DOWNLOAD PDF GLOMERULAR DISEASE

Chapter 1 : Glomerulonephritis Disease Reference Guide - www.nxgvision.com

For example, diabetic nephropathy is a form of glomerular disease that can be placed in two categories: systemic diseases, since diabetes itself is a systemic disease, and sclerotic diseases, because the specific damage done to the kidneys is associated with scarring.

Increased thirst and urine output Weight loss Halitosis bad breath Documentation of excessive protein in the urine There are four different laboratory tests that can be used to assess protein within the urine. The urinalysis is a very useful first screening test, but it is fraught with false positive results. Additionally, it is not always sensitive enough to detect the very earliest stages of glomerular damage. For these reasons, follow up testing via another methodology see below should be performed, either when the urinalysis is positive for protein or when the urinalysis is negative for protein but glomerular damage is suspected e. Urine protein to creatinine ratio: This test helps quantify the amount of protein in the urine. This accomplishes two things. It verifies whether the amount of protein in a urine sample is normal or increased. Secondly, it establishes a baseline to which future samples can be compared. This helps in the monitoring of disease progression. This test detects very small amounts of a protein called albumin within the urine, even before proteinuria can be documented via a urinalysis. As such a sensitive test, it is recommended in the following situations: The amount of protein in this duration of urine was then measured. This methodology was a pain-in-the-you-know-what for everyone involved, particularly the poor dog. Thank goodness, such testing is now considered antiquated and is rarely if ever used. Diagnosing glomerular disease If a dog demonstrates persistent proteinuria repeatable on multiple tests over the course of a few weeks , a battery of tests is typically recommended to rule out non-glomerular causes of excess protein within the urine such as urinary tract infection, stones, or bleeding. This testing typically includes the following: Complete blood cell count CBC Blood chemistry profile Urine culture Abdominal ultrasound or x-rays Other tests may be recommended for purposes of ruling out heartworm disease, infectious processes, cancer, and other diseases that can cause secondary glomerular damage. Heartworm testing Abdominal ultrasound Specific screening tests for infectious diseases A clear-cut diagnosis of glomerular disease requires a kidney biopsy. This can be accomplished via surgery, laparoscopy, or with ultrasound guidance. Whichever methodology is used, collection of a kidney biopsy has the potential to cause significant complications. Thoughtful discussion with a veterinarian about risks and benefits should always precede a kidney biopsy. Hypertension high blood pressure: Thromboembolism blood clot formation can occur within any organ Kidney failure Treatment of glomerular disease There are four primary goals when treating canine glomerular disease. Identify and eliminate the underlying cause of the glomerular damage: Doing so may resolve the proteinuria altogether the best outcome possible. For example, successful treatment of heartworm disease often eliminates the associated glomerular damage. Unfortunately, in many cases, the underlying cause of the glomerular disease cannot be identified or successfully eliminated. Attempt to lessen the degree of proteinuria: Doing so is the best bet for slowing the progression of kidney damage and other complications associated with glomerular disease. The mainstays of such therapy include: This decreases the amount of protein filtered by the kidneys. Their anti-inflammatory effects are thought to be responsible for decreasing proteinuria. Alters microscopic blood flow at the level of the kidneys. Suppression of the immune system may help diminish glomerular inflammation. Treatment of glomerular disease complications: Examples of such treatments include administration of medications to control high blood pressure, anticoagulant therapy to help prevent blood clot formation, and daily subcutaneous fluids to manage kidney failure. Once the diagnosis of glomerular disease is made and treatment is instituted, there will be a need for ongoing monitoring. A typical follow up veterinary visit for a dog with glomerular disease would include: For this reason, when glomerular disorder is suspected or has been diagnosed in your dog, I strongly encourage consulting with a veterinarian who specializes in small animal internal medicine. Prognosis The earlier glomerular disease is detected and managed, the greater the likelihood of deterring a negative outcome. Left unchecked, glomerular disease is known to increase the severity and progression of kidney failure. Canine glomerular disease is often associated with kidney failure which may progress very slowly, very quickly, or

anything in between. Some dogs live for several years with glomerular disease. The likelihood of such an outcome is far greater with appropriate treatment and monitoring. Nephrotic syndrome Dogs with severe glomerular disease can progress to a condition that is referred to as nephrotic syndrome. This is characterized by the following four abnormalities: Excess protein loss in the urine Decreased protein specifically albumin within the blood stream Elevated blood cholesterol level Presence of edema accumulation of watery fluid under the skin or within body cavities Nephrotic syndrome represents an advanced stage of glomerular disease. In addition to the treatment options mentioned above, measures to manage the edema fluid drainage, specific medications may be warranted. Have you had any experience with glomerular disease in your dogs?

DOWNLOAD PDF GLOMERULAR DISEASE

Chapter 2 : Glomerular Diseases Clinic â€“ Penn Medicine

Will glomerular disease cause chronic kidney disease? Over time, glomerular disease may stop your kidneys from getting rid of wastes in your blood. When this goes on for a long time, waste builds up in your blood, and you may have chronic kidney disease.

