



# Diagnostic Exercise

## From The Davis-Thompson Foundation\*

Case #: **178** Month: **December** Year: **2021**

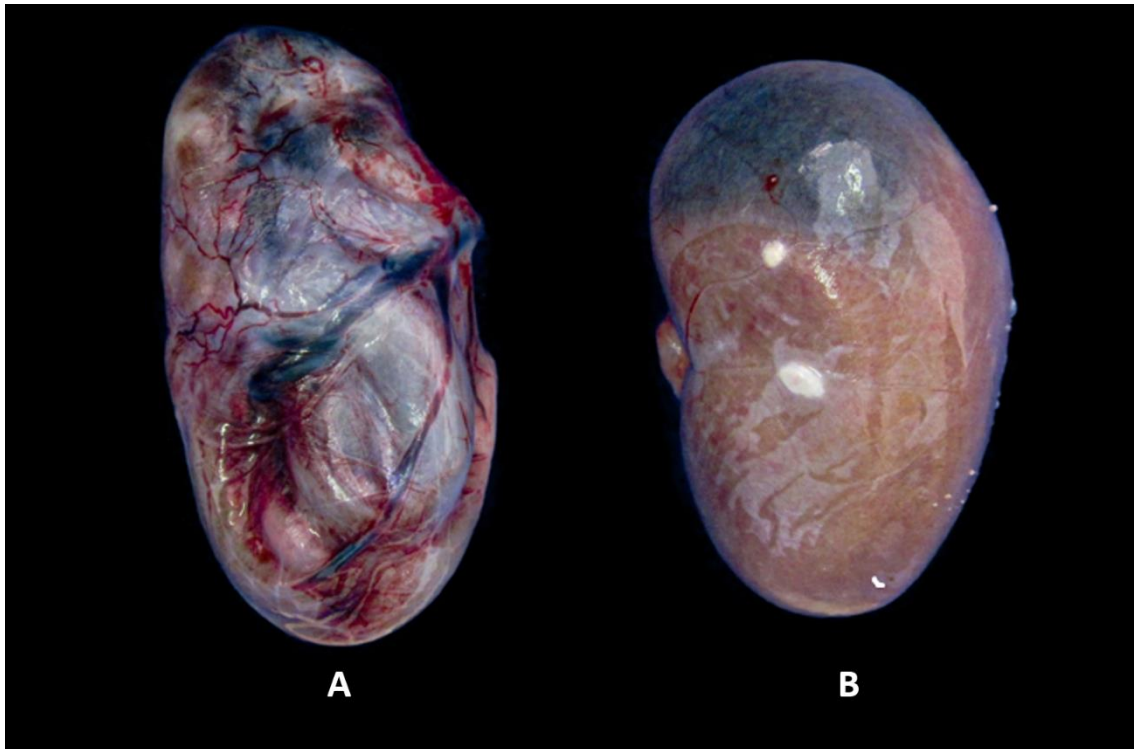
### *Answer Sheet*

**Title:** Diotophymosis in a free-ranging South American coati (*Nasua nasua*)

**Contributors:** Ticiana Brasil Ervedosa<sup>1</sup>, BVSc; Pedro Enrique Navas-Suárez<sup>1,2</sup>, BVSc, MSc, PhD candidate; Eduardo Ferreira Machado<sup>1,2</sup>, BVSc, PhD candidate; Ticiana Zwarg<sup>3</sup>, BVSc, MSc; Isis Paixão de Jesus<sup>1</sup>, BMSc, MSc; Julia de Carvalho<sup>1</sup>, BSB; Juliana Mariotti Guerra<sup>1</sup> BVSc, MSc, PhD; Natália C.C. de A. Fernandes<sup>1,2</sup>, BVSc, MSc, PhD candidate ([nccafernandes@yahoo.com.br](mailto:nccafernandes@yahoo.com.br))<sup>1</sup>Pathology Division, Adolfo Lutz Institute, São Paulo, Brazil. <sup>2</sup>Laboratory of Wildlife Comparative Pathology (LAPCOM), Department of Pathology, School of Veterinary Medicine and Animal Science, University of São Paulo. <sup>3</sup>Wildlife Division - São Paulo City Hall, São Paulo, Brazil.

