Disease of kidney

Nephrosis

Nephrosis includes degenerative and inflammatory lesions primarily affecting the renal tubules. It can be occurs as a sequel or renal ischemia and following toxic insult to the kidney.

Nephrosis is the most common cause of acute kidney failure. Uremia from nephrosis may be develop acute or may occur in the terminal stage of chronic renal disease.

Renal ischemia

Reduced blood flow through the kidneys usually result from general circulatory failure. There is transitory Oliguria followed by uremia if the circulatory failure is not corrected.

Etiology

Any conditions which predisposes the animal to marked hypotension and release of endogenous pressure agents potentially can initiate hemodynamically mediated acute renal ischemia and renal failure. Ischemia may be acute or chronic.

Acute renal ischemia

- General circulatory emergencies such as shock, dehydration, acute hemorrhagic anaemia, acute heart failure.
- Embolism of renal artery, recorded in horses.
- Extrinsic ruminal distension in cattle.
- Chronic renal ischemia.

❖ Chronic circulatory insufficiency such as congestive heart failure.

Pathogenesis

Acute ischemia of kidneys occurs when compensatory vasoconstriction affects the renal blood vessels in response to sudden reduction in cardiac out put.

As blood pressure fall, glomeriolar decrease and metabolites that are normally excreted accumulate in the blood stream. The concentration of urea in the blood increase (pre renal uremia).

AS glomeriolar & filtration falls, tubular reabsorption increases causing reduced urine flow. Up to certain stage, the degenerative changes are severe enough and sufficient duration, the renal damage is promenant. Acute circulatory disterbances are more likely to be followed by degenerative lesions than chronic congestive heart failure.

The paranchymatous lesions vary from tubular necrosis to diffuse cortical necrosis in which both tubules and glomeerioli are effected.

Clinical findings

Renal ischemia does not appear as distict disease ad its sings are masked by the clinical signs of primary disease.

Oliguria and azotemia will go unnoticed is most cases if the renal insufficiency may cause a poor response to treatment with transfusion are the infusion of other fluids in haemorrhagicand haemolytic anaemia, shocks and dehydration. In these cases un explained depression or poor response to therapy indicates that, renal involvement should be investigated. The general clinical picture is not one of acute renal failure and uremia.

Clinical pathology

- ❖ Evaluation of renal function
- ❖ Urinalysis (blood urea nitrogen & creatinine)
- ❖ Protein urea is an early indication to damage of renal parenchyma.
- ❖ The passage of large volume of urine of low specific gravity after a period of Oliguria is usually a good indication of a return of normal glomeriolar and tubular function.

Necropsy findings

Lesions of renal ischemia are present primary in the cortex which is pale and swollen. There may be distinct line of necrosis visible at the corticomedullary junction.

Histologically there is necrosis of tubular epithelium and in severe cases of the glomeerioli.

In haemoglobinuria and Myoglobinuria hyaline castes are present in the tubule.

Differential diagnosis

1- Glomeriolone nephritis
2- Toxic nephrosis

Treatment

Treatment must be directed at correcting fluid, electrolytes and acid – base disturbance as soon as possible. If renal damage has occurred , supportive treatment as in acute renal failure should be used.

Toxic nephrosis

The kidney are particularly vulnerable to endogenous and exogenous toxins because they receive a large proportion of the total cardiac out put and because substances are concentrated in the kidneys for excretion.

Etiology

Most cases of nephrosis are caused by direct action of toxins but hemodynamic changes may contribute to the pathogenesis .

Toxins

- metals : mercury , arsenic , cadmium, selenium, organic copper compound.
- Antimicrobials like Amnioglycosides, tetracycline & sulfa
- Vitamin such as Vit K, D_2 and D_3.
- Non steroidal oil inflammatory drugs such as phenyl butazon and Flunixin meglumin.
- Benzimidazole compounds used as antihelmentics such as thiabendazole
- Monensim in ruminant
- Highly chlorinated naphthalins.
- Oxalate in plants and fungi.
- Mycotoxins.

Pathogenesis

1- In acute nephrosis there is obstruction to the flow of glomeriolar filtrate through the tubules as a result of interstitial odema and interluminal casts.

2- If there is sufficient tubular damage, there may be back leakage of glomeriolar filtrate into the interstetial tissue.
3- There may also be a direct toxic effect on glomeerioli which decrease glomeriolar filtration the combined effect is Oliguria and anuria.
4- In subacute cases impaired tubular resorption of solutes lead to polyuria.

Clinical findings

Clinical signs may not be associated with urinary system. In peracute cases such as those caused by Vit K_3 administered by injection there may be colic and Stranguria.