Having the opportunity to discuss cases with nephrologist and nephropathologist from different centers and even countries enriches our knowledge of glomerular diseases, where gaps in diagnostic and therapeutics still exist. Our fellows love it to the point that they stay after hours for the sessions. The combination of excellent cases, high level discussions and innovative use of media for participant interaction make glomcon a must for nephrologists taking care of patients with glomerular diseases. The case are well selected and the discussion is brilliant. I have learned a lot from these and thoroughly enjoy the discussions. I think your effort and hard work shows, brilliant initiative. It is especially great for us in private practice who get to interact with key opinion leaders in Nephrology. Your education initiative is a very impressive one, as anyone in any part of the world could actually have the same chance to learn nephrology from the best in a relax atmosphere. I would recommend your website to my pathologist and nephrologist colleagues so we could built a strong nephrology team together. Each month we are having more participants from around the world. It is truly a unique format to present challenging cases with expert pathology review and expert discussion. I see a continued growth in the number of participants and expert discussants. I consider it a unique learning experience because you get live evidence-based feedback from nephrologists, pathologists and other specialists involved in the cases. Definitely, a great learning opportunity for community nephrologists like me. A must for all nephrologists. You can simply listen or join in. Great way to learn. Sign-up for email alerts! Effective use of audiovisual media in medical education. It promotes the interaction among different centers locally and internationally. This provides a unique opportunity for nephrologists all around the world to interact with experts. This consortium is at work to create a seamless infrastructure for its members and be a catalyst for:

Chapter 3 : Glomerular Disease in Cats

Glomerular diseases damage the glomeruli, letting protein and sometimes red blood cells leak into the urine. Sometimes a glomerular disease also interferes with the clearance of waste products by the kidney, so they begin to build up in the blood.

Specific tests for tick-borne infections Blood pressure measurment A clear-cut diagnosis of glomerular disease requires a kidney biopsy. This can be accomplished via surgery , laparoscopy, or with ultrasound guidance. Whichever methodology is used, collection of a kidney biopsy has the potential to cause significant complications. Thoughtful discussion with a veterinarian about risks and benefits should always precede a kidney biopsy. Hypertension high blood pressure: Thromboembolism blood clot formation can occur within any organ Chronic kidney disease rarely, acute kidney injury Treatment of glomerular disease There are four primary goals when treating canine glomerular disease. Identify and eliminate the underlying cause of the glomerular damage Doing so may resolve the proteinuria altogether the best outcome possible. For example, successful treatment of heartworm disease often eliminates the associated glomerular damage. Unfortunately, in many cases, the underlying cause of the glomerular disease cannot be identified or successfully eliminated. Attempt to lessen the degree of proteinuria Doing so is the best bet for slowing the progression of kidney damage and other complications associated with glomerular disease. The mainstays of such therapy include: Feeding a diet that is low in protein quantity; this decreases the amount of protein filtered by the kidneys. Supplementation with omega-3 fatty acids; their anti-inflammatory effects are thought to be responsible for decreasing proteinuria. Administration of an angiotensin-converting-enzyme inhibitor drug enalapril, benazepril ; this alters microscopic blood flow at the level of the kidneys. Treatment of glomerular disease complications Examples of such treatments include, administration of medications to control high blood pressure, anticoagulant therapy to help prevent blood clot formation, and daily subcutaneous fluids to manage dehydration associated with chronic kidney disease. Follow up monitoring Once the diagnosis of glomerular disease is made and treatment is instituted, there will be a need for ongoing monitoring. The treatment of glomerular disease can be challenging, and the more experience a veterinarian has with this disease, the better. For this reason, when glomerular disorder is suspected or has been diagnosed, I strongly encourage consulting with a veterinarian who specializes in small animal internal medicine check with your veterinarian. Prognosis of glomerular disease The earlier glomerular disease is detected and managed, the greater the likelihood of deterring a negative outcome. Left unchecked, glomerular disease is known to increase the severity and progression of kidney disease. Canine glomerular disease is often associated with chronic kidney disease which may progress very slowly, very quickly, or anything in between. Some dogs live for several years with glomerular disease. The likelihood of such an outcome is far greater with appropriate treatment and monitoring. Nephrotic syndrome Dogs with severe glomerular disease can progress to a condition that is referred to as nephrotic syndrome. This is characterized by the following four abnormalities: Excess protein loss in the urine Decreased protein specifically albumin within the blood stream Elevated blood cholesterol level Presence of edema accumulation of watery fluid under the skin or within body cavities Nephrotic syndrome represents an advanced stage of glomerular disease with a poor prognosis. In addition to the treatment options mentioned above, measures to manage the edema fluid drainage, specific medications may be warranted.

Chapter 4 : Glomerular Diseases | Nephrology | Stanford Medicine

Glomerular Disease: Condition affecting the glomerules in the kidney. More detailed information about the symptoms, causes, and treatments of Glomerular Disease is.

