Glomerulonephritis in the Canine

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is then sutured with 5-0 or 6-0 silk with an atraumatic swaged-on needle using an over and over suture pattern.

Repair of an irregular laceration or badly damaged vessel is slightly more complicated. The vessel is clamped proximally and distally as before. Then the damaged vessel segment is debrided or excised from both ends of the artery. The artery ends are then approximated with the bulldog forceps and two separate suture lines are started at opposite points on the vessel. The two sutures are then worked around the vessel and meet the other suture line. The over and over suture pattern is used and the sutures are place 1–3 mm. from the edge of the vessel and 1–2 mm. apart.1

Postoperative care

Postoperative care should include systemic antibiotic therapy and a very close monitoring of the animal. The animal should be watched for any signs that a thrombus has formed in the repaired vessel. If this occurs the same clinical signs that were seen before repair or surgery will again reoccur. These signs include pain to the area, coldness of the area supplied by the artery, loss of function of the limb, absence of an expected pulse in the artery distal to the repair, and also any indication of a hematoma forming proximal to the repair site.

ACKNOWLEDGEMENTS

The actual surgery was performed at Wilmington Veterinary Hospital, Wilmington, Massachusetts, by Dr. Steven C. Draheim, and assisted by Paul R. Wade. The author gratefully acknowledges Dr. R. L. Grier for his valuable assistance in preparing the text.

REFERENCES


Glomerulonephritis in the Canine

(A Case Report)

by

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Robert W. Carithers, D.V.M., Ph.D.†

Summary

The case report given shows some of the characteristic clinical and histopathological signs of glomerulonephritis. The clinical signs include proteinuria, elevated blood urea nitrogen and creatinine levels.

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The histopathology of the kidney reveals glomerular tuft proliferation, hyaline formation, and Bowman’s capsule adhesions. The differentiation of the various renal disorders can be made early in their clinical course by renal biopsy with some degree of prognostication possible at that time. As the renal disease progresses toward chronicity the differentiation becomes more difficult and prognosis graver. In all cases symptomatic treatment is all that is available.
**Introduction**

Glomerulonephritis is classically described as a renal condition characterized clinically by an acute phase of hematuria, proteinuria and oliguria followed by transient edema, azotemia and hypertension. In man the acute phase is usually preceded by a streptococcal Type A infection and is thought to result from a delayed antigen-antibody reaction localizing in the glomerulus. The hematuria and proteinuria may persist for a variable length of time—ranging from several weeks to years. In the latter case the usual course is a progressive renal impairment with systemic hypertension and eventual death.

In the dog most of the reported work has dealt with experimentally induced glomerulonephritis as a result of an immunological, bacterial or chemical procedure, or as a secondary manifestation of some other illness. Examples of this being lupus erythematosis and pyometra. The following case report represents a typical glomerular response to injury and could have resulted from any one of a number of stimuli.

**Glomerulonephritis in the canine**

Clinical. Case No. 72-C-0200

**History and physical exam**

A 5-year old 36 lb. male Dachshund was admitted to the Iowa State University Veterinary Clinic on January 30, 1972 with a history of uncontrolled vomiting since January 25, 1972. The dog was unable to retain food or water for longer than 10 minutes after ingestion. Signs of diarrhea were also reported by the owner. The bronchial sounds were louder than normal, and no abnormal heart sounds were heard. Some respiratory dyspnea and dehydration were present. The pulse rate was 100 beats per minute. The sclera were slightly injected and the dog's rectal temperature was 99.8°F.

The dog was receiving treatment for acanthosis nigricans.

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<th>Clinical pathology</th>
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Thoracic and abdominal radiographs showed an enlarged cardiac silhouette along with an enlarged liver and prostate. EKG findings indicated left cardiac enlargement. 350 cc of 5% dextrose with bicarbonate was administered intravenously. An antibiotic* was given intramuscularly. Antiemetic** and antiarrheal† agents were given subcutaneously. This regimen of treatment was repeated the following day with the addition of a bronchial smooth muscle relaxant‡ intramuscularly. The persistence of the dyspnea, the elevated blood urea nitrogen and creatinine values, and cardiac enlargement offered a poor prognosis. Euthanasia was performed on 2/1/72.