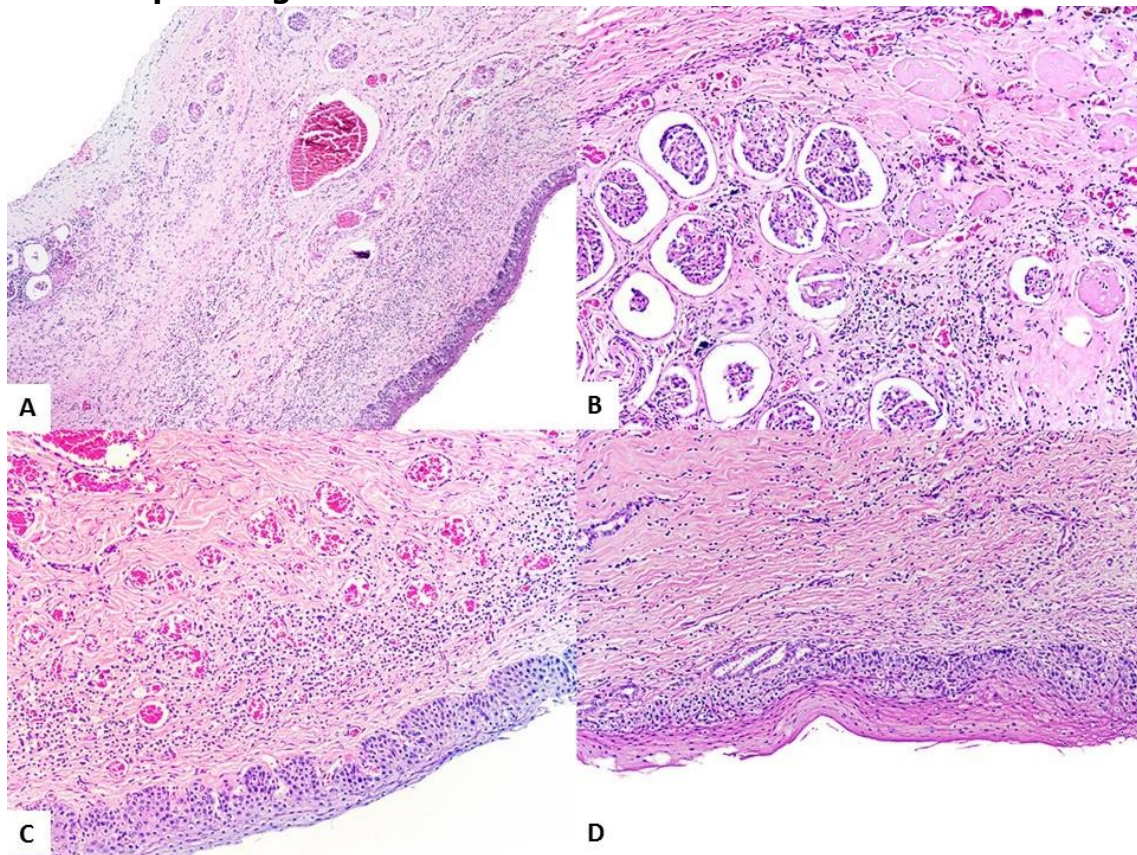
**Clinical history:** a free ranging South American Coati (*Nasua nasua*) was found in a farm, apparently injured. Clinical examination at the Wildlife Division revealed cachexia, alopecia along the dorsal neck, suggestive of scabies, secretion in the oral cavity and neurological signs (jaw paralysis, paraplegia, unresponsive to physical restraint). Due to poor prognosis, euthanasia was performed. After necropsy, tissue samples were sent to the Pathology Division of the Adolfo Lutz Institute in São Paulo, Brazil as part of a wildlife disease surveillance program.

**Gross picture:**



**Figure 1.** Right (A) and left (B) kidneys.

**Microscopic images:**



**Figure 2.** A, B, C and D: Kidney sections, H&E stain.

**Microscopic description:** Right kidney: Marked dilatation of the renal pelvis leading to distortion of the renal parenchymal architecture, characterized by corticomedullary compressive atrophy. Renal parenchyma is markedly replaced by connective tissue with loss of approximately 90% of the renal tubules and glomeruli; moderate multifocal mononuclear infiltration (lymphocytic and histiocytic) and moderate congestion. In the remaining tissue, glomeruli are compressed, concentrated, and with multifocal glomerulosclerosis. In the renal pelvis, there is diffuse squamous metaplasia and multifocal mineralization. Renal capsule is moderately thick.