Overview Resources Many diseases affect kidney function by attacking the glomeruli, the tiny units within the kidney where blood is cleaned. Glomerular diseases include many conditions with a variety of genetic and environmental causes, but they fall into two major categories: Glomerulonephritis gloh-MEHR-yoo-loh-nef-RY-tis describes the inflammation of the membrane tissue in the kidney that serves as a filter, separating wastes and extra fluid from the blood. Although glomerulonephritis and glomerulosclerosis have different causes, they can both lead to kidney failure. What are the kidneys and what do they do? The two kidneys are bean-shaped organs located near the middle of the back, just below the rib cage to the left and right of the spine. Each about the size of a fist, these organs act as sophisticated filters for the body. They process about quarts of blood a day to sift out about 2 quarts of waste products and extra water that eventually leave the body as urine. Blood enters the kidneys through arteries that branch inside the kidneys into tiny clusters of looping blood vessels. Each cluster is called a glomerulus , which comes from the Greek word meaning filter. The plural form of the word is glomeruli. There are approximately 1 million glomeruli, or filters, in each kidney. The glomerulus is attached to the opening of a small fluid-collecting tube called a tubule. Blood is filtered in the glomerulus, and extra water and wastes pass into the tubule and become urine. Eventually, the urine drains from the kidneys into the bladder through larger tubes called ureters. Each glomerulus-and-tubule unit is called a nephron. Each kidney is composed of about 1 million nephrons. In healthy nephrons, the glomerular membrane that separates the blood vessel from the tubule allows waste products and extra water to pass into the tubule while keeping blood cells and protein in the bloodstream. How do glomerular diseases interfere with kidney function? Glomerular diseases damage the glomeruli, letting protein and sometimes red blood cells leak into the urine. Sometimes a glomerular disease also interferes with the clearance of waste products by the kidney, so they begin to build up in the blood. Furthermore, loss of blood proteins like albumin in the urine can result in a fall in their level in the bloodstream. In normal blood, albumin acts like a sponge, drawing extra fluid from the body into the bloodstream, where it remains until the kidneys remove it. But when albumin leaks into the urine, the blood loses its capacity to absorb extra fluid from the body. Fluid can accumulate outside the circulatory system in the face, hands, feet, or ankles and cause swelling. What are the symptoms of glomerular disease? The signs and symptoms of glomerular disease include Proteinuria: But how would you know, for example, whether you have proteinuria? Before seeing a doctor, you may not. But some of these symptoms have signs, or visible manifestations: Proteinuria may cause foamy urine. Blood may cause the urine to be pink or cola-colored. Edema may be obvious in hands and ankles, especially at the end of the day, or around the eyes when awakening in the morning, for example. How is glomerular disease diagnosed? Patients with glomerular disease have significant amounts of protein in the urine, which may be referred to as "nephrotic range" if levels are very high. Red blood cells in the urine are a frequent finding as well, particularly in some forms of glomerular disease. Urinalysis provides information about kidney damage by indicating levels of protein and red blood cells in the urine. Blood tests measure the levels of waste products such as creatinine and urea nitrogen to determine whether the filtering capacity of the kidneys is impaired. If these lab tests indicate kidney damage, the doctor may recommend ultrasound or an x ray to see whether the shape or size of the kidneys is abnormal. These tests are called renal imaging. But since glomerular disease causes problems at the cellular level, the doctor will probably also recommend a kidney biopsyâ€”a procedure in which a needle is used to extract small pieces of tissue for examination with different types of microscopes, each of which shows a different aspect of the tissue. A biopsy may be helpful in confirming glomerular disease and identifying the cause. What causes glomerular disease? A number of different diseases can result in glomerular disease. It may be the direct result of an infection or a drug toxic to the kidneys, or it may result from a disease that affects the entire body, like diabetes or lupus. Many different kinds of diseases can cause

swelling or scarring of the nephron or glomerulus. Sometimes glomerular disease is idiopathic, meaning that it occurs without an apparent associated disease. The categories presented below can overlap: For example, diabetic nephropathy is a form of glomerular disease that can be placed in two categories: In an autoimmune disease, the immune system creates autoantibodies, which are antibodies or immunoglobulins that attack the body itself. Autoimmune diseases may be systemic and affect many parts of the body, or they may affect only specific organs or regions. Systemic lupus erythematosus SLE affects many parts of the body: Because women are more likely to develop SLE than men, some researchers believe that a sex-linked genetic factor may play a part in making a person susceptible, although viral infection has also been implicated as a triggering factor. Lupus nephritis is the name given to the kidney disease caused by SLE, and it occurs when autoantibodies form or are deposited in the glomeruli, causing inflammation. Ultimately, the inflammation may create scars that keep the kidneys from functioning properly. Conventional treatment for lupus nephritis includes a combination of two drugs, cyclophosphamide, a cytotoxic agent that suppresses the immune system, and prednisolone, a corticosteroid used to reduce inflammation. A newer immunosuppressant, mycophenolate mofetil MMF , has been used instead of cyclophosphamide. Preliminary studies indicate that MMF may be as effective as cyclophosphamide and has milder side effects. Often, the first indication that patients have the autoantibody is when they cough up blood. Treatments include immunosuppressive drugs and a blood-cleaning therapy called plasmapheresis that removes the autoantibodies. IgA nephropathy is a form of glomerular disease that results when immunoglobulin A IgA forms deposits in the glomeruli, where it creates inflammation. IgA nephropathy was not recognized as a cause of glomerular disease until the late s, when sophisticated biopsy techniques were developed that could identify IgA deposits in kidney tissue. The most common symptom of IgA nephropathy is blood in the urine, but it is often a silent disease that may go undetected for many years. The silent nature of the disease makes it difficult to determine how many people are in the early stages of IgA nephropathy, when specific medical tests are the only way to detect it. This disease is estimated to be the most common cause of primary glomerulonephritisâ€”that is, glomerular disease not caused by a systemic disease like lupus or diabetes mellitus. It appears to affect men more than women. Although IgA nephropathy is found in all age groups, young people rarely display signs of kidney failure because the disease usually takes several years to progress to the stage where it causes detectable complications. No treatment is recommended for early or mild cases of IgA nephropathy when the patient has normal blood pressure and less than 1 gram of protein in a hour urine output. Blood pressure medicinesâ€”angiotensin-converting enzyme inhibitors ACE inhibitors or angiotensin receptor blockers ARBs â€”that block a hormone called angiotensin are most effective at achieving those two goals simultaneously. Hereditary Nephritis â€” Alport Syndrome The primary indicator of Alport syndrome is a family history of chronic glomerular disease, although it may also involve hearing or vision impairment. This syndrome affects both men and women, but men are more likely to experience chronic kidney disease and sensory loss. Men with Alport syndrome usually first show evidence of renal insufficiency while in their twenties and reach total kidney failure by age Women rarely have significant renal impairment, and hearing loss may be so slight that it can be detected only through testing with special equipment. Usually men can pass the disease only to their daughters. Women can transmit the disease to either their sons or their daughters. Treatment focuses on controlling blood pressure to maintain kidney function. Infection-related Glomerular Disease Glomerular disease sometimes develops rapidly after an infection in other parts of the body. Acute post-streptococcal glomerulonephritis PSGN can occur after an episode of strep throat or, in rare cases, impetigo a skin infection. PSGN can bring on sudden symptoms of swelling edema , reduced urine output oliguria , and blood in the urine hematuria. Tests will show large amounts of protein in the urine and elevated levels of creatinine and urea nitrogen in the blood, thus indicating reduced kidney function. High blood pressure frequently accompanies reduced kidney function in this disease. PSGN is most common in children between the ages of 3 and 7, although it can strike at any age, and it most often affects boys. It lasts only a brief time and usually allows the kidneys to recover. In a few cases, however, kidney damage may be permanent, requiring dialysis or transplantation to replace renal function. Bacterial endocarditis, infection of the tissues inside the heart, is also associated with subsequent glomerular disease. Researchers are not sure whether the renal lesions that

