Diseases of the urinary system

Diseases of the bladder and urethra are more common and more important than Diseases on the kidneys in farm animals .

Occasionally ,renal insufficiency develops as a sequel to diseases such as pyelonephritis embolic nephritis ,amyloidosis and nephrosis.

**The principles of renal insufficiency**

The kidneys excrete the end- products of tissue metabolism (except for carbon dioxide ) and maintain fluid and electrolyte metabolism and acid – base balance by selective excretion of these substances.

The kidney maintains homeostasis by varying the volume of water and the concentration of solutes in the urine .

It is convenient to think of the kidney as composed of many similar nephrons, the basic functional units of the kidney.

Each nephron is composed of blood vessels ,the glomerulus and a tubular system which consists of the proximal tubule ,the loop of Henle ,the distal tubule and the collecting duct.

In general the tubules maintains homeostasis while the glomeruli control excretion of metabolic end-products .

The glomerulus is a semi permeable filter that allows easy passage of water and low molecular weight solutes but restricts passages of high molecular weight substances such as plasma proteins.

Glomerulus filtrate is derived from plasma by simple passive filtration driven by arterial blood pressure .

Glomerular filtrate is identical to plasma except that it contains little protein or lipids .

The volume of filtrate and therefore its content of metabolic end- products depends upon the hydrostatic pressure and the glomerular capillaries and on the proportion of glomeruli which are functional .

The epithelium of the tubules actively and selectively reabsorbs substances from the glomerular filtrate which are needed for utilization and participation in metabolic processes ,while permitting the excretion of waste products.

Glucose is reabsorbed entirely ,within the normal range of blood vessels ,phosphate is reabsorbed in varying amounts depending upon the needs of the body to convert it.

Other substances such as inorganic sulfates and creatinine are not reabsorbed in appreciable amounts.

The tubules also actively secrete substances ,particularly electrolytes as they function to regulate acid –base balance.

The principal mechanism that regulates water reabsorption by the renal tubules is antidiuretic hormone (ADH).

Tissue dehydration and an increase in the serum osmotic pressure of tissue fluid stimulates secretion of ADH from the posterior pituitary gland.

The renal tubules respond to ADH by conserving water and producing concentrated urine.

A partial loss of function is described

as renal insufficiency .when the kidneys can no longer regulate body fluid and solute composition ,renal failure occurs .

**Renal insufficiency and renal failure**

Renal function depends upon the number and functionality of the individual nephrons. Insufficiency can occur from :1- Abnormality in the rate of renal blood flow ,2-the glomerular filtration rate 3-the efficiency or tubular reabsorption .

The latter two are intrinsic functions of the kidney, the first depended largely on vasomotor control which in animals is affected by circulatory emergencies such as shook, dehydration ,and hemorrhage .

Circulatory emergencies may lead to a marked reduction in glomerular filtration but they are external in origin and cannot be considered as true causes of renal insufficiency .

Glomerular filtration and tubular reabsorption can be affected independently in disease states.

In hemoglobin uric nephrosis glomerular filtration ins un affected but tubular reabsorption is seriously depressed .

The common blood supply of the glomerular and tubule means that damage to one part of the nephron is followed by damage to the remaining parts.

As a result, it is probably more accurate to state that kidney disease cause a loss of entire nephrons rather than selective loss of tubular on glomerular components.

The progress and the end result of any type of renal disease tend to be very similar.

In glomerular nephritis the primary reaction is glomerular , but secondary involvement of the tubular occurs.

Similarly glomerular dysfunction follows nephrosis where the initial lesion is in the tubules ,and interstitial nephritis where the primary and meager lesion is tubular degeneration .

**Causes of renal insufficiency and uremia**

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

**Pre renal causes :**  included :-

congestive heart failure ,acute circulatory failure , either cardiac or peripheral in which acute renal ischemia occurs.

Hemoglobin uric and myoglobin uric nephrosis are also included in this category.

In ruminants severe bloat can interfere with cardiac output and lead to renal ischemia.

**Renal causes include:**

Glomerulonephritis ,interstitial nephritis, pyelonephritis ,embolic nephritis and amyloidosis .

**Postrenal uremia**

May also occur ,specifically complete obstruction of urinary tract by vesicle or urethral calculus, or more rarely by bilateral urethral obstruction .

Internal rupture of any part of the urinary tract will also cause post renal uremia.

**Pathogenesis of renal insufficiency and renal failure**

Damage to the glomerular epithelium destroys its selective permeability and permits the passage of plasma protein into the glomerular filtrate .