In acute nephrosis there is Oliguria and proteinuria with clinical sings of uremia in terminal stage , these sings include: depression , dehydration, anorexia, hypothermia , a slow or an elevated heart rate and weak plus. Diarrhea may be present that is sufficiently intense to cause severe clinical dehydration. In cattle there is a continuous mild hypocalcemia. Polyuria is present in chronic cases. The degree of renal epithelial loss is not sufficient to cause complete renal failure and provided the degree of renal damage is small , complete function is regained.

Clinical pathology

1- urinalysis

*Increase of blood urea and creation

* proteinuria , Glucosuria and haematuria.

2- Detection of enzyme gamma, glutamyle transferase (GGT) in urine.

3- Hypoprpteinemia may be present.

4- Hypercalcemia and hypophosphateemia but in Vit D intoxication serum calcium and phosphate are both increased.

5- Azutemia occurs when uremia is present.

Necropsy findings

In acute cases the kidney is swollen and wet on the cut surface and edema especially of perirenal tissue, may be apparent. Histologically there is necrosis and desquamation of tubular epithelium and hyaline casts may be present in dilate tubules in phenyl butazon poisoning the renal lesion is specifically a renal medullary necrosis. There may be also be ulcers in all or any part of the alimentary tract from the mouth of the colon.

Differential diagnosis

1- Clinical dedifferentiation from acute glomeriolonephritis is difficult but clinical sings of involment of other oranges in the toxic process may be present.
2- Occasional cases of diabetes mellitus.
3- Cushing's syndrome (chronic hyper adernocorticoticism).

Treatment

Treatment should be direct at general supportive care for acute renal disease. if the toxin can be identified, it should be removed treatment for specific toxins may be available**.**

Glomerulonephritis

Glomerulonephritis can occurs as a primary disease or as a component of disease affecting several body system such as equine infectious anaemia and chronic swine fever. In primary glomerulonephritis the disease involves only the kidney, predominantly affecting the glomeruli although the inflammatory process extend to affect the surrounding interstitial tissue and blood vessels. Primary and secondary glomerulonephritis are not common causes of clinical disease in farm animals. Proliferative glomerulonephritis is reported as incidental finding in normal sheep, cattle and goats. Clinical disease from glomerulonephritis is rare these species but has reported in cattle and as a conginital condition in sheep. Proliferative glomerulonephritis is a relatively common causes of chronic renal failure in horses.

Interstitial nephritis

Interstitial nephritis is rarely recognized as a cause of clinical disease in farm animals although it is a frequent postmortem finding in some species . interstitial nephritis may be diffuse or have a focal distribution. In calves focal Interstitial nephritis (white – spotted kidney) occurs as a sequel to bacteremia. It is a common incidental finding at necropsy but dose not present as a clinical urinary tract disease.

Chronic interstitial fibrosis is a common postmortem finding in horses suffering from chronic renal failure. Horses with chronic Interstitial nephritis have the clinical syndrome of chronic renal failure with uremia.

Embolic nephritis

Embolic lesions in the kidney do not cause clinical sings unless they are very extensive, in which case septicemia may be followed by uremia.

Etiology

Embolic suppurative nephritis or renal abscess may occur after any septicemia or bacteremia when bacteria lodge in renal tissue.

Embolic may be originate from localized septic process such as :

- Valvular endocarditis in all species.
- Suppurative lesions in uterus, udder, navel , peritoneal cavity in cattle.

or may be associated with systemic infections such as

- Septicemia in neonatal animals including shigellosis in foals and *E.coli* septicemia in calves.
- Erysipelas in pigs .
- Septicemic or bacteremic strangles.

Pathogenesis

Bacterial emboli localize in renal tissue and cause the development of focal suppurative lesions. Embolic can block larger vessels and cause infraction of portions of kidney. Infarcts are not usually so large that the residual renal tissue can not compansate fully and usually cause no clinical sings.

If the urine is cheeked repeatedly , the sudden appearance of proteinuria , casts and microscopic haematuria without other sings of renal disease suggest the occurance of a renal infarct. The gradual enlargement of focal embolic lesions leads to the development of toxemia and gradual loss

of renal function. Clinical sings usually develop only when multiple emboli destroy much of the renal parenchyma or when there is one more large infected infarcts.

Clinical findings

Usually there is insufficient renal damage to cause sings of renal disease. sings of toxemia and primary disease are usually present. The kidney may be enlarged on rectal examination. Repeated showers of emboli or gradual spread from several large, suppurative infarcts may cause sings similar to pyelonephritis. Large infarcts may cause bouts of transient abdominal pain.

Clinical pathology

- ❖ Haematuria and Pyuria are present but microscopic examination may be necessary to detect these abnormalities when the lesions are minor.
- ❖ Protein urea is present but is also normally present in neonatal animals in the first 30- 40 hours of life.
- ❖ Culture urine at the time when proteinuria occurs may reveal the identity of the bacteria infecting the embolus.
- ❖ Haematology usually reveals evidence of an acute or chronic inflammatory process.