form after a heart infection are caused entirely by the immune response or whether some other disease mechanism contributes to kidney damage. Treating the heart infection is the most effective way of minimizing kidney damage. Endocarditis sometimes produces chronic kidney disease CKD. HIV-associated nephropathy usually begins with heavy proteinuria and progresses rapidly within a year of detection to total kidney failure. Sclerotic Diseases Glomerulosclerosis is scarring sclerosis of the glomeruli. In several sclerotic conditions, a systemic disease like lupus or diabetes is responsible. Glomerulosclerosis is caused by the activation of glomerular cells to produce scar material. This may be stimulated by molecules called growth factors, which may be made by glomerular cells themselves or may be brought to the glomerulus by the circulating blood that enters the glomerular filter. Diabetic nephropathy is the leading cause of glomerular disease and of total kidney failure in the United States. Kidney disease is one of several problems caused by elevated levels of blood glucose, the central feature of diabetes. In addition to scarring the kidney, elevated glucose levels appear to increase the speed of blood flow into the kidney, putting a strain on the filtering glomeruli and raising blood pressure. Diabetic nephropathy usually takes many years to develop. People with diabetes can slow down damage to their kidneys by controlling their blood glucose through healthy eating with moderate protein intake, physical activity, and medications. Blood pressure medications called ACE inhibitors and ARBs are particularly effective at minimizing kidney damage and are now frequently prescribed to control blood pressure in patients with diabetes and in patients with many forms of kidney disease.

Chapter 5 : Glomerulonephritis - Wikipedia

Glomerulonephritis (GN), also known as glomerular nephritis, is a term used to refer to several kidney diseases (usually affecting both kidneys). Many of the diseases are characterised by inflammation either of the glomeruli or of the small blood vessels in the kidneys, hence the name, but not all diseases necessarily have an inflammatory component.

May present with the nephrotic syndrome. May present with the nephritic syndrome. The normal filtration barrier limits passage of molecules by size and charge selectivity. Glomerular diseases can be caused by a variety of mechanisms, including immune mediated, hemodynamic e. The most common cause of glomerulonephritis is immune-mediated injury. One is the deposition of circulating antibodies directed against specific antigens in the glomerulus. The major example is Goodpasture syndrome, in which an antibody is directed against the noncollagenous portion of the a3 chain of type IV collagen in the GBM. Immune complexes generally activate either the alternative or the classical pathway of the complement cascade, thereby initiating injury. In contrast to immune-complex mechanisms, certain glomer-ular diseases develop primarily from cell-mediated immunity. A classic example is minimal change disease, which has been postulated to occur when a T cell product injures podocytes and induces a permeability defect. Diagnosis Although renal biopsy remains the definitive diagnostic tool in glomerular disease, a thorough clinical evaluation is essential in determining the etiology and nature of these cases. History A carefully obtained history may help define a possible cause of glomerular disease. A family history of renal disease may suggest Alport syndrome, especially if affected individuals have hearing loss or another of the manifestations that are characteristic of this syndrome; there are also familial forms of IgA nephropathy, focal segmental glomerulosclerosis FSGS , and hemolytic-uremic syndrome. A number of glomerular diseases may be caused by drugs or toxins. For example, minimal change disease has been associated with the use of nonsteroidal anti-inflammatory drugs NSAIDs and interferon. Thrombotic microangiopathy has been associated with the use of cy-closporine, tacrolimus, mitomycin C, and oral contraceptives. Certain glomerular disorders are associated with malignancies. These include membranous nephropathy, which is associated with lung, breast, and gastrointestinal cancers; minimal change disease with Hodgkin disease; MPGN with non-Hodgkin lym-phoma; and amyloid with renal cell carcinoma. Signs and symptoms Patients with glomerular disease may be asymptomatic or may present with manifestations ranging from minimal findings to full-blown nephritic or nephrotic syndrome. Others present with painless gross hematuria. The classic example is hematuria immediately following an intercur-rent infection, which occurs in IgA nephropathy; onset of hema-turia 2 to 3 weeks after infection is typical of postinfectious glomerulonephritis. Patients with rapidly progressive glomeru-lonephritis RPGN present with rapid onset of renal failure over days or weeks with hematuria from RBC casts and proteinuria. Patients with chronic glomerulonephritis generally have hypertension, renal insufficiency, and proteinuria. On renal ultra-sonography, the kidneys are somewhat smaller than normal and display increased echogenicity. Renal biopsy The ultimate diagnostic tool in many renal diseases is a renal biopsy, which is performed percutaneously with a spring-loaded biopsy needle, using local anesthesia, or surgically as an open biopsy with the patient under general anesthesia. Renal biopsies should be performed only after the patient has been evaluated by a nephrologist, but they also should be performed in a timely fashion, because early initiation of therapy is essential in preserving renal function and preventing further, potentially irreversible, injury to the kidney. Although renal biopsy is generally a safe procedure, complications include hematoma, gross hematuria, arteriovenous fistula, and infection. In rare cases, renal biopsy leads to complications requiring surgical intervention. Four situations mandate consideration of renal biopsy: Some patients with nonnephrotic protein-uria, hematuria, and chronic renal failure may also benefit from a renal biopsy for diagnostic and prognostic purposes. The renal biopsy establishes a diagnosis, determines whether disease is mediated by antibody or by complement, and assists in determining disease-specific therapy. The biopsy also provides important information on the degree of glomerular and interstitial fibrosis. The latter is particularly important as a prognostic index. The renal pathology report includes a description of the kidney by light microscopy, immunofluorescence, and electron