The protein is principally albumin ,probably because it has a lower molecular weight that than globulins.

Glomerular filtration may cease completely when there is extensive damage to glomeruli ,particularly if there is acute swelling of the kidney ,but it is belived that anuria in the terminal stages of acute renal disease is caused by back diffusion of all glomerular filtrate through the damage tubular epithelium renal than failure of filtration.

When the kidney damage is less severe ,the remaining nephrous compensate to maintain total glomerular filtration by increasing their filtration rates . when this occurs ,the volume of glomerular filtration may exceed the capacity of the tubular epithelium to reabsorb fluid and solutes .

The tubules may be an able to achieve normal urine concentration , As a result, an increased volume of urine with a constant specific gravity is produce and solute diuresis occurs. this is exacerbated if the tubular function of the compensating nephrous is also impaired.

The inability to concentrate urine is clinically evident as poly uric and is characteristic of developing renal insufficiency .

Deceased glomerular filtration also results in retention of metabolic waste products such as urea .

Phosphate and sulfate retention also occurs when total glomerular filtration is reduced and may contribute to renal metabolic acidosis .

Phosphate retention also causes a secondary hypocalcemia . due in part to an increase in calcium excretion in the urine.

In horses the kidneys are an important route of excretion of calcium so the decreased glomerular filtration rate may resulting hypercalcemia if there is a large dietary intake of calcium.

Variations in serum potassium level also occur and appear to depend on potassium intake .

Hyperkalemic can be a serious complication in renal insufficiency in man when it is one of the principal causes of the myocardial asthenia and fatal heart failure which occur in uremia .

Loss of tubular receptive function is evidenced by a continued loss of sodium , hypernatremia eventually occur in all cases of renal failure .

The continuous loss of large quantities of fluid due to solute diuresis may cause clinical dehydration.

The terminal stage of renal insufficiency renal failure is the result of the cumulative effects of impaired renal excretory and homeostatic function.

Continued loss of large volumes of dilute urine causes dehydration .

If other circulatory emergencies arise .acute renal ischemia may result leading to acute renal failure.

Prolonged hypoproteinemia results in rapid loss of body condition and muscle weakness .

Acidosis is also a contributing factor to muscle weakness and mental attitude  Hyponatremia and hyperkalemia cause skeletal muscle weakness and myocardial asthenia .

**Clinical features of urinary tract disease**

The major clinical manifestation of urinary tract disease are :

1-Abnormal constituents of urine.

2-Variations in daily urine flow .

3-Abdominal pain and painful and difficult urination (dysuria and strong urine).

4-Abnormal size of kidneys.

5-Abnormalities of the bladder and urethra .

6-Acute and chronic renal failure .

Abnormalities of urine constituents protein uric.

proteinuria is observed in normal foals ,calves ,kids and lambs in the first 40 hours after they receive colostrum .

Protein may be present in hemoglobinuria ,myoglobinuria ,and hematuria ,and when urinary tract infection are present. These causes for protein in urine should be excluded before making a diagnosis of renal diseases .

The demonstration that proteinuria originates in the kidney is easier if elements that from in the kidney such as casts one also present in the urine .

Proteinuria is often designated as albuminuria because of the high proportion of albumin present , it occur in :glomerulonephritis ,renal infarction ,tubular nephrosis and a myloidosis .

Small amounts of protein may be present in the urine of animals suffering from fever and toxemia .

Plasma protein enters urine when glomerular permeability is increased .

Protein also can enters urine when tubular cells in acute tubular nephrosis.

**Casts and cells**

Casts one organized ,tubular structures which vary in appearance depending on their composition .

The occur only when the kidney is involved in the disease process.

The one present as one indication of inflammatory of degenerative changes in the kidney where they from by agglomeration of desquamated cells and Tamm- Horsefall protein .

Erythrocytes , leukocytes and epithelial cell in urine may originate in any part of the urinary tract.

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Hematuria:-

Hematuria can result from pre renal causes when vascular occurs , such as trauma to the kidney ,septicemia and purpura hemorrhagica.

Renal causes include:-

Acute glomerulonephritis ,renal infarction embolism of renal ,tubular damage as caused by toxic insult ,and pyelonephritis.

Post renal hematuria occur particularly in urolithiasis and cystitis .

A special instance of hematuria is enzootic hematuria of cattle when hematuria originates from tumors of the urinary bladder .

In horses ,there is a syndrome of ulceration of the proximal urethra that causes hematuria at the end a urination .

In severe cause of hematuria blood may be voided as grossly visible clots but more commonly it causes a deep red to brown coloration of the urine.