**Morphologic diagnosis:**

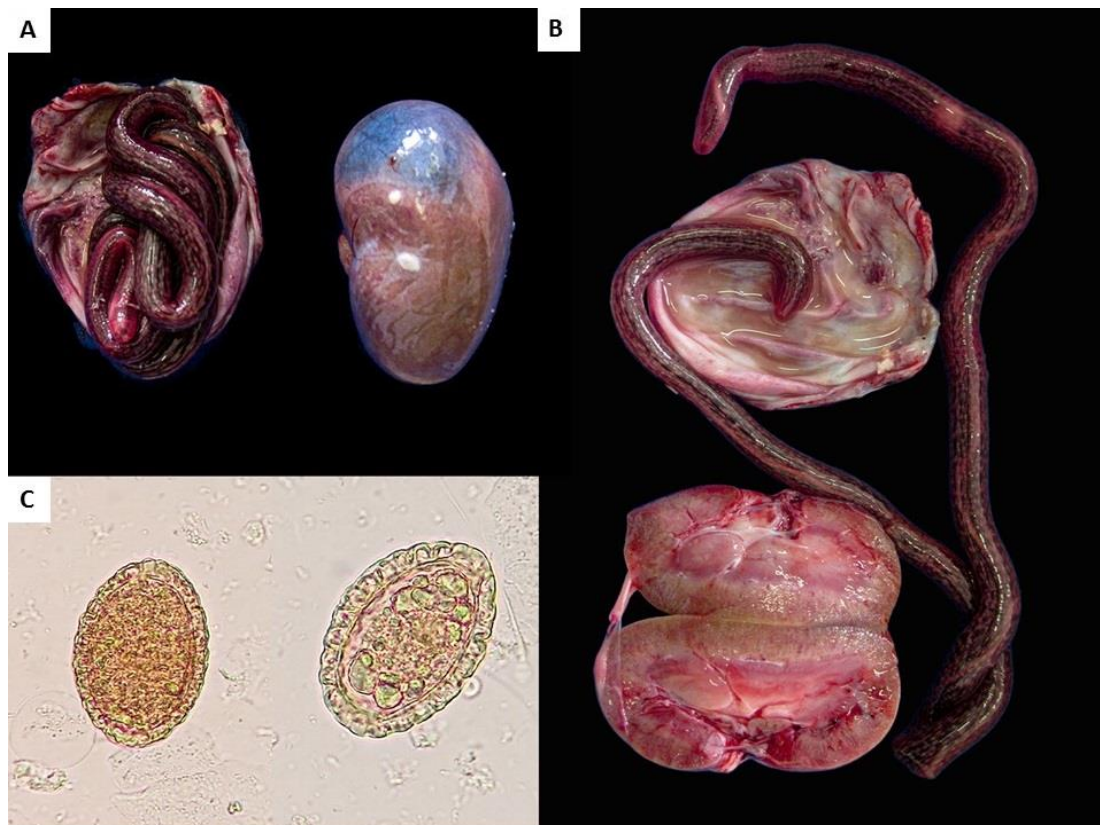
Kidney, corticomedullary compression and atrophy, chronic, diffuse, severe with fibrosis, pyelonephritis lympho-histiocytic, hydronephrosis, urothelium squamous metaplasia and mineralization, South american coati.

**Etiology:**

Giant kidney worm (*Dioctophyma renale*).

**Necropsy findings:** As an incidental finding, the right kidney had severe hydronephrosis and harbored a single red, cylindrical, 22,5cm long parasite, with morphology consistent with *Dioctophyma renale* (figure 3A and 3B). Contralateral kidney was diffusely pale, with reduced corticomedullary ratio. Urine cytology was performed (figure 3C).