microscopy. Figure 1 a Light micrograph illustrating the different components of a normal glomerulus. Light Microscopy Light microscopy describes glomerular cellularityâ€”that is, whether the number of glomerular cells is normal or increased hypercellularity. Often, light microscopy can distinguish which cell type resident glomerular cells or infiltrating cells such as neutrophils is increased; whether the GBMs are thickened and whether the capillary loops are patent, collapsed, or filled with material such as hyaline; and the presence or absence of glomerulosclerosis scarring. Although the glomerulus is the primary site of injury in glomerular disease, the tubules and the interstitium must be carefully inspected because the degree of tubulointerstitial fibrosis is the best predictor of the prognosis in renal disease. Light microscopy is also used to classify glomerular disease as focal or diffuse. If a small portion or segment of an individual glomerulus is involved,the disease is described as segmental; if most of an individual glomerulus is involved, it is called global. The presence of glomerular crescents can also be detected on light microscopy. Crescents are layers of cells parietal epithelial cells, podocytes, lymphocytes, and macrophages in the Bowman space, and their presence signifies severe disease. Immunofluorescence Immunofluorescent immunostaining determines the presence or absence of any underlying immune processes. Staining is directed against specific antibodies e. The pattern of the immune components is also diagnostic. A granular pattern is typical of antigen-antibody complexes, such as in membranous nephropathy, whereas a linear pattern occurs in anti-GBM disease. The location of antibody or complement e. Immunostaining can determine the presence of matrix proteins silver stain , amyloid fibrils Congo red , and viral inclusions. Electron Microscopy Electron microscopy provides information about the presence and subcellular location of immune complexes which are seen as electron-dense deposits , the degree of injury to glomerular cells, and the consistency of the basement membrane. Electron microscopy also detects fibrils and provides information on the ultrastructure of the kidney, such as podocyte effacement and flattening, which cannot be readily detected by light microscopy.

Chapter 6 : Glomerulonephritis: MedlinePlus Medical Encyclopedia

Glomerular disease reduces the kidney's ability to maintain a balance of specific substances in the blood stream. The kidney's job is to filter the bad toxins in the blood from the good proteins and red blood cells.

Microscopic hematuria - blood in urine that can only be seen by a microscope Edema Rapidly Progressive Glomerulonephritis RPGN - nephritic syndrome plus a relatively rapid loss of kidney function. Urinalysis - to assess levels of protein and blood in the urine. Blood tests - to assess levels of waste in the blood and can indicate kidney damage. Blood tests are often used to help pinpoint the cause of glomerular disease. Ultrasound or advanced radiographic imaging may be performed to indicate kidney abnormalities renal imaging. A kidney biopsy may be performed to confirm and help determine the cause of a glomerular disease. Genetic testing to identify genetic causes of glomerular disease. Penn physicians provide comprehensive, high quality patient care for all types of glomerular diseases. Diagnosis, including renal biopsy and biopsy interpretation services. Careful integration of care with referring physicians. Specialized nursing care provided to ensure careful follow up and close communication with patients undergoing intensive therapies. Dietary counseling by knowledgeable nutritionists. Opportunities to participate in clinical trials of novel therapies or other relevant clinical studies. Common treatments for glomerular disease include: If caused by an infection, antibiotics may be prescribed. Treatment at the Infusion Center by highly skilled nurses specially trained to administer and monitor immunosuppressive drugs. Monitoring of diet and nutrition. Penn Rheumatology , where rheumatologic diseases are the cause of glomerulonephritis. Penn High Risk Obstetrics , for care of women with glomerular disease considering pregnancy or who are pregnant. Penn Oncology , where glomerular disease is caused by malignancy including multiple myeloma. Penn Infectious Diseases , where glomerular disease is caused by chronic bacterial infection. Penn Liver GI where glomerular disease is caused by hepatitis C viral infection. In This Section Penn Kidney physicians provide expert evaluation, diagnosis and treatment for glomerular diseases.

Chapter 7 : The Glomerular Disease Study & Trial Consortium (GlomCon)

Glomerular disease (chronic kidney disease) occurs quite commonly in www.nxgvision.com affects purebreds and mixed-breeds alike, and can be an inherited disorder in certain breeds.