Necropsy findings

The early lesion are seen as small gray spots in the cortex in the latter stage the lesions may developed into large abscesses which may be confluent and in some cases extend into pelvis.

Fibrous tissue may surrounding longstanding lesions and healed lesions consist of area of scar tissue in the cortex. The area have depressed surfaces and indicates that destruction of cortical tissue has occurred. Extensive scaring may cause an obvious irregular reduction in the size of the kidney.

Differential diagnosis

- Pyelonephritis
- Ischemic tubular nephrosis
- Acute intestinal obstruction (abdominal pain)

Treatment

- Antimicrobials according to culture and sensitivity (Avoid potentially nephrotoxic drugs). Antibacterial treatment should be continued for fairly lengthy period (7-14) days.
- In embolic nephritis the primary disease should be controlled.

Pyelonephritis

Pyelonephritis develops by ascending infection form lower urinary tract. Clinically it is characterized by Pyuria, suppurative nephritis, cystitis and uretritis.

Etiology

- Secondary to bacterial infections of the lower urinary tract.
- Spread from embolic nephritis of haematological origin such as septicemia in cattle caused by *pseudomonas areuginosa*.
- Specific pyelonephritis caused by corynebacterium renal in cattle and eubacterium suis in pigs.

Pathogenesis

Pyelonephritis develops when bacteria from the lower urinary tract ascend the ureters and become established in the renal pelvis and medulla. Bacteria are assisted to ascending to ureter by urine stasis and reflux of urine in the bladder.

Urinary stasis can occur as a result from blocking by the ureter by inflammatory swelling or debris, pressure from the uterus in pregnant females, and obstructive urolithiasis. Initially the renal pelvis and medulla are affected although infection may extend to the cortex.

Pyelonephritis cause systemic sings of toxemia and fever and renal involvement is bilateral and sufficiently extensive, uremia develops. Pyelonephritis is always a compained by Pyuria and haematuria become of inflammatory lesions of ureters and bladder.

Clinical findings

The clinical findings of pyelonephritis vary between species such as bovine pyelonephritis.

Clinical pathology

- Erythrocytes, leukocytes and cell debris and present in the urine on microscopic examination and may be grossly evident in severe cases.
- Quantitive urine in culture in necessary to determine the causative bacteria.

Necropsy findings

The kidney is usually enlarged and lesions in the parenchyma are varying stages of development. Characteristic lesions are necrosis and contains clots of pus and turbid urine. Streaks of gray , necrotic materials radial out through the medulla and may extend to cortex.

Affected area of Paranchyma are necrotic and may be separated by apparently normal tissue. Healed lesions, appear as contructed scar tissue. Histologically the lesions are similar to those of embolic nephritis except that there is extensive necrosis of the spices of the papillae. Necrotic, suppurative lesions are usually present in the bladder and ureters.

Differential diagnosis

- Cystitis, embolic nephritis (pus & blood in urine)

Treatment

As mention in the principals of treatment of urinary tract disease.

Hydronephrosis

Hydronephrosis is a dilatation of renal pelvis with progressive atrophy of the renal Paranchyma.

Etiology

- Conginital condition
- Obstruction of the urinary tract

Pathogenesis and clinical sings

Any urinary tract obstruction can lead hydronephrosis but the extent and duration of the obstruction are important in determining the severity of renal lesions.

- Urinary tract obstruction that are chronic, unilateral and incomplete are more likely to lead to hydronephrosis.
- Acute obstruction of bladder and urethra that are corrected promptly are not usually associated with significant kidney damage.

As a result recurrence of obstruction rather than renal failure in the major sequel to urolithiasis un ruminants. Clinical picture is dominated by sings of unuria , dysuria and Stranguria.

Chronic or partial obstruction cause progressive distension of the renal pelvis and pressure atrophy of the renal parenchyma. If the obstruction is unilateral, the unaffected kidney can compensate fully for the loss of function and the obstruction may be detectable by rectal examination. Chronic bilateral obstruction although they rare in large animals can cause chronic renal failure.

Renal neoplasms

Primary tumors of kidney are uncommon. Carcinomas occurs in cattle and horse and nephroblastomas occure in pigs.

Enlargement of kidney is the characteristics sings, in cattle and horses neoplasms should be considered in the differential diagnosis of renal enlargement.

Renal adenocacinomas are very slow growing but are not usually diagnosis until the disease is well advanced.

In horses the most common sings are weight loss, reduced appetite and intermittent bouts of abdominal pain. Some affected horses have masive ascitis, haemopertonium or haematuria. Masses are usually readily palpable on rectal examination. Local invasion and metastasis to the liver and lungs are common.

Metastatic neoplasms occur fairly commonly in the kidney particularly in enzootic bovine leukosis but they do not cause clinical renal disease. tumor masses may be palpable as discrete enlargements in the kidneys of cattle or may involve the kidney diffusely causing generalized enlargement of the kidney.