES, G. A., GUZMÁN, V. H., and ANGEL, D.

Source: Journal of Wildlife Diseases, 14(1) : 15-21

Published By: Wildlife Disease Association

URL: https://doi.org/10.7589/0090-3558-14.1.15

VASCULAR DAMAGE CAUSED BY *Cruorifilaria tuberocauda* IN THE CAPYBARA *(Hydrochoerus hydrochaeris)*

G. A. MORALES, V. H. GUZMÁN and D. ANGEL [1]

Abstract: Infection with *Cruorifilaria tuberocauda* caused vascular damage in the kidneys, lungs and heart of the capybara *(Hydrochoerus hydrochaeris).* The most significant lesions observed were in the renal and pulmonary arteries. They consisted of villous endarteritis, intimal and medial hypertrophy of the vessel walls, and large rugose protuberances that encroached upon the lumen leading to pyramidal infarcts in the kidneys. Coronary vascular lesions were related to dead and calcified parasites.

INTRODUCTION

In the course of wildlife studies conducted at the Instituto Colombiano Agropecuario (ICA) Field station at Carimagua, State of Meta, Colombia, *Cruorifilaria tuberocauda* was recognized as a new filarial worm from the capybara *(Hydrochoerus hydrochaeris).*[2]

The purpose of this paper is to describe the vascular lesions observed in the kidneys, lungs and heart of the host.

MATERIALS AND METHODS

During October, 1972 through December, 1975, 37 capybaras were examined at necropsy. Thirty-four were infected with filarial worms in the renal arteries. The parasite also was found in pulmonary arteries of 17 animals and two had worms in the coronary vessels as well.

Tissues for histopathology were fixed in Bouin's fluid, processed routinely and stained with hemotoxylin and eosin. Selected sections were treated with Hart's elastic stain, Masson's trichrome and Prussian blue.

RESULTS

Macroscopically, tangled masses of *C. tuberocauda* were causing extensive tissue damage in the kidneys; nodular lesions were present in the lungs and myocardium.

The most significant microscopic vascular lesions were those affecting the elastic renal and pulmonary arteries. Blood vessels in the kidneys had greatly thickened walls, and the adult parasites had caused an inflammatory response characterized by villous projections growing from the inner wall of the vessels into the lumen, as if in an attempt to wall off the filariae (Fig. 1). The projections were covered by endothelium and composed of fibroblasts resting upon and continuous with the dense fibrous tissue of the intimal and medial coats.

More advanced vascular lesions were characterized by severe thickening of the vessel walls, and large rugose protuberances that encroached upon the lumen (Fig. 2). This type of lesion generally was associated with pyramidal infarcts.

Live adult nematodes were present in arteries affected by both of the foregoing types of lesions.

[1] From the Pathology Section, Animal Health Program, CIAT, Apartado Aéreo 67-13, Cali, Columbia, S.A.

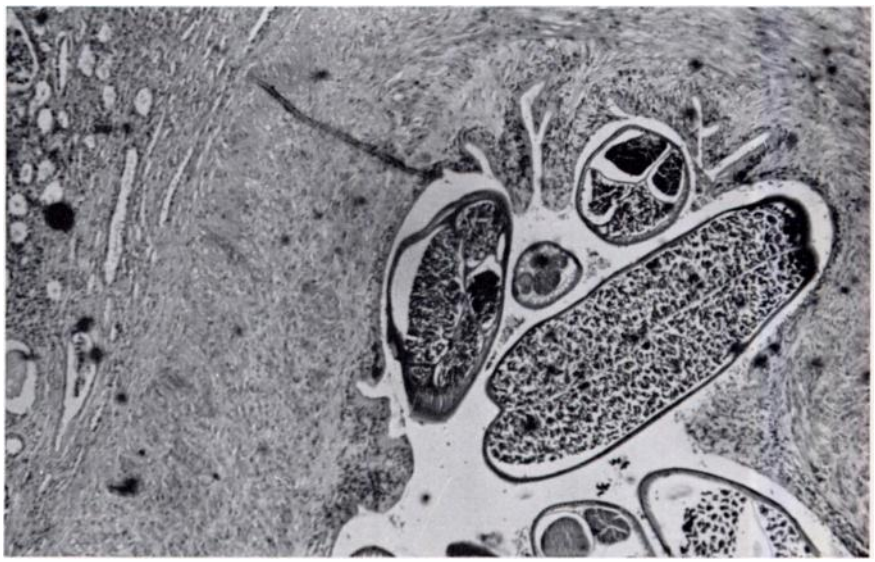

FIGURE 1. Renal artery containing filariae. Notice the villous projections growing from the inner wall into the lumen in an attempt to wall off the parasites. Intimal and medial fibrosis is marked. H&E × 50.