* Combiotic. Chas Pfizer and Co., Inc. New York, New York.
** Darbucine. Norden Laboratories, Lincoln, Nebraska.
† Biosol—M. Upjohn Company, Kalamazoo, Michigan.
‡ Aminophylline injection. Torigian Laboratories Inc. Queens Village, New York.
**Necropsy Findings**

The right atrioventricular valve had severe mucoid degeneration. There were several 1–2 mm. yellow colored foci on the surface of the pulmonary artery, 1–2 mm. ulcers were present on the gastric and duodenal mucosa. The kidneys were mildly enlarged with cortical surface mottling. The prostate was enlarged 2 times normal and contained gray areas of discoloration. The lungs were turgid and congested.

**Histopathology**

Many renal tubules were filled with proteinaceous fluid and contained swollen epithelial cells whose cytoplasm stained as containing eosinophilic particles and brown granular pigment. The glomeruli were swollen and dense with glomerular tuft thickening and adhesions between the parietal and visceral layers of Bowman’s capsules. A few areas of interstitial fibrosis with lymphocytes and plasma cells were seen. Pulmonary artery thrombosis and alveolar accumulations of fibrin and red blood cells were found. The liver contained areas of central lobular congestion with central vein wall thickening. The prostate was hyperplastic with glandular dilation and interstitial fibrosis. Edema was present in the adrenals. The thyroid appeared quite cellular with very little colloid present.

The diagnosis was glomerulonephritis, acanthosis nigricans, pulmonary artery thrombosis, prostatic hyperplasia, and thyroid exhaustion atrophy.

**Differential diagnosis**

The presence of proteinuria, an elevated blood area nitrogen and creatinine level is indicative of renal involvement. This clinical picture might easily fit with amyloidosis, primary or secondary glomerulonephritis, chronic pyelonephritis, or chronic interstitial nephritis. All of these syndromes are characterized by some degree of hypoproteinemia, proteinuria, variable hematuria, azotemia, anemia, hypertension and creatininemia. The exceptions would be the diagnosis of chronic pyelonephritis based on a positive urine culture of a known pathogenic organism or amyloidosis based on a history of chronic suppuration or neoplasm.

At the present time no specific diagnosis of renal disease can be made in man or animal based solely on history and clinical signs with the exception of acute glomerulonephritis in man. A diagnosis can usually be made where a history of recent streptococcal infection is recorded from a patient showing an abrupt onset of proteinuria, hematuria, with edema, hypertension and impaired renal function. In the dog the transient nature of the hematuria and edema does not alert the owner to the situation. Usually the acute stage has passed on to the chronic stage before overt clinical signs are noticed. Examination of the kidney at necropsy is presently the most accurate means available for differentiating the various renal diseases. This is not to say that renal biopsy could not be of diagnostic value in the live animal. The kidney will appear shrunken and contracted with a diffuse granularity of the capsular surface. The capsule will be adherent to the cortical surface and the cortex itself will be narrowed. This is characteristic of most chronic renal diseases. Pyelonephritis usually does not have the diffuse lesions but will have some areas of normal parenchyma.
The normal glomerular response to injury is either: 1) the proliferation of endothelial or epithelial cells, or 2) the accumulation of basement membrane-like material in the glomerulus. Therefore, it is not unlikely that the histopathology of renal disease will show the following:

**Chronic glomerulonephritis**—a proliferation of either or both endothelial cells and epithelial cells of the glomerulus and Bowman's capsule is indicative of this condition. In the case of primarily epithelial proliferation, adhesions between the visceral and parietal layers of Bowman's capsule will be seen. This may appear as crescent like thickenings of the parietal epithelium. This usually terminates in fibrosis with complete obliteration of the capsular space. If the proliferation was primarily endothelial, then an increased cellularity of the glomerular tuft will be seen. There will be no crescent shaped cells or parietal-visceral epithelial adhesions. The final outcome is usually collagen deposition in the glomerular tuft with preservation of the capsular space. The final possibility is if both endothelial and epithelial proliferation occur. This results in obliteration of both the glomerulus and capsule.