Less severe causes may show only cloudiness which settles to from a red deposit on standing .

Hemoglobinuria:-

False Hemoglobinuria can occur in hematuria when erythrocytes are lysed and release their hemoglobin.

True hemoglobinuria causes a deep red to brown coloration of urine and gives appositive reaction to biochemical tests for hemoglobin .

There is no erythrocyte debris in sediment .

Myoglobinuria:-

The presence of myoglobin (myohemoglobin) in the urine is evidence of sever muscle damage

The only notable occurrence in animals is azoturia of horses

The myoglobin molecule is much smaller than hemoglobin and passes the glomerulus much more readily.

As with hemoglobin ,myoglobin can contribute to uremia.

Pyuria:-

Leukocytes or pus in urine ,indicates inflammatory exudation at some point in the urinary tract ,usually the renal pelvis or bladder.

Pyuria may occur as grossly visible clots or shreds, but often is detectable only by microscopic examination of urine sediment .

Crystalluria:-

Crystalluria should not be overinterpreted in farm animals .

Crystals in the urine of herbivorous animals have no special significance unless they occur in very large numbers and are associated with clinical signs of irritation of the urinary tract.

Calcium carbonate and triple phosphate crystals are commonly present in normal urine .

If they occur in large numbers it may suggest that the urine is concentrated and indicate the possible future development of urolithiasis .

Glycosuria and Ketonuria:-

Glycosuria in combination with Ketonuria occur only in diabetes mellitus ,a rare disease in large animals .

Glycosuria may occur in association with enterotoxaemia due to *Clostridium perfringens* type D and may occur after parenteral treatment with dextrose solution , adrenocorticotropic hormones or cortisone analogs .

It occurs also in acute tubular nephrosis due to failure of tubular resorption.

Ketonuria is a more common finding in ruminants .It occur in starvation ,acetonemia of cattle and pregnancy toxemia of ewes and does.

Variation in daily urine flow

An increase or decrease in urine flow is often described in animals .

**Polyuria:**

Polyuria occur when there is an increase in the volume of urine produced .

Polyuria can result from extra renal causes as when horses habitually drink excessive quantities of water ,and less commonly , in central diabetes insipidus when there is inappropriate section or antidiuretic hormone (ADH) from the pituitary central diabetes insipides is mast common in horses suffering from pituitary tumors.

**Oliguria and anuria:**

Reduction in the daily output (oliguria ) and complete absence of urine (anuria) occur under the same conditions and vary only in degree .

In dehydrate animals ,urine flow naturally decreases in an effort to conserve water as plasma osmotic pressure increases .

Congestive heart failure and peripheral circulatory failure may cause such a reduction in renal blood flow that oliguria follows.

Complete anuria occurs most commonly in urethral obstruction ,although it may result from acute tubular nephrosis .

Oliguria occur in the terminal stages of all forms of nephritis .

Anuria and polyuria lead to retention of solutes and disturbances of acid-base balance that contribute to the pathogenesis of uremia .

**Pollakiuria :-**

This is an abnormally frequent passage of urine .

It may occur with or without an increase in the volume of urine excreted and is commonly associated with the disease of lower urinary tract such as cystitis ,the presence of calculi in the bladder ,urethritis , and partial obstruction of the urethra .

**Dribbling**:

Is a steady ,intermittent passage of small volumes of urine , sometime precipitated by a change in pasture or increase in intra – abdominal pressure.

Reflecting inadequate or lack of sphincter control .Dribbling occurs in large animals with incomplete obstructive urolithiasis and from persistent urachus .

**Abdominal pain ,painful and difficult urination (Dysuria ,and stranguria )**

Abdominal pain and painful urination (dysuria)and difficult and slow urination (stranguria) are manifestation of discomfort caused by disease of the urinary tract .

Acute abdominal pain from urinary tract disease occur only rarely and is usually associated with sudden distention of the renal pelvis or ureter ,infarction of the kidney .

None of these condition is common in animals ,but occasionally cattle affected with pyelonephritis may have short episodes of acute abdominal pain ,due to either renal infarction or obstruction of the pelvis by necrotic debris.

During these acute attacks of pain , the cow may exhibit downward arching of the back ,paddling with the hind feet , rolling and bellowing .

Abdominal pain from urethral obstruction and distention of the bladder is manifested by tail – switching ,kicking at the belly, and repeated straining efforts at urination accompanied by grunting .

Horses with acute tubular nephrosis following vitamin k3 administration may show renal colic with arching of the back ,backing into corners and rubbing of the perineum and tail head .