Glomerulonephritis refers to an inflammation of the glomerulus , which is the unit involved in filtration in the kidney. This inflammation typically results in one or both of the nephrotic or nephritic syndromes. Nephrotic syndrome The nephrotic syndrome is characterised by the finding of edema in a person with increased protein in the urine and decreased protein in the blood , with increased fat in the blood. Inflammation that affects the cells surrounding the glomerulus, podocytes , increases the permeability to proteins , resulting in an increase in excreted proteins. With decreased proteins in the blood, there is a decrease in the oncotic pressure of the blood. This results in edema, as the oncotic pressure in tissue remains the same. Although decreased intravascular oncotic i. Hyperlipidemia is thought to be a result of the increased activity of the liver. When damaged by inflammation , this can result in an increased permeability to proteins Main article: Nephritic syndrome The nephritic syndrome is characterised by blood in the urine especially Red blood cell casts with dysmorphic red blood cells and a decrease in the amount of urine in the presence of hypertension. In this syndrome, inflammatory damage to cells lining the glomerulus are thought to result in destruction of the epithelial barrier, leading to blood being found in the urine. At the same time, reactive changes, e. The reninâ€”angiotensin system may be subsequently activated, because of the decrease in perfusion of juxtaglomerular apparatus , which may result in hypertension. These forms usually result in the nephrotic syndrome. Minimal change disease[edit] Main article: Minimal change disease Minimal change disease is characterised as a cause of nephrotic syndrome without visible changes in the glomerulus on microscopy. Minimal change disease typically presents with edema , an increase in proteins passed from urine and decrease in blood protein levels, and an increase in circulating lipids i. Although no changes may be visible by light microscopy, changes on electron microscopy within the glomerules may show a fusion of the foot processes of the podocytes cells lining the basement membrane of the capillaries of glomerulus. It is typically managed with corticosteroids and does not progress to chronic kidney disease. Focal segmental glomerulosclerosis Focal segmental glomerulosclerosis is characterised by a sclerosis of segments of some glomerules. It is likely to present as a nephrotic syndrome. This form of glomerulonephritis may be associated with conditions such as HIV and heroin abuse, or inherited as Alport syndrome. On microscopy, affected glomerules may show an increase in hyalin , a pink and homogenous material, fat cells , an increase in the mesangial matrix and collagen. Treatment may involve corticosteroids , but up to half of people with focal segmental glomerulonephritis continue to have progressive deterioration of kidney function, ending in kidney failure. Membranous glomerulonephritis Membranous glomerulonephritis may cause either nephrotic or a nephritic picture. About two-thirds are associated with auto-antibodies to phospholipase A2 receptor, but other associations include cancers of the lung and bowel, infections such as hepatitis B and malaria , drugs including penicillamine , and connective tissue diseases such as systemic lupus erythematosus. Individuals with cerebral shunts are at risk of developing shunt nephritis , which frequently produces MGN. Microscopically, MGN is characterized by a thickened glomerular basement membrane without a hyperproliferation of the glomerular cells. Immunofluorescence demonstrates diffuse granular uptake of IgG. The basement membrane may completely surround the granular deposits, forming a "spike and dome" pattern. Tubules also display the symptoms of a typical Type III hypersensitivity reaction, which causes the endothelial cells to proliferate, which can be seen under a light microscope with a PAS stain. As the glomerulonephritis progresses, the tubules of the kidney become infected, leading to atrophy and hyalinisation. The kidney appears to shrink. Treatment with corticosteroids is attempted if the disease progresses. In extremely rare cases, the disease has been known to run in families, usually passed down through the females. This condition, similarly, is called Familial Membranous Glomerulonephritis. There have only been about nine documented cases in the world. Thin basement membrane disease[edit] Main article: Thin basement membrane disease Thin basement membrane disease is an autosomal dominant inherited disease characterized by thin glomerular basement

membranes on electron microscopy. It is a benign condition that causes persistent microscopic hematuria. This also may cause proteinuria which is usually mild and overall prognosis is excellent. These forms usually present with a triad of blood in the urine , decreased urine production , and hypertension , the nephritic syndrome. These forms usually progress to end-stage kidney failure ESKF over weeks to years depending on type. IgA nephropathy is classically described as a self-resolving form in young adults several days after a respiratory infection. It is characterised by deposits of IgA in the space between glomerular capillaries. Post-infectious glomerulonephritis Post-infectious glomerulonephritis can occur after essentially any infection, but classically occurs after infection with the bacteria Streptococcus pyogenes. It typically occurs 1â€”4 weeks after a pharyngeal infection with this bacterium, and is likely to present with malaise, a slight fever , nausea and a mild nephritic syndrome of moderately increased blood pressure, gross haematuria, and smoky-brown urine. Circulating immune complexes that deposit in the glomerules may lead to an inflammatory reaction. A biopsy is seldom done, and the disease is likely to self-resolve in children in 1â€”4 weeks, with a poorer prognosis if adults are affected. These forms present with the nephritic syndrome , hypocomplementemia , and have a poor prognosis. Two primary subtypes exist: Circulating immune complexes may activate the complement system , leading to inflammation and an influx of inflammatory cells. The C3 Nephritic Factor autoantibody stabilizes C3-convertase , which may lead to an excessive activation of complement.

Chapter 8 : Renal function - Wikipedia

The two basic types of glomerular disease include nephritic and nephrotic, The Basics patient education pieces answer the four or five key questions a patient might have about a given condition.