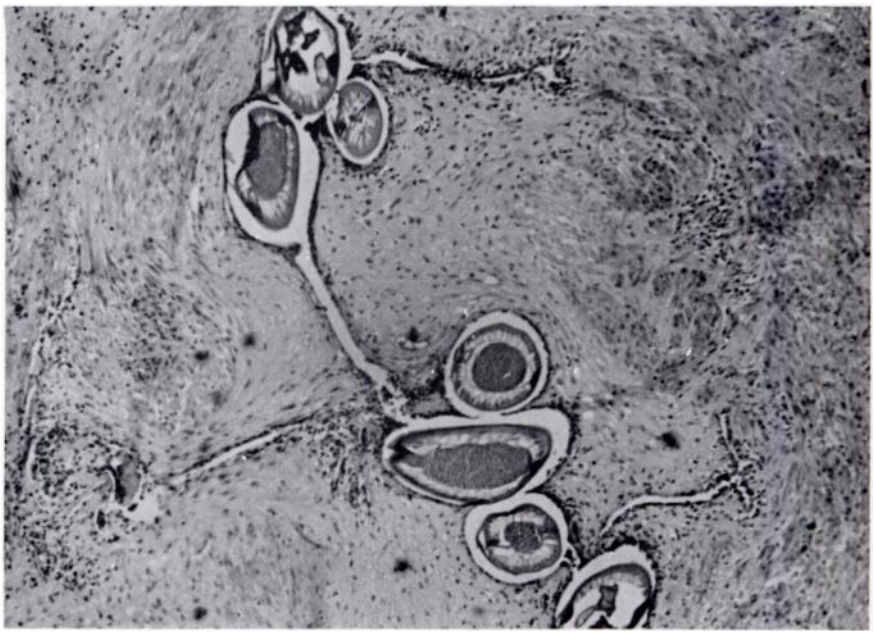

FIGURE 2. Advanced arterial obstruction resulting from rugose connective tissue protuberances encroaching upon the lumen. Filariae are not degenerated. H&E × 50.

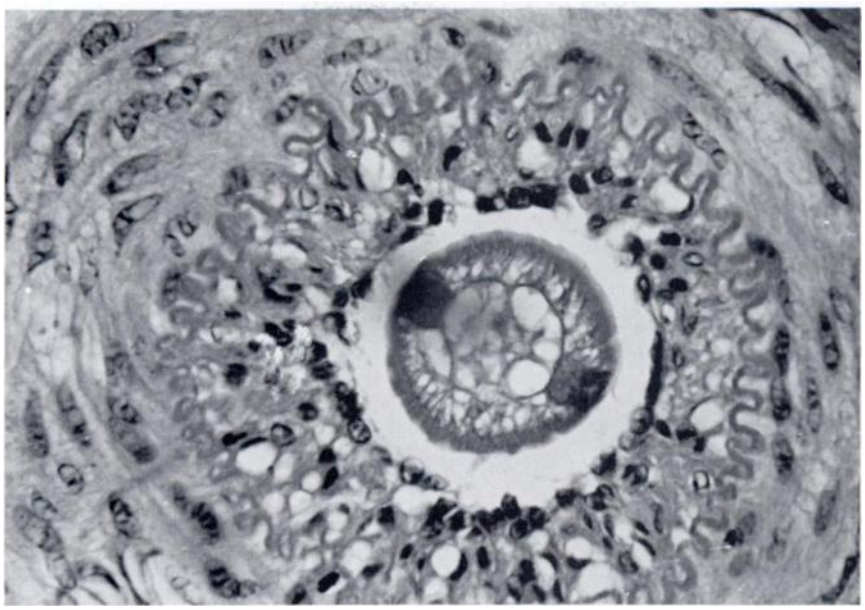

FIGURE 3. Muscular branch of renal artery. Notice proliferative response around intact filaria and medial hypertrophy. H&E × 125.

Small muscular arteries often had proliferative intimal lesions and medial hypertrophy initiated by contact of the parasite with the vessel wall (Fig. 3). A granulomatous response, extending into the kidney parenchyma, was seen only in those arteries having dead and calcified worms (Fig. 4). The inflammatory cells consisted of lymphocytes, plasma cells and eosinophils.

