Diseases of Urinary system

Principles of renal insufficiency

- Renal insufficiency and renal failure

Renal function

The main functions of kidney are :

1- Elimination of water formed in or introduced into the body in excess of the amount required for normal metabolism.

2- Elimination of inorganic elements according to the need of the body.

3- Elimination of non volatile end products of metabolic activity.

4- Retention within the body of substances required for maintenance of normal function including amino acids , hormones , vitamins , plasma protein , glucose …. Etc.

5- Elimination of certain foreign substances such as H^+ and ammonia.

Therefore the kidney play an important role in the regulation of water imbalance, electrolyte balance and acid – base balance.

Renal function depend on the integrity of the individual nephrons. Insufficiency can occur from

A- Abnormality in the rate of renal blood flow.

B- Abnormality in the glomerular filtration rate.

C - Abnormality in the efficiency of tubular reabsorption.

Of theses the latter two are intrinsic function, whereas the first depend largely on vasomotor control, which in animals affected by circulatory emergencies such as shock, dehydration and haemorrhage. Circulatory emergencies may lead to marked reduction in glomerular infiltration but they are external in origin and can not be considered as true causes of renal insufficiency. However prolonged circulatory disruption can cause renal ischemia and renal insufficiency.

Glomerular infiltration and tubular reabsorption can be affected independently of each other in disease states. In haemoglobinuric nephrosis, glomerular infiltration is unaffected but tubular reabsorption is seriously depressed.

Usually the common blood supply of the glomeruli and tubules means that damage to the one part of the nephrons is followed by damage to the remaining parts. As a result , it is probably more accurate to state that kidey disease causes a loss of entire nephrons rather than selective loss of tubular or glomerular components. At least this is true when trying to establish a prognosis in a particular case. Even when entire nephrons are lost rather than a selective loss in tubular function the end result may be remaining residual nephrons may result in impaired renal function by imbalance between glomerular and tubular function.

The progress and the end result of any type of renal disease tend to be very similar. In glomerulonephritis the primary reaction is glomerular but secondary involvement of tubules occur. Similarly glomerular dysfunction follows nephrosis where the initial lesion is in the tubules and interstitial nephritis where the primary and major lesion is tubular degeneration.

The clinical and clincopathological sings of renal dysfunction often depend on the anatomical location of the lesion and on the imbalance in function between glomeruli and tubules. Renal dysfunction tend to be a dynamic process so the degree of dysfunction varies with time. If renal dysfunction is so severe that the animals continued existence is not possible it is said to be in a state of renal failure and the clinical syndrome of uremia will be present.

Causes of renal insufficiency and viremia:

The causes of renal insufficiency and therefore of renal failure and uremia can be divided into prerenal, renal and post renal causes.

1- Pre- renal causes include:-

1- Congestive heart failure and acute circulatory failure, in which acute renal ischemia occurs.

2- Haemoglobinuric and myoglobinuric nephrosis.

3- in ruminant severe bloat can interfere with renal function.

2- Renal causes

- ❖ Glomerulonephritis
- ❖ Interstitial nephritis
- ❖ Pyelonephritis
- ❖ Embolic nephritis

3- Post – renal causes

- ❖ Complete obstruction of urinary tract.
- ❖ Rapture of any part of urinary tract.

Pathogenesis of renal insufficiency and renal failure.

1- Damage of glomerular epithelium destroy the selective permeability and permits the passage of plasma protein into glomerular filtrate . the protein is principally albumin (albumin urea).

2- Glomerular filtration may cease completely when there is extensive damage to glomeruli particularly if it there is acute swelling of the kidney but it is believed that anurea in the terminal stages of acute renal

disease is caused by back diffusion of glomerular filtrate through the damage tubular epithelium rather than failure of filtration.

3- When the kidney damage is less severe, the remaining nephrons compensate to maintain total glomerular filtration by increasing their filtration rates. When this occurs the volume of glomerular filtrate may exceed the capacity of the tubular epithelium to reabsorb fluid and solute. The tubules may be unable to achieve normal urine concentration. As a result an increased volume of urine with constant specific gravity is produce and solute diuresis occurs. This is exacerbated if the tubular function of the compensating nephrons is also impaired.

4- Decreased glomerular filtration also result in retention of metabolic products such as urea, phosphate and sulfate the letter two lead to renal metabolic acidosis. Phosphate retention also lead to secondary hypocalcemia due to increase calcium excretion in urine in horses , the kidneys are an important rout of excretion of calcium so the decrease glomerular filtration rate may result in hypercalcemia is there is large dietary intake of calcium .

5- Loss of tubular reabsorptive function is evident by a continued loss of sodium, hyponatremia eventually occurs in all cases of renal failure , the continuous loss of large quantities of fluid due to solute diuresis may cause clinical dehydration.

6- In the terminal stage, prolonged hypoportenemia result in rapid loss of body condition and muscle weakness. Acidosis also a contributing factor to muscle weakness and mental attitude. Hyponatermia and Hyperkalemia cause skeletal muscle weakness and myocardial asthenia. Hypocalcemia may be sufficient to contribute to circulatory failure and

to nervous sings. All of these factors play some part in the production of clinical signs. In some cases one or other of them may be of major importance so the clinical syndrome is variable and is rarely diagnostic for renal failure.

Renal failure is seen as the clinical state of uremia. It is characterized biochemically by an increase in blood levels of urea and creatinine (Azotomia) and by retention of other solutes. Uremia can also occur in urinary tract obstraction.