Lake Shore Pet Hospital

Glomerulonephritis (Kidney Inflammation Involving the Glomerulus, the "Blood Filter")

Basics

OVERVIEW

- The kidney filters the blood and removes various waste products from the body as it produces urine; the kidney also is involved in maintaining the normal fluid volume of the body; each kidney is composed of thousands of nephrons (the functional units of the kidney, each consisting of the glomerulus [a tuft of blood capillaries—the "blood filter"] and a series of tubes and ducts, through which the filtered fluid flows, as urine is produced)
- Glomerulonephritis is inflammation and accompanying dysfunction of glomeruli (plural of glomerulus); most commonly due to the presence of immune complexes in the glomerulus with or without inflammatory cell infiltration
- In veterinary medicine, "glomerulonephritis" has been used as an "umbrella" term for all glomerular diseases; however, certain glomerular diseases (membranous glomerulopathy; kidney amyloidosis [condition in which insoluble proteins, amyloid, are deposited outside the cells in the kidney, compromising its normal function]; and most inherited kidney diseases) are not truly types of "glomerulonephritis," as the primary lesion is not due to inflammation of the glomerulus

GENETICS

- Familial glomerular disease (glomerular disease that runs in certain families of animals) has been reported in Bernese mountain dogs, Brittany spaniels, bull terriers, French mastiffs, Dalmatians, Samoyeds, Doberman pinschers, cocker spaniels, Newfoundlands, Rottweilers, Pembroke Welsh corgis, beagles, and soft-coated Wheaten terriers

SIGNALMENT/DESCRIPTION OF PET

Species

- Dogs
- Cats—less common

Breed Predilections

- See "Genetics"
- Labrador and golden retrievers appear to be more likely to develop glomerulonephritis; sudden (acute) death of cells and tissue of the kidney tubules (known as "tubular necrosis"); and interstitial inflammation associated with Lyme disease (*Borrelia burgdorferi* infection)

Mean Age and Range

- Dogs—mean age, 6.5–8.5 years; range, 0.8–17 years
- Dogs with kidney inflammation due to a genetic abnormality (known as “hereditary nephritis”) usually have changes in the kidney at very young ages, and may develop protein in the urine (known as “proteinuria”) before 6 months of age
- Cats—mean age, 4 years

Predominant Sex

- Dogs—no difference between males and females
- Cats—75% are males

SIGNS/OBSERVED CHANGES IN THE PET

- Signs depend on severity of kidney failure
- Significant proteinuria (protein in the urine) often is discovered on yearly health screens or while evaluating other problems
- Occasionally, signs associated with an underlying infection, inflammation, or cancer are the reasons why owners seek veterinary care
- If protein loss into the urine is mild-to-moderate, dogs usually are asymptomatic (that is, show no signs of kidney disease); however, non-specific signs may include weight loss and sluggishness (lethargy)
- If protein loss into the urine is severe, (in which levels of albumin [a type of protein] in the blood drop to less than 1-1.5 g/dL), fluid buildup (known as “edema”) in body tissues and/or fluid buildup in the abdomen (known as “ascites”) often occurs; albumin normally plays a major role in holding fluid within the blood/circulation—when the levels of albumin drop to less than 1-1.5 g/dL, fluids move from the circulation into surrounding tissues, leading to edema or ascites
- If the disease has progressed to kidney failure, excessive urination (known as “polyuria”), excessive thirst (known as “polydipsia”), lack of appetite (known as “anorexia”), nausea, and vomiting may occur
- Sudden (acute) difficulty breathing (known as “dyspnea”) or severe panting in dogs may be caused by blood clots in the lungs (known as “pulmonary thromboembolism”), an uncommon development, which occurs in association with moderate-to-severe low levels of albumin in the blood (known as “hypoalbuminemia,” with serum albumin concentration of less than 2-2.5 g/dL)
- Sudden (acute) blindness may occur due to bleeding in the retina or back part of the eye (known as “retinal hemorrhage”) or to detachment of the retina; may be associated with systemic high blood pressure (hypertension)

CAUSES

- Several infectious and inflammatory diseases have been associated with deposits or formation of immune complexes in the glomerulus. In many cases, no antigen source or underlying disease process is identified, so the glomerulonephritis is considered to be “idiopathic” (meaning that no cause is known). The following diseases have been associated with glomerulonephritis:
  - Dogs—infectious disease (such as, infectious canine hepatitis [viral inflammation of the liver]; bacterial endocarditis [bacterial infection/inflammation of the lining of the heart]; brucellosis [infection caused by *Brucella canis*]; dirofilariasis [heartworm infection]; ehrlichiosis [infection caused by *Ehrlichia*]; leishmaniasis [infection caused by *Leishmania*]; pyometra [inflammation/infection of the uterus]; borreliosis [infection caused by *Borrelia burgdorferi*, also known as “Lyme disease”]; any chronic bacterial infection)
  - Cancer
  - Inflammatory diseases (such as systemic lupus erythematosus)
  - Endocrine or hormonal diseases (such as excessive production of steroids by the adrenal glands [known as “hyperadrenocorticism” or “Cushing's syndrome”]; diabetes mellitus
  - Long-term administration of steroids)
  - Inherited kidney disorders
  - Miscellaneous causes (such as medications; for example, sulfonamides)
  - Cats—infectious disease (such as feline leukemia virus [FeLV] infection; feline infectious peritonitis [FIP]; feline immunodeficiency virus [FIV] infection; and *Mycoplasma*-caused inflammation of several joints [known as “polyarthritis']); cancer