Vascular lesions in the lungs were very similar to those seen in the kidneys. However, the villous endarteritis and the cellular inflammatory infiltration of intimal and medial coats were more pronounced (Figs. 5 and 6). Granulomata seen at this location were also related to dead and calcified parasites. Siderocytes were seen in adjacent lung parenchyma as determined by the Prussian blue reaction.

Coronary vascular lesions in the myocardium in both cases were characterized by granulomata associated with degenerated parasites (Fig. 7).

Microfilariae occasionally were seen in glomeruli of the kidneys, vessels of the lungs and adjacent bronchial lymphnodes (Fig. 8).

DISCUSSION

The type of vascular lesions observed in capybaras are similar to those caused by *Dirofilaria immitis*.[1,7] Rugose endarteritis in canine dirofilariasis seems to stem from intimal inflammation due to contact with live nematodes, and partly from thromboemboli, but primarily from the latter. Vascular changes in small muscular arteries and arterioles are attributed to primary damage to large elastic arteries, and as a result of inflammation stimulated by microfilariae and thromboses.

While the mechanism of vascular damage in large elastic arteries of

capybaras resembles that of dirofilariasis in dogs and other animals[3], vascular lesions in the small muscular arteries in capybaras also involves intimal contacts with live nematodes, due primarily to the smaller size of the parasite[2].

The presence of *C. tuberocauda* within the arteries of the kidney is not an unique characteristic among filarioid nematodes, for there are several filarioid nematodes, including other Onchocercinae, which live in the circulatory system of the final host. These include *Brugia buckleyi, Deraiophorema evansi, Elaeophora böhmi, Elaeophora schneideri, Onchocerca armillata, Cordophilus abramovi,* and *Cordophilus sagittus.*

This study raises several questions that offer challenging opportunities for research, such as the life cycle of the parasite, its real clinicopathologic significance considering the high prevalence of infection and the extensive tissue damage produced in the kidneys of the host, and the relationship of this parasite to other animals living in the same area.

Gross and histologic lesions similar to those observed in the capybaras have been seen in the feral pig *(Sus scrofa)* but no adult filarioid nematodes have been seen or recovered.

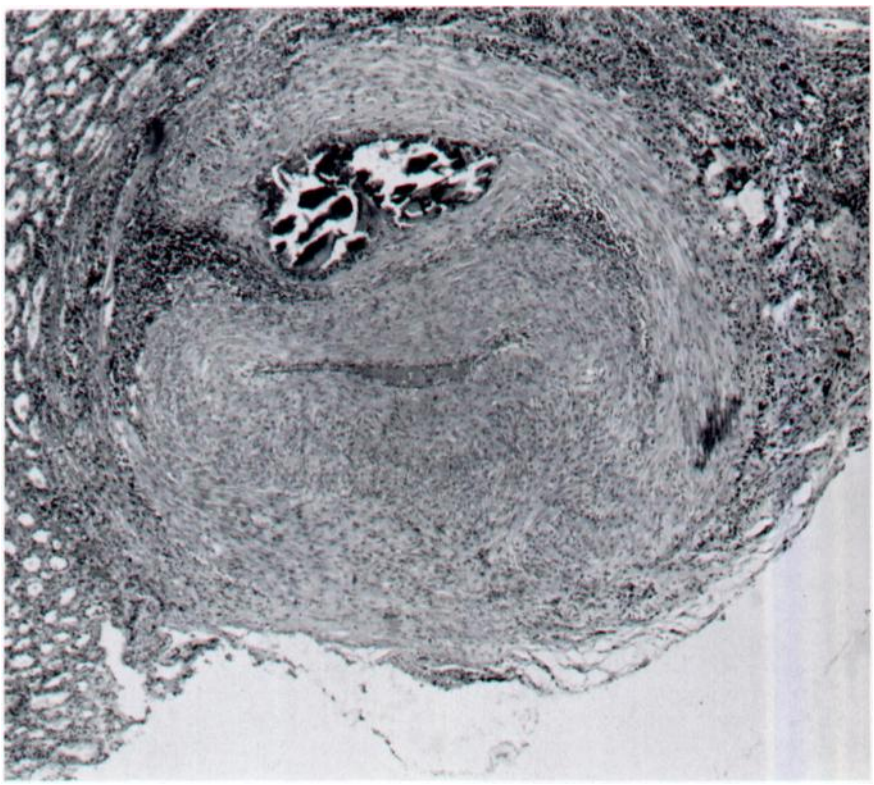

FIGURE 4. Incorporation of a calcified parasite into the arterial wall. Notice the granulomatous response extending into the kidney parenchyma and the almost complete obstruction of the vessel below. H&E × 50.

