may result if inappropriate materials are used. Cerebrospinal fluid should be routinely collected at necropsy in all cases where unexplained seizures or other neurologic signs are manifest because this may prove to be the critical sample needed for toxicologic or other diagnostic testing modalities. Ideally, CSF would be collected at the time of the animal’s euthanasia and submitted with the animal to the diagnostic laboratory for further analysis.

Sources and manufacturers
a. Hypaque Sodium, Winthrop Pharmaceuticals, New York, NY.
b. Waters Corporation, Milford, MA.
c. TSQ7000, Finnigan MAT, San Jose, CA.

References


Congenital polycystic kidney in a white-tailed deer (Odocoileus virginianus)

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Abstract. Polycystic kidney and liver disease was seen in a stillborn white-tailed deer (Odocoileus virginianus) fawn. Bilaterally enlarged kidneys were characterized by severe dilatation of all renal tubules. Glomeruli were sparse, small, and located within a dilated Bowman’s capsule. The liver was characterized by marked periportal fibrosis, biliary hyperplasia, and bile duct ectasia with dilated ducts containing inspissated bile. The presentation and morphology of this case are most similar to autosomal recessive polycystic disease in humans.

Renal cystic diseases comprise a heterogenous group of hereditary, developmental, and acquired disorders. In humans, polycystic kidney disease (PKD) is a significant cause of morbidity and mortality, being more common than other important hereditary disorders such as sickle cell anemia, cystic fibrosis, Huntington’s chorea, and hemophilia.1 Polycystic kidney disease is characterized by progressive enlargement of the kidneys because of numerous expansile cysts and ultimately leads to renal failure. In humans, PKD is heritable and recognized in at least 2 genetically distinguishable forms: infantile or autosomal recessive PKD (ARPKD) and adult or autosomal dominant PKD (ADPKD). The autosomal dominant form is slowly progressive, often associated with a variety of extrarenal manifestations and usually leads to death from renal failure in late adulthood. The autosomal recessive form is rare, often diagnosed in early infancy by massive nephromegaly, and is rapidly progressive.2,3 Syndromes resembling both the recessive and dominant forms of human PKD have been recognized in animals including cats,1,2 where Persian cats appear disproportionately affected,2 dogs,9,15 mice,13 pigs, raccoons, and ruminants such as cattle,14 goats,7,10 sheep,3 and springbok (Antidorcas marsupialis).6 To the authors’ knowledge, PKD has not been reported in any member of the family Cervidae.

Twin full-term fawns were born to a 2.5-year-old white-tailed deer (Odocoileus virginianus). One fawn was stillborn, whereas the other appeared healthy. Grossly, the stillborn fawn had marked abdominal distention because of marked bilateral nephromegaly. Enlarged kidneys retained their reniform shape but were pale, tan, smooth, and 8.8 × 6.0 cm in size. The capsule was thin, tightly adherent, and translucent, through which could be seen numerous fluid-filled cysts. On cut section, there were numerous 1–5 mm, round to fusiform cysts that contained clear fluid. There was no clear distinction between cortex and medulla because cysts were diffusely distributed throughout both cortex and medulla (Fig. 1). The ureters and
bladder were grossly normal. The surface of the liver was irregular, covered by coalescent raised, pale, tan plaques. On cut surface, the intrahepatic bile ducts were variably ectatic with irregular outlines and contained bile. Gross lesions were not seen in other organs. Samples of kidney, liver, pancreas, spleen, heart, and lung were fixed by immersion in neutral-buffered 10% formalin and processed routinely for microscopic analysis.

Microscopically, there was severe dilatation of all renal tubules. The corticomedullary junction was obscured. Dilated tubules were lined by low cuboidal to flattened squamous epithelium (Fig. 2). Little normal renal parenchyma was present interspersed among dilated tubules. Most dilated tubules were empty, but some contained a homogenous or flocculent eosinophilic material. Dilated tubules were occasionally separated by variable amounts of expanded, loose interstitial tissue. The number of glomeruli was greatly reduced. Those present were small and located within a dilated Bowman’s capsule. The liver was characterized by marked biliary hyperplasia. Bile ducts were dilated to 1–5 mm in diameter, irregular in contour, and the lumina of many ducts contained inspissated bile. Irregularly shaped bile ducts were surrounded by variable amounts of an expanded loose fibrous connective tissue that appeared mesenchymal in nature and stained blue with the Massons trichrome staining method, consistent with collagen (Fig. 3). This expanded connective tissue extended from superficial bile ducts to the capsular surface, where the expanded tissue gave the capsular surface an irregular contour. Microscopic lesions were not noted in other organs examined.

In domestic animals, PKD is most often consistent with the human ARPKD in that the disease manifests as stillbirths or death within the first few weeks of life, although manifestations consistent with the ADPKD have also been described. Reported extrarenal manifestations of PKD in animals include biliary and pancreatic cysts.\(^{2,6,7,9,10,15}\) Pancreatic lesions were not observed in the present case.

This case of PKD in a white-tailed deer is most
consistent with ARPKD. In humans, ARPKD is generally seen in the neonate and presents as bilaterally symmetrical nephromegaly that is invariably associated with generalized portal and interlobular hepatic fibrosis and biliary hyperplasia. Many humans with ARPKD have been found to have mutations in the gene denoted as polycystic kidney and hepatic disease-1. The longest continuous open reading frame of this gene is predicted to code for a protein that is known by 2 different names, fibrocystin and polyductin. This protein is expressed on adult and fetal kidney, liver, and pancreas and may be a receptor protein that plays a role in collecting duct and bile duct differentiation. The basic defect in ARPKD may therefore be a failure of terminal differentiation in collecting and bile ducts. Polycystic kidney and hepatic disease-1 gene products are members of a novel class of proteins that share structural features with hepatocyte growth factor receptor and plexins, members of a class of proteins involved in the regulation of cell proliferation, cellular adhesion, and repulsion.

The doe in the present study had delivered a normal singleton fawn the previous year. The twin to the affected fawn described in this report appeared healthy at birth and continued to develop normally without clinical signs consistent with renal disease. Disease in only 1 twin is not unexpected because white-tailed deer ovulate 1–5 ova; therefore, twins or triplets do not arise from the same embryo. The role played by specific genetic mutations in white-tailed deer with PKD, similar to those seen in ARPKD of humans, remains to be determined. Genetic factors may be involved in congenital PKD of Cairn Terriers, and Persian cats because the condition has been described in groups of related animals.

References