- True “autoimmune” glomerulonephritis in which antibodies are directed against the kidney is rarely documented in dogs and cats

Treatment

HEALTH CARE

- Most pets can be treated as outpatients; exceptions include pets that have very high levels of urea and other nitrogenous waste products in the blood (condition known as “uremia” or “azotemia”) and/or high blood pressure (known as “hypertension”); pets with blood-clotting disease (known as “thromboembolic disease”); and pets with fluid buildup in the space between their lungs and chest wall (known as “pleural effusion”) or fluid-build up in the lungs (known as “pulmonary edema”) secondary to low levels of albumin (a protein) in the blood (hypoalbuminemia)
- Since most glomerular diseases are caused by immune mechanisms, the most specific and effective therapy is elimination of the source of antigenic stimulation (that is, the substance to which the immune system is responding and producing antibodies); often this is difficult, because the disease process or antigen source is not identified or is impossible to eliminate (such as cancer)

ACTIVITY

- Restrictions usually are not necessary
- Pets with severely low levels of albumin in the blood (hypoalbuminia) may benefit from exercise restriction, because of the possibility of developing blood clots (thromboembolic disease)

DIET

- Sodium-reduced, high-quality, low-quantity protein diets; many commercially manufactured prescription “renal or kidney diets” meet these criteria

Medications

Medications presented in this section are intended to provide general information about possible treatment. The treatment for a particular condition may evolve as medical advances are made; therefore, the medications should not be considered as all inclusive

- Angiotensin-converting enzyme (ACE) inhibitors, specifically enalapril, decrease loss of protein into the urine (proteinuria); the decrease in protein loss may slow the progression of kidney disease in dogs with idiopathic glomerulonephritis (glomerulonephritis of unknown cause), because protein itself is directly toxic to kidney tubules
- ACE-inhibitor therapy will be initiated at the time of diagnosis, unless severely high levels of urea and other nitrogenous waste products in the blood (known as “uremia” or “azotemia”) are present
- Although glomerulonephritis has an immune basis, no controlled clinical trials in veterinary medicine have demonstrated any benefit from drugs designed to suppress the immune response (known as “immunosuppressive therapy”); on the contrary, steroids and cyclosporine (immunosuppressive drugs) have been shown to worsen prognosis in many pets
- Low-dose aspirin decreases production of thromboxane, a major cause of glomerular inflammation, and decreases platelet clumping and resultant blood-clotting disease (known as “thromboembolic disease”); low-dose aspirin therapy usually is initiated once serum albumin is below 2.2-2.5 g/dL; use aspirin only as directed by your pet's veterinarian

Follow-Up Care

PATIENT MONITORING

- Follow the urine protein:creatinine (UP/C) ratio closely to determine progression or remission of glomerular disease (known as “glomerulopathy”)
- Magnitude of protein loss into the urine (proteinuria) will decrease as more nephrons (the functional units of the kidney) are lost to progressive disease; therefore, the veterinarian will interpret changes in the urine UP/C ratio in light of changes in serum creatinine concentration and urine specific gravity (measurement of the dissolved substances in a solution, in this case urine; it is used to evaluate the ability of the kidney tubules to remove water from [that is, concentrate] or add water to [that is, dilute] the urine)

- The veterinarian will monitor bloodwork (such as serum urea nitrogen, creatinine, albumin, and electrolyte concentrations), blood pressure, and body weight
- Ideally, a reexamination schedule in stable pets will be at 1, 3, 6, 9, and 12 months after initiation of treatment

PREVENTIONS AND AVOIDANCE

- Do not use affected pets of breeds with suspected familial glomerular disease for breeding purposes

POSSIBLE COMPLICATIONS

- Nephrotic syndrome (a medical condition in which the pet has protein in its urine, low levels of albumin [a type of protein] and high levels of cholesterol in its blood, and fluid accumulation in the abdomen, chest, and/or under the skin)
- High blood pressure (hypertension)
- Long-term (chronic) kidney failure
- Blood-clotting disorders (thromboembolic disorders)

EXPECTED COURSE AND PROGNOSIS

- Long-term prognosis is guarded to poor
- Often progresses to chronic kidney failure, despite treatment

Key Points

- If the underlying cause cannot be identified and corrected, the disease often is progressive, resulting in chronic kidney failure
- Once azotemia (high levels of urea and other nitrogenous waste products in the blood; also known as “uremia”) and kidney failure develop, prognosis often is poor due to rapidly progressive disease

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