Specific screening tests for infectious diseases Measurement of blood pressure A clear-cut diagnosis of glomerular disease requires a kidney biopsy. This can be accomplished via surgery , laparoscopy or with ultrasound guidance. Whichever methodology is used, collection of a kidney biopsy has the potential to cause significant complications. Thoughtful discussion with a veterinarian about risks and benefits should always precede a kidney biopsy. Hypertension high blood pressure: Thromboembolism blood clot formation can occur within any organ Chronic kidney disease Treatment of glomerular disease There are four primary goals when treating feline glomerular disease. Identify and eliminate the underlying cause of the glomerular damage: Doing so may resolve the proteinuria altogether the best outcome possible. For example, successful treatment of hyperthyroidism may eliminate the associated glomerular damage. Unfortunately, in many cases, the underlying cause of the glomerular disease cannot be identified or successfully eliminated. Attempt to lessen the degree of proteinuria: Doing so is the best bet for slowing the progression of kidney damage and other complications associated with glomerular disease. The mainstays of such therapy include diet changes, supplements and medication. Treatment of glomerular disease complications: Examples of such treatments include administration of medications to control high blood pressure, anticoagulant therapy to help prevent blood clot formation, and daily subcutaneous fluids to manage dehydration associated with chronic kidney disease. Once the diagnosis of glomerular disease is made and treatment is instituted, there will be a need for ongoing monitoring. The treatment of glomerular disease can be challenging, and the more experience a veterinarian has with this disease, the better. For this reason, when a glomerular disorder is suspected or has been diagnosed in your cat, I strongly encourage consulting with a veterinarian who specializes in small animal internal medicine, ask your veterinarian for a recommendation. Prognosis of glomerular disease The earlier glomerular disease is detected and managed, the greater the likelihood of avoiding a negative outcome. Left unchecked, glomerular disease in cats is known to increase the severity and progression of kidney disease. Some cats live for several years with glomerular disease and kidney disease. The likelihood of such an outcome is far greater with appropriate treatment and monitoring.

Chapter 9 : Glomerular Disease Primer : Kidney Disease

Glomerulonephritis is a type of kidney disease in which the part of your kidneys that helps filter waste and fluids from the blood is damaged. Causes The filtering unit of the kidney is called the glomerulus.

Glomerular diseases include many conditions with a variety of genetic and environmental causes, but they fall into two major categories: Glomerulonephritis describes the inflammation of the membrane tissue in the kidney that serves as a filter, separating wastes and extra fluid from the blood. Glomerulosclerosis describes the scarring or hardening of the tiny blood vessels within the kidney. Although glomerulonephritis and glomerulosclerosis have different causes, they can both lead to kidney failure. What are the kidneys and what do they do? The two kidneys are bean-shaped organs located just below the rib cage, one on each side of the spine. Every day, the two kidneys filter about to quarts of blood to produce about 1 to 2 quarts of urine, composed of wastes and extra fluid. Blood enters the kidneys through arteries that branch inside the kidneys into tiny clusters of looping blood vessels. Each cluster is called a glomerulus, which comes from the Greek word meaning filter. The plural form of the word is glomeruli. There are approximately 1 million glomeruli, or filters, in each kidney. The glomerulus is attached to the opening of a small fluid-collecting tube called a tubule. Blood is filtered in the glomerulus, and extra fluid and wastes pass into the tubule and become urine. Eventually, the urine drains from the kidneys into the bladder through larger tubes called ureters. In the nephron left , tiny blood vessels intertwine with fluid-collecting tubes. Each kidney contains about 1 million nephrons. Each glomerulus-and-tubule unit is called a nephron. Each kidney is composed of about 1 million nephrons. In healthy nephrons, the glomerular membrane that separates the blood vessel from the tubule allows waste products and extra water to pass into the tubule while keeping blood cells and protein in the bloodstream. How do glomerular diseases interfere with kidney function? Glomerular diseases damage the glomeruli, letting protein and sometimes red blood cells leak into the urine. Sometimes a glomerular disease also interferes with the clearance of waste products by the kidney, so they begin to build up in the blood. Furthermore, loss of blood proteins like albumin in the urine can result in a fall in their level in the bloodstream. In normal blood, albumin acts like a sponge, drawing extra fluid from the body into the bloodstream, where it remains until the kidneys remove it. But when albumin leaks into the urine, the blood loses its capacity to absorb extra fluid from the body. Fluid can accumulate outside the circulatory system in the face, hands, feet, or ankles and cause swelling. What are the symptoms of glomerular disease? The signs and symptoms of glomerular disease include albuminuria: But how would you know, for example, whether you have proteinuria? Before seeing a doctor, you may not. But some of these symptoms have signs, or visible manifestations: Proteinuria may cause foamy urine. Blood may cause the urine to be pink or cola-colored. Edema may be obvious in hands and ankles, especially at the end of the day, or around the eyes when awakening in the morning, for example. How is glomerular disease diagnosed? Patients with glomerular disease have significant amounts of protein in the urine, which may be referred to as "nephrotic range" if levels are very high. Red blood cells in the urine are a frequent finding as well, particularly in some forms of glomerular disease. Urinalysis provides information about kidney damage by indicating levels of protein and red blood cells in the urine. Blood tests measure the levels of waste products such as creatinine and urea nitrogen to determine whether the filtering capacity of the kidneys is impaired. If these lab tests indicate kidney damage, the doctor may recommend ultrasound or an x-ray to see whether the shape or size of the kidneys is abnormal. These tests are called renal imaging. But since glomerular disease causes problems at the cellular level, the doctor will probably also recommend a kidney biopsy â€”a procedure in which a needle is used to extract small pieces of tissue for examination with different types of microscopes, each of which shows a different aspect of the tissue. A biopsy may be helpful in confirming glomerular disease and identifying the cause. What causes glomerular disease? A number of different diseases can result in glomerular disease. It may be the direct result of an infection or a drug toxic to the kidneys, or it may result from a disease that affects the entire body, like diabetes or lupus. Many different kinds of diseases can cause swelling or scarring of the nephron or glomerulus. Sometimes glomerular disease is idiopathic, meaning that it

