LESIONS IN MINK (*Mustela vison*) INFECTED WITH GIANT KIDNEY WORM (*Dioctophyma renale*)

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Abstract: Adult *Dioctophyma renale* occupied the enlarged renal pelvis of the right kidney of naturally infected mink. Lesions in the kidney parenchyma consisted of connective tissue proliferation in the interstitial tissue, tubular atrophy and fibrosis, and periglomerular fibrosis. The luminal surface of the renal pelvis wall was formed of numerous papillae covered with transitional epithelium. The nematodes in the lumen were bathed in an albuminous fluid containing red blood cells, epithelial cells and *D. renale* eggs. The left (uninfected) kidney was 60% larger than the left kidney of normal mink.

INTRODUCTION

Lesions associated with a patent infection of the giant kidney worm, *Dioctophyma renale* (Goeze 1782) have been described by few workers. McNeil[9] examined 34 naturally-infected mink, noting adult worms were restricted to the enlarged renal pelvis of the right kidney. The cortical and medullary regions of this kidney were compressed into a thin layer composed predominantly of elastic and collagenous tissue. A bony plate was in the portion of the wall nearest the backbone in about 70% of the infections.

In dogs, adult kidney worms generally are in the abdominal cavity or right kidney.[3,6] Signs of infection include anorexia, irritability[2] and convulsions.[11] Microscopic lesions associated with canine renal dioctophymiasis are similar to lesions described in mink.[9,10,12]

Material from naturally-infected mink was obtained during a recent study of the biology and life-cycle of *D. renale*.[7] This provided an opportunity to examine lesions associated with dioctophymiasis.

MATERIALS AND METHODS

Carcasses of 39 naturally-infected mink were collected from a commercial trapper. The animals had been trapped along the Black River approximately 15 km northeast of Washago, Ontario, Canada. They were shipped to the University of Guelph and examined for *D. renale*. Kidneys were removed from the carcasses, stripped of fat and capsules, and weighed.

Parts of the liver, infected and uninfected kidneys of 5 mink were excised and fixed in 10% neutral buffered formalin. These tissues were embedded, sectioned at 7 μm and stained with haematoxylin and eosin. Tissues of 3 infected kidneys containing a calcified deposit were decalcified using formic acid-sodium citrate decalcifying solutions[5] and were prepared using the histologic technique described above.

Fluid from the renal pelvis of fresh carcasses was smeared onto slides. After drying, the material was fixed with 95% methanol and stained with haematoxylin and eosin.

RESULTS

The infection was localized within the enlarged right kidney of 38 mink, 36 of which had living adult *D. renale* in the pelvis. The length of the kidney in these animals ranged from 5 to 10 cm, depending upon the number of worms present,[7]

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and outlines of adult worms were often easily discernible. The nematodes contained within the lumen were bathed in a viscous translucent fluid. The ureter was not obstructed. In five specimens the surfaces of the right lobes of the liver were uneven. These lobes felt abnormally firm when palpated. Occasionally adhesions were formed between the right lobe of the liver and the surface of the infected kidney. Other organs appeared normal.

The left kidney was enlarged and weighed about 60% more than the left kidney of an uninfected mink (Table 1). Mean diameter of glomeruli in the left kidney of an infected mink was significantly greater than the mean diameter of glomeruli in a normal kidney (Table 1. $t' = 11.0$, P 0.05).

TABLE 1. Comparison of left kidneys of wild mink trapped in the Washago region of Ontario, Canada (1971-1973) uninfected or naturally-infected with **Dioctophyma renale**.

	Uninfected	Infected
Wet-Weight (g)		
male mink	4.1 (n=5)	6.4 (n=5) (58%)[a]
female mink	2.7 (n=3)	4.4 (n=5) (63%)[a]
Mean Glomerular Diameter[b] (μm) (± 1 S.D.)	97. (± 10.)	134 (± 67.)

[a] Percent increase in mean weight.

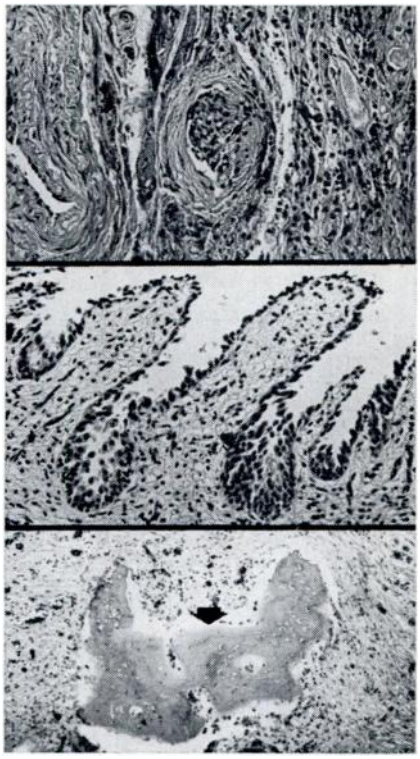
[b] One kidney from uninfected and naturally-infected male mink; fixed in 10% neutral buffered formalin, embedded in paraffin and sectioned. Random measurement of 50 glomeruli from each kidney.