**Chronic pyelonephritis**—Focal lesions are the hallmark of this condition. Plasma cells and lymphocytes are present with increased amounts of interstitial tissue. Periglomerular fibrosis and basement membrane thickening are found in the later stages of the disease. Evidence of prior pelvic inflammation such as increased connective tissue with some plasma cells and lymphocytes is also helpful.

**Chronic interstitial nephritis**—Its appearance is very similar to terminal chronic glomerulonephritis. There will be much fibrosis with plasma cells present in both the medullary and cortical regions of the kidney.

**Amyloidosis**—This condition is characterized by the deposition of amyloid in the basement membrane of the glomerulus which is identified by its fibrillar nature under the electron microscope or its affinity for the crystal violet stain under the light microscope.

### Prognosis

Serial determinations of the blood urea nitrogen level offer an indication of the extent and progression of renal disease. Blood creatinine levels also point to the degree of renal damage. Thumb rules for both of these measurements are a blood urea nitrogen value of over 80 mg% and/or a blood creatinine value of over 6 mg% indicate a poor prognosis. Renal biopsy and function tests are becoming more important as prognostication aids. Biopsy, of course, allows for a specific morphologic diagnosis. The renal function tests allow for establishing the progression of the disease. Any complications to renal disease must be figured into the prognosis. For example, an enlarged cardiac silhouette, pulmonary congestion with respiratory dyspnea certainly offers a poor prognosis without mentioning the renal involvement.

### Treatment

Treatment of any chronic renal disease is usually less than satisfying. A few guidelines to follow are:

1. A definite diagnosis should be made prior to therapy since some renal conditions such as amyloidosis are aggravated by steroids whereas chronic glomerulonephritis may be helped by steroid administration;
2. The treatment should be of such degree and duration as to reduce the possibility of relapse. The symptomatic treatment is directed at the hypoproteinemia, proteinuria, and edema. An enriched protein diet with salt restriction and steroid therapy is quite helpful at times, although the steroid might be contraindicated if other granulomatous or inflammatory processes are occurring simultaneously with the renal disease. Diuretics are certainly of value in reducing the ascitic fluid or peripheral edema. The thiazides are thought to be the drug of choice in these cases, but their effectiveness depends on the patient having a normal
blood sodium level and some urinary sodium.⁷

Etiology of Glomerulonephritis

The reaction at the glomerular tuft is probably due to an antigen-antibody reaction. This is based on: 1) the presence of immunofluorescent antibodies to kidney glomerular membrane being present at the glomerular membrane, 2) the presence of a recent bacterial infection in the clinical history and the preferential location of bacterial M-protein at the glomerular membrane, 3) the frequency of production of the syndrome of glomerulonephritis by experimental immunological procedures.³ This antigen-antibody reaction results in the breakdown of basement membrane synthesis by the glomerular epithelial cells. This “moth eaten” appearance of the basement membrane allows blood protein and red blood cells to easily pass into the renal tubules and out in the urine.¹⁰

REFERENCES


Hidden Danger for Dogs in Snow

Most dogs love to romp in the snow, but snow can contain a hidden danger to a dog’s health, warns the Gaines Dog Research Center.

In urban and suburban areas, snow frequently becomes sprinkled with a commercial snow melter. Such a product is toxic to most animals and a dog can accidentally eat some of it.

Crystals of the snow melter can lodge between a dog’s foot pads, so it’s a good idea to check a dog’s feet after a walk or a play period in the snow.

Playing in the snow can be a very good exercise for any dog, the Center adds, as long as it is done in moderation. If a dog is outdoors for a long period of time and becomes soaked to the skin, a brisk rubdown with a towel should be given as soon as he is brought indoors.

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