**Figure 3.** A) Right kidney (on the left) with effaced parenchyma, harboring a single female nematode and contralateral kidney grossly unaffected. B) Image showing a single long nematode. C) Urinalysis: brown barrel-shaped eggs, with a thick and irregular shell.

### Discussion:

Necropsy exam revealed severe unilateral hydronephrosis in the right kidney due to nematode parasitism, consistent with *Dioctophyma renale*.

Hydronephrosis is caused by complete or partial urinary obstruction at any level from urethra to renal pelvis. The condition may be congenital or acquired, for instance, congenital due to anomalous development of the lower urinary tract or acquired due to urinary tract obstruction by calculi, urinary tract compression due to prostatic hyperplasia, neoplasm, or inflammatory processes. Depending on the site of obstruction, kidneys may be affected unilaterally or bilaterally. The pathogenesis comprises ischemic lesions secondary to the urinary obstruction, with persistent glomerular filtration, which results in increased pressure to the renal parenchyma components, tubular apoptosis, and reduced function capacity. Tubular cells are activated, and leukocytes release vasoactive factors, growth factors and cytokines, producing an interstitial inflammatory response. With persistence of this process, glomeruli decrease, and lose function and renal parenchyma progressively become replaced by fibrous tissue. The clinical significance of hydronephrosis depends upon whether it is unilateral or bilateral, the degree of obstruction and other factors. For instance, bilateral obstruction can quickly evolve to death due to rapid development of uremia. On the other hand, unilateral obstruction will lead to a high degree of

hydronephrosis, because glomerular filtration persists longer and even with unilateral kidney loss, the remaining kidney can compensate (3).

Gross findings are characterized by dilation of the pelvis and calyces, with severe parenchymal atrophy, resulting in a grossly saccular structure, with an extremely thin parenchyma. Microscopically, first lesions are dilation of the proximal convoluted tubules, followed by luminal enlargement of the other nephron segments, with tubular lining epithelial cells compression. Progressively, tubules are replaced by fibrous tissue, but glomeruli can persist longer. Depending on the type of obstruction, different degrees of ischemia and venous infarction can occur. Inflammatory response is usually mild (3).

The main kidney parasites reported in literature are metazoan *Toxocara canis* in dogs, *Stephanurus dentatus* in porcine, *Pearsonema plica* in dogs, foxes and some small carnivores, *Halicephalobus gingivalis* in horses, *Dioctophyma renale* in mustelids, canids and procyonids and protozoan *Klossiella equi* in equids. Known as giant kidney worm, metazoan *Dioctophyma renale* belongs to the class *Adenophorea*, order *Ascaridia*, family *Dioctophymatidea* and parasitizes only mammals, while *Eustrongylides* spp. parasites birds (2,5). *Dioctophyma renale* is a red nematode whose females range from 20-100cm and males from 14-45cm in length (1). It has a global distribution and has a heteroxenous life cycle involving 2 aquatic intermediate hosts. Aquatic oligochaetes ingest embryonated eggs from the environment, which will develop to first and second larval stages. The third and fourth larval stages can occur inside the worm, a fish, or few frog species. Those hosts are ingested by mammals, the definitive hosts in which infective larvae will penetrate the gut wall and migrate to the kidney. In affected species, the right kidney is often affected due to higher proximity to the stomach and duodenum (1,5). Total time spent to complete the parasite cycle takes from 3 to 6 months, and sometimes up to 2 years (3). Adult worms remain at the level of the renal pelvis, although there are reports of parasitism in body cavity, bladder, urethra, cervix, ovary, mammary gland and stomach in different animals (10; 8). Renal parenchyma is destructed by the parasite, leading from hemorrhagic to suppurative pyelitis, aggressive parenchyma compression and destruction resulting in a thin renal capsule containing the adult parasite and exudate (3).

Histologically, *Dioctophyma renale* has thick cuticle, hypodermis, coelomyarian musculature, pseudocoelomic membranes and an enlarged ventral cord. Within the body cavity, there is an extensively glandular oesophagus, with multiple nuclei, four rows of somatic intestinal muscles. There is sexual dimorphism, and males have a spicule and caudal sucker. Within the host, females lay non embryonated eggs, which will be shed in the definitive host urine and become embryonated in the environment (2). Eggs are brown, barrel-shaped, with a thick, mammillated shell, with bipolar plugs that can be seen through urinalysis (9). However, not all sections will demonstrate the presence of adult form or eggs, but the pattern of injury suggests parasite infection as a potential etiology.

