Renal Dysplasia in Dogs

By Vetbook

enal dysplasia is an autosomal-dominant multifactorial genetic renal disease of dogs characterized by delayed maturation of renal and other parenchymal tissue, leading to progressive renal degeneration, protein-losing nephropathy and chronic renal disease.

This disease is usually an inherited syndrome but

congenitally-acquired neonatal urinary tract infections, viruses and vitamin A deficiency may result in a phenotypicallyidentical presentation of arrested or delayed nephric development.

An autosomal-recessive genetic predisposition has been noted in the Soft Coated Wheaten Terrier^[2].

Because of the typically adult-onset nature of this condition and the multiorgan involvement, it can be confused with nonhereditary causes, and has few consistent clinical signs

Renal dysplasia in a dog, showing characteristic a large, normally developed glomerulus, and a small fetal glomerulus with a dilated Bowman's capsule (arrow)^[1]

hereditary causes, and has few consistent clinical signs.

The age of onset and clinical disease symptoms are highly

variable, and kidney failure from this birth defect can occur

at any age, even in dogs that are up to 10 years of age or

Clinically affected dogs are often middle-aged dogs of any breed which present with weight loss and extreme polyuria and polydipsia. The extreme polyuria and polydipsia seen in younger dogs (under 6 months of age), is thought to be caused by lack of response by immature renal tubules to antidiuretic hormone activity^[3].

Renal biopsy confirms the histological presence of immature fetal glomeruli, persistent primitive mesenchyme and metanephric ducts, asynchronous differentiation of nephrons, and atypical tubular epithelium^[4].

Blood tests usually reveal non-regenerative anemia, hyperammonemia, hypercreatinemia and hyperphosphatemia^[5]. Urinalysis may reveal concurrent pyelonephritis^[6]. Hyperfibrinogenemia is a consistent finding, and these dogs have a higher risk of thromboembolism^[7].

Ultrasonography often features poor corticomedullary definition, multifocal hyperechoic speckles in the renal medulla or a diffusely hyperechoic medulla^[1].

Diagnosis requires renal biopsy, as no mutation-based DNA test is available. A surgical wedge biopsy is the only reliable diagnostic method, as approximately 100 glomeruli have to be evaluated, and the architecture of the renal cortex has to be assessed^[8]. Histopathology usually demonstrate abnormal or asynchronous differentiation of renal tissue, with the presence of immature or fetal glomeruli and mesenchymal tissue in the medulla^[9]. Other histological features include mineralization of renal tubules and diffuse interstitial fibrosis in the cortex and medulla^[10].

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beyond.

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Concurrent genetic abnormalities have been reported in dogs with pre-existing renal dysplasia, including urolithiasis, ectopic ureter^[11], renal agenesis, hepatic capsular fibrosis, lymphatic duplication of connective tissue, pulmonary calcification^[12] and half-circular tracheal rings^[13].

A differential diagnosis would include psychogenic polydipsia, diabetes insipidus, hyperadrenocorticism, hypoadrenocorticism, other causes of chronic renal disease disease such as polycystic kidney disease, chronic pyelonephritis, *Leptospira spp*, as well as congenital disorders such as renal agenesis or hypoplasia (Ask-Upmark kidney)^[14].

There is no specific treatment for this disease, and in clinically affected dogs, renal degeneration continues to end-stage renal disease. Poor quality of life factors usually dictates renal transplantation or euthanasia.

Renal transplantation may be the only viable curative procedure for this condition^[15].

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