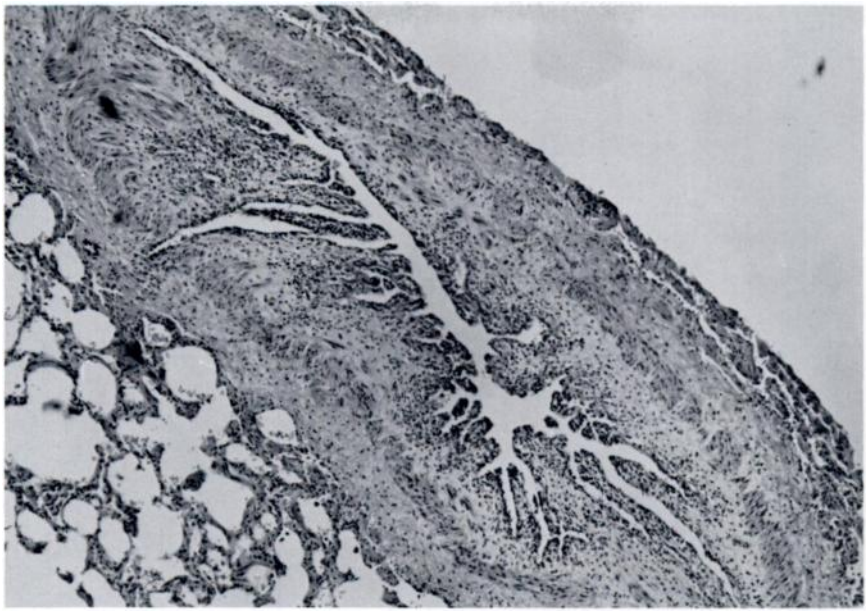

FIGURE 5. Villous intimal projections in a pulmonary artery. Intimal and medial fibrosis is marked. H&E × 50.

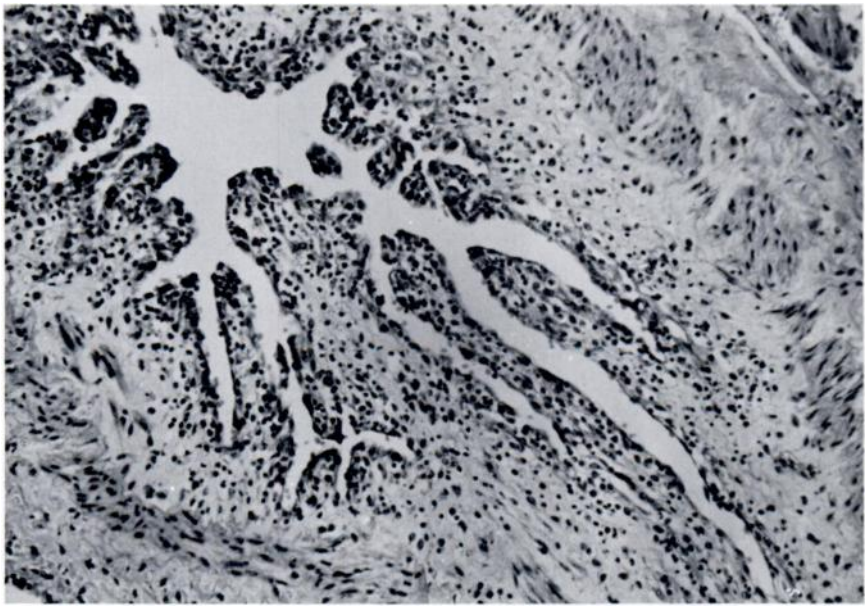

FIGURE 6. Higher magnification of portion of artery in figure 5. Notice the well defined slender projections infiltrated by inflammatory cells (lymphocytes, plasma cells and eosinophils). H&E × 100.