Dioctophymosis is often described in mink and dogs, but there are few reports of this type of infection in humans, herbivores, and other mammals. In Brazil, this parasite was reported in wild animals such as ferrets (*Galictis cuja*), maned wolf (*Crysocyon brachyurus*), sloths (*Choloepus didactylus*), otters (*Lutra longicaudis*), bush dogs (*Speothos*

*venaticus*) and coatis (*Nasua nasua*) (6; 7). Infected animals are usually asymptomatic due to function compensation by the remaining kidney, and the injury often is a necropsy incidental finding, but diagnosis is possible through urine cytology and ultrasound exams in live animals (4). Some animals develop symptoms such as renal colic, depression, anorexia, pyrexia, and ascites, with hematuria, proteinuria, and pyuria (4). In a desexing program for coatis conducted at the Tiete Ecological Park, from 68 animals submitted to laparotomy, 51 were positive for this nematode, with 24 of them presenting adult forms free in the abdominal cavity, 17 with parasites restricted to the right kidney and 10 with parasites inside the right kidney and free in the abdominal cavity. All of them were asymptomatic, however laparotomy revealed peritonitis in these animals (7). The ring-tailed coati reported here had other significant histological lesions, such as moderate suppurative meningitis, associated with multiple thrombi and microfilariae, systemic intravascular filariasis, variably associated with thrombi and tissue inflammation and disseminated intravascular coagulation. The remaining kidney had proliferative glomerulonephritis, focally extensive tubular degeneration and multifocal acute tubular injury.

### References:

1. Anderson R.C. 2000. Nematode parasites of vertebrates: Their development and transmission. 2ed. p. 595-597.
2. Chitwood M., Lichtenfels J. 1972. Parasitological Review: Identification of Parasitic Metazoa in Tissue Sections. *Experimental Parasitology*. 32:407-519.
3. Cianciolo R.E., Mohr F.C. 2016. Urinary system. In: Maxie MG, ed. Jubb, Kennedy, and Palmer's Pathology of Domestic Animals. Vol 2. 6ed. p. 400-443.
4. Javorouski M.L., Passerino A.S.M. 2014. Carnivora - Mustelidae (Ariranha, Lontra, Irara) In: Cubas Z.S., Silva J.C.R., Catão-Dias J.L. (Eds). *Tratado de Animais Selvagens*. Vol.1. 2ed. p. 547-570.
5. Keel M.K., Terio K.A., McAloose D. 2018 *Canidae*, *Ursidae*, and *Ailuridae*. In: Terio K.A., McAloose D., St. Leger J. *Pathology of Wildlife and Zoo Animals*. P.248-249.
6. Kommers G.D., Ilha M.R.S., Barros C.S.L. 1999. Dioctofimose em cães: 16 casos. *Ciência Rural*. 29(3): 517-522
7. Milanelo L., Moreira M.B., Fitorra, L.S., Petri, B.S.S., Alves, M., Santos, A.C. 2009. Occurrence of parasitism by *Dioctophyma renale* in ring-tailed coatis (*Nasua nasua*) of the Tiete Ecological Park. *Pesquisa Veterinária Brasileira*, 29(12):959-962.
8. Measures L.N. 2001. Dioctophymatosis. In: Samuel W.M., Pybus M.J., Kocan A.A. *Parasitic Diseases of Wild Mammals*. 2ed. p. 357-364.
9. Sloss M.W., Zajac A.M., Kemp, R.L. 1999. *Parasitologia clínica veterinária*. p. 94-97.
10. Zabott M.V., Pinto S.B., Viott A.M., Tostes R.A., Bittencourt, L.H.F.B., Konell A.L., Gruchouskei L. 2012. Ocorrência de *Dioctophyma renale* em *Galictis cuja*. *Pesquisa Veterinária Brasileira*. 32(8):786-788.

**Associate Editor for this Diagnostic Exercise:** Ingeborg Langohr

**Editor-in-chief:** Claudio Barro