The wall from the dorsal regions of the pelvis was approximately 15 mm thick and appeared to be composed of two layers. The outermost layer was a dense collagen fibre matrix within which were arterioles, arteries, veins, glomeruli and renal tubules. The renal tubules contained hyaline casts and were surrounded by concentric layers of collagen fibres and fibrocytes. Fibrocytes and fibres proliferated in and around the wall of arteries and arterioles. Normal appearing glomeruli were located at the periphery of the cyst wall. In contrast, some glomeruli had an intense periglomerular fibrosis. Proliferation of the cells of the glomerular tuft occurred in these fibrotic glomeruli, and Bowman's capsule was filled with a fibrous eosinophilic mass (Fig. 1).

The inner (luminal) layer of the pelvic wall was formed of loose fibrous connective tissue. No glomeruli or tubules were within this layer. Venules and capillaries were evenly distributed throughout the matrix. Numerous small papillae projected from the wall into the lumen (Fig. 2). Each papilla had a central fibrous core covered with a layer of transitional epithelium. Some areas of local haemorrhage were observed between papillae. Luminal fluid contained red blood cells, epithelial cells and *D. renale* eggs.

Areas of focal lymphocyte and plasma cell infiltration were in both layers of the wall. Occasionally *D. renale* eggs were trapped between connective tissue fibres within the wall. Eosinophils were present in the area immediately around the eggs.

The "staghorn" bone formation was present in 18 mink (45%) within the portion of the wall closest to the sublumbar muscles. Spicules radiated from a central plate and extended ventrad partially enclosing the enlarged pelvis. Sections of decalcified tissue revealed that the bone was within the luminal layer of the wall. The decalcified structure contained osteoblasts within lacunae. Central portions of the bone mass were basophilic and contained many osteoblasts with little interstitial matrix (Fig. 3).



FIGURES 1-3. Wall of kidney cyst which contained **Dioctophyma renale** (naturally infected mink).

1. Periglomerular fibrosis of Bowman's capsule, x 250.

2. Papillae on luminal surface of cyst wall, x 100.

3. Staghorn bone formation (arrow), x 20.

The ventral wall of the pelvis was approximately 5 mm thick and structurally different from the dorsal wall. No glomeruli or renal tubules were present. The two layers of connective tissue appeared more distinct and were separated by large arteries or veins. The luminal layer was infiltrated by numerous small arteries, veins and capillaries originating in the outer dense connective tissue layer. Foci of lymphocytes were present in the luminal layer, but eosinophils were distributed evenly throughout the connective tissue of both layers.

The right kidneys of 2 mink were shrunken, flaccid and contained cuticles of dead adult *D. renale* within the pelvic lumen. There was little luminal fluid.

One adult male (length 10.8 cm) and one adult female (length 28.2 cm) *D. renale* were free within the peritoneal cavity of one of the mink examined. Eggs removed from the uterus of the female developed normally when incubated at 25 C. The kidneys of this mink appeared normal but the mesentery and serosa of the liver were covered with a layer of soft, brown fibrous material approximately 5 mm thick. Portions of the liver and mesentery were examined histologically, and revealed *D. renale* eggs within the matrix of fibrin and fibrous connective tissue. Many eggs were surrounded by giant cells. Large lymphocytes, macrophages, fibroblasts and plasma cells were distributed throughout the fibrous mass.

DISCUSSION

Lesions in the wall of the infected kidney of mink with a patent infection of *D. renale* are similar to a chronic interstitial nephritis. Characteristically there is connective tissue infiltration of the interstitial tissue, tubular atrophy and fibrosis, thickening of Bowman's capsule and periglomerular fibrosis.

D. renale larvae within the kidney pelvis would cause partial blockage of the ureter and consequent hydronephrosis (obstructive interstitial nephritis). The pressure of this retained fluid results in

destruction of the renal pyramids, followed by reduction of the cortex. Probably, pressure on the renal artery causes partial ischaemia of the infected kidney thus increasing the rate of destruction of renal tissue. Pseudostratified epithelium of the renal pelvis would proliferate accompanying enlargement of the pelvic lumen.

The fluid within the enlarged pelvis may be recirculated through a venous backflow or through the lymphatic system.[4] This continuously filtered environment is ideal for the parasite.

The uninfected kidney of mink infected with kidney worm was about twice the size of the normal mink kidney. The diameter of the glomeruli was similarly increased. McNeil[9] observed that collecting tubules and glomeruli were enlarged, although cells lining the tubules were not hyperplastic. The uninfected kidney apparently compensates for the infected kidney; signs of uraemia are rare in animals infected with *D. renale*.[6] The form of renal compensation in mink may be similar to that observed in the rat, a combination of cellular hypertrophy and hyperplasia.[13] The mean diameter of glomeruli doubles or triples as a result of hypertrophy of the cells of the glomeruli.[8] Hyperplasia and hypertrophy of the cells lining the proximal convoluted tubules occurs, accompanying hyperplasia of the cells lining the distal convoluted tubules, ascending limbs of the loop of Henle and collecting tubules.[14]

Osseous metaplasia ("staghorn bone formation") often was observed in the wall of kidney cysts of naturally infected mink. Such metaplasia is not commonly associated with hydronephrotic kidneys of other etiology.[4] Living *D. renale* in the kidney may induce metaplasia of interstitial tissue, since, in other tissues, connective tissue metaplasia is related to an altered environment.[1]

Acknowledgements

The author wishes to express his gratitude to Prof. R. C. Anderson of the Department of Zoology, University of Guelph, who supervised this study. Mr. K. Clarke provided carcasses of naturally infected mink. The project was supported by an operating grant (R. C. A.) and a postgraduate scholarship (T. F. M.) from the National Research Council of Canada.

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Received for publication 13 February 1975