occurs without an apparent associated disease. The categories presented below can overlap: For example, diabetic nephropathy is a form of glomerular disease that can be placed in two categories: In an autoimmune disease, the immune system creates autoantibodies, which are antibodies or immunoglobulins that attack the body itself. Autoimmune diseases may be systemic and affect many parts of the body, or they may affect only specific organs or regions. Systemic lupus erythematosus SLE affects many parts of the body: Because women are more likely to develop SLE than men, some researchers believe that a sex-linked genetic factor may play a part in making a person susceptible, although viral infection has also been implicated as a triggering factor. Lupus nephritis is the name given to the kidney disease caused by SLE, and it occurs when autoantibodies form or are deposited in the glomeruli, causing inflammation. Ultimately, the inflammation may create scars that keep the kidneys from functioning properly. Conventional treatment for lupus nephritis includes a combination of two drugs, cyclophosphamide, a cytotoxic agent that suppresses the immune system, and prednisolone, a corticosteroid used to reduce inflammation. A newer immunosuppressant, mychophenolate mofetil MMF , has been used instead of cyclophosphamide. Preliminary studies indicate that MMF may be as effective as cyclophosphamide and has milder side effects. Goodpasture Syndrome involves an autoantibody that specifically targets the kidneys and the lungs. Often, the first indication that patients have the autoantibody is when they cough up blood. But lung damage in Goodpasture Syndrome is usually superficial compared with progressive and permanent damage to the kidneys. Goodpasture Syndrome is a rare condition that affects mostly young men but also occurs in women, children, and older adults. Treatments include immunosuppressive drugs and a blood-cleaning therapy called plasmapheresis that removes the autoantibodies. IgA nephropathy is a form of glomerular disease that results when immunoglobulin A IgA forms deposits in the glomeruli, where it creates inflammation. IgA nephropathy was not recognized as a cause of glomerular disease until the late s, when sophisticated biopsy techniques were developed that could identify IgA deposits in kidney tissue. The most common symptom of IgA nephropathy is blood in the urine, but it is often a silent disease that may go undetected for many years. The silent nature of the disease makes it difficult to determine how many people are in the early stages of IgA nephropathy, when specific medical tests are the only way to detect it. This disease is estimated to be the most common cause of primary glomerulonephritisâ€”that is, glomerular disease not caused by a systemic disease like lupus or diabetes mellitus. It appears to affect men more than women. Although IgA nephropathy is found in all age groups, young people rarely display signs of kidney failure because the disease usually takes several years to progress to the stage where it causes detectable complications. No treatment is recommended for early or mild cases of IgA nephropathy when the patient has normal blood pressure and less than 1 gram of protein in a hour urine output. Blood pressure medicinesâ€”angiotensinâ€”converting enzyme inhibitors ACE inhibitors or angiotensin receptor blockers ARBs â€”that block a hormone called angiotensin are most effective at achieving those two goals simultaneously. Hereditary Nephritisâ€”Alport Syndrome The primary indicator of Alport syndrome is a family history of chronic glomerular disease, although it may also involve hearing or vision impairment. This syndrome affects both men and women, but men are more likely to experience chronic kidney disease and sensory loss. Men with Alport syndrome usually first show evidence of renal insufficiency while in their twenties and reach total kidney failure by age Women rarely have significant renal impairment, and hearing loss may be so slight that it can be detected only through testing with special equipment. Usually men can pass the disease only to their daughters. Women can transmit the disease to either their sons or their daughters. Treatment focuses on controlling blood pressure to maintain kidney function. Infection-related Glomerular Disease Glomerular disease sometimes develops rapidly after an infection in other parts of the body. Acute post-streptococcal glomerulonephritis PSGN can occur after an episode of strep throat or, in rare cases, impetigo a skin infection. The Streptococcus bacteria do not attack the kidney directly, but an infection may stimulate the immune system to overproduce antibodies, which are circulated in the blood and finally deposited in the glomeruli, causing damage. PSGN can bring on sudden symptoms of swelling edema , reduced urine output oliguria , and blood in the urine hematuria. Tests will show large amounts of protein in the urine and elevated levels of creatinine and urea nitrogen in the blood, thus indicating reduced kidney function. High blood pressure frequently accompanies reduced kidney function in this disease.

PSGN is most common in children between the ages of 3 and 7, although it can strike at any age, and it most often affects boys. It lasts only a brief time and usually allows the kidneys to recover. In a few cases, however, kidney damage may be permanent, requiring dialysis or transplantation to replace renal function. Bacterial endocarditis, infection of the tissues inside the heart, is also associated with subsequent glomerular disease. Researchers are not sure whether the renal lesions that form after a heart infection are caused entirely by the immune response or whether some other disease mechanism contributes to kidney damage. Treating the heart infection is the most effective way of minimizing kidney damage. Endocarditis sometimes produces chronic kidney disease CKD. HIV-associated nephropathy usually begins with heavy proteinuria and progresses rapidly within a year of detection to total kidney failure. Sclerotic Diseases Glomerulosclerosis is scarring sclerosis of the glomeruli. In several sclerotic conditions, a systemic disease like lupus or diabetes is responsible. Glomerulosclerosis is caused by the activation of glomerular cells to produce scar material. This may be stimulated by molecules called growth factors, which may be made by glomerular cells themselves or may be brought to the glomerulus by the circulating blood that enters the glomerular filter. Diabetic nephropathy is the leading cause of glomerular disease and of total kidney failure in the United States.