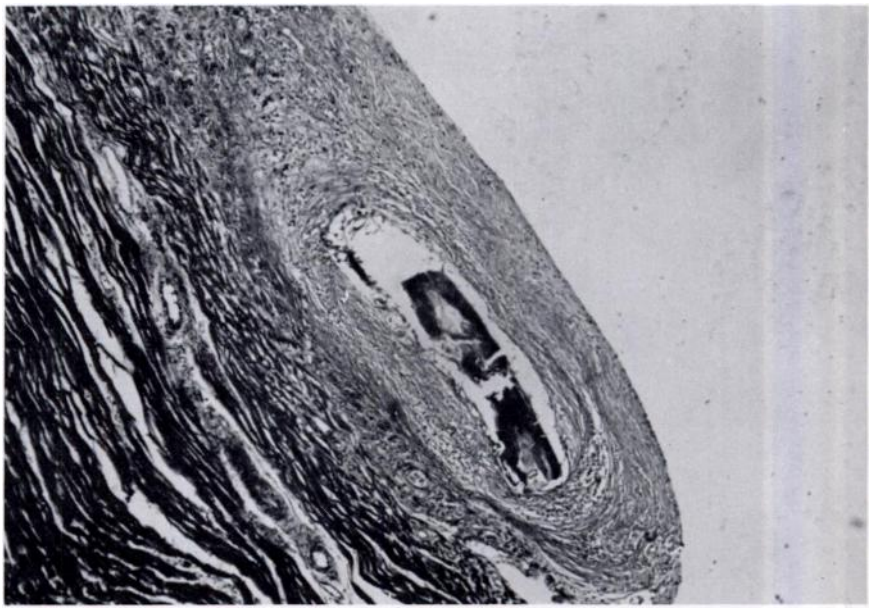

FIGURE 7. Granulomatous reaction in coronary vessel related to degenerated filaria. Notice circumferential intimal and medial fibrosis. Masson's trichrome stain. × 50.

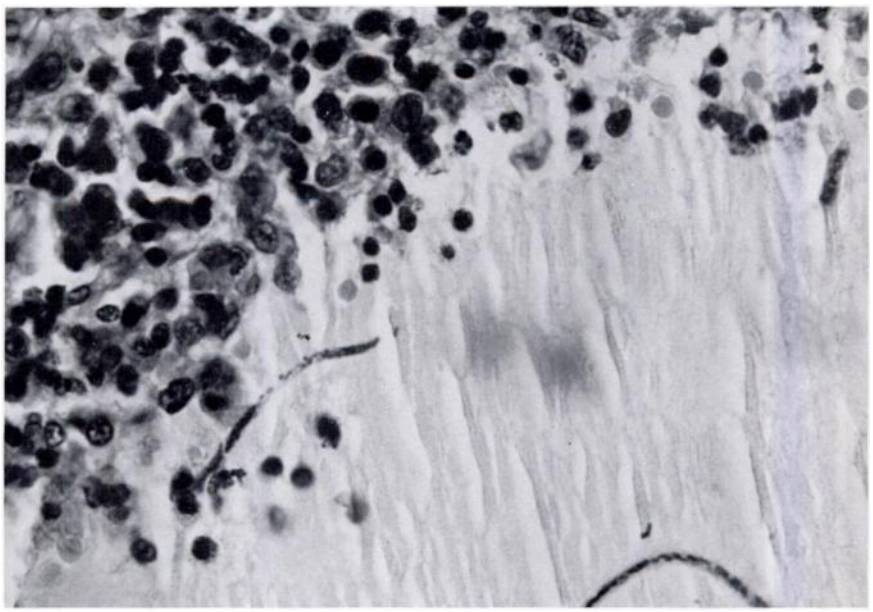

FIGURE 8. Bronchial lymphnode showing microfilariae in a central sinus. H&E × 400.

LITERATURE CITED

1. ADCOCK, J. L. 1961. Pulmonary arterial lesions in canine dirofilariasis. Am. J. Vet. Res. 22: 655-662.
2. EBERHARD, M. L., G. A. MORALES and T. C. ORIHEL. 1976. *Cruorifilaria tuberocauda* GEN. ET SP. N. (NEMATODA: FILARIOIDEA) from the capybara, *Hydrochoerus hydrochaeris*, in Colombia. J. Parasit. 62: 604-607.
3. HIRTH, R. S. and S. W. NIELSEN. 1966. Vascular lesions of *Dirofilaria immitis* in the red fox. J. Am. vet. med. Ass. 149: 915-919.
4. WINTER, H. 1959. The pathology of canine dirofilariasis. Am. J. Vet. Res. 20: 366-371.

Received for publication 14 June 1977