**Dysuria or painful of difficult urination**

Occurs in cystitis ,vesicle calculus and urethritis and is manifested by the frequent passage of small amounts of urine .

Grunting may occur with painful urination and the animal may remain in the typical pasture after urination is completed .

**Stranguria:** Is slow and painful urination associated with disease of the lower urinary tract including cystitis ,visical calculus ,urethral obstruction and urethritis .

The animal strains to pass each drop of urine .Groaning and straining may precede and accompany urination when there is urethral obstruction .

In urethritis ,groaning and straining occur immediately often urination has ceased and gradually disappear and do not recur until urination has been repeated .

Morphological abnormalities of kidney and ureters

Enlargement or decreased size of kidney may be palpable on rectal examination or defected by ultrasonography .

Palpable abnormalities of the bladder and urethra:-

Abnormalities of the bladder: which may be palpable by rectal examination include: gross enlargement of the bladder , rupture of the bladder ,a shrunken bladder following rupture ,and palpable abnormalities in the bladder such as cystic calculi .

Abnormalities of the urethra:

include :enlargement and pain of the pelvic urethra and its external aspects in male cattle with obstruction urolithiasis and obstruction of the urethral process of male sheep with obstructive urolithiasis .

Acute and chronic renal failure:

The clinical findings of urinary tract disease vary with the rate of development and stage of the disease . In most cases ,the clinical signs are those of the initiating cause .In horses ,mental depression ,colic and diarrhea are common with oliguria or polyuria .

Cattle with uremia are similar and in addition are frequently recumbent And may have a bleeding diathesis .

In chronic renal disease of all species , there is a severe loss of body weight ,anorexia ,polyuria ,polydipsia and ventral edema .

Uremia:-

Uremia is the systemic state which occur in the terminal stages of renal insufficiency .

Anuria or oliguria may occur with uremia . Oliguria is more common unless there is complete obstruction of the urinary tract .

Chronic renal disease is usually manifested by polyuria ,but oliguria appears in the terminal stages when clinical uremia develops .

The uremia animal is depressed and anorexia with muscular weakness and tremor .

In chronic uremia ,the body condition is poor ,due probably to continued loss of protein in the urine , dehydration and anorexia .

The respiration is usually increased in rat and depth but is not dyspneic .

In the terminal stage it may become periodic in character .

The heart rate is markedly increased because of terminal dehydration and myocardial asthenia .but the temperature remain except in infection processes and some cases of acute tubular nephrosis .

The animal becomes recumbent and comatose in the terminal stage .

The temperature falls to below normal and death occurs quietly , the whole course of the disease having been one of gradual intoxication .

Special examination of the urinary system:

Renal function tests : by restraining the animal on clean ,dry floor , which is examined periodically to determine whether or not urine is being voided. Renal function tests evaluate the functional capability of the kidney and, in general, assess blood flow to the kidneys, glomerular filtration and tubular function.

Test of urine : specific gravity normal specific gravity is (1.028-1.032) in chronic diseases this decreases to about (1.010).

Test of blood : blood urea and creatinine determinations are essential components of an evaluation of urinary system .

3

Principles of treatment of urinary tract disease

**Fluid and electrolytes :**

Treatment acute renal failure in all species is aimed at removing the primary cause and restoring at normal fluid balance by correction dehydration ,acid – base disorders and electrolyte abnormalities .

The prognosis for acute renal failure will depend on the initiating cause and severity of the lesion .

If the acute disease process can be stopped the animal may be able to survive on its remaining function renal tissue.

Balanced electrolyte solutions or normal saline supplemented with potassium and calcium can be used to correct fluid and electrolyte deficits .

If anuria or oliguria is present the rate of fluid administration should be monitored to prevent over hydration .

If the patient has oliguria often the fluid volume deficit is corrected a diuretic may be used to help restore urine flow .

Furosemide (1-2 mg /kg Bw every 2 hours ) or mannitol(o.25 -2.0 g/kg B.w in a20% solution ) may be used until .

Diuretics should not be used until dehydration has been corrected .

Animals that remain anuria have a grave prognosis and can only be managed with peritoneal of vascular dialysis .

In chronic failure ,therapy is aimed at prolonging life .Animal in chronic failure should have free access to water and salt , un less edema is present .

In food –producing animal ,emergency slaughter is not recommended because the carcass is usually unsuitable for human consumption .

**Antimicrobials:**

Selection of antimicrobials for the treatment of urinary tract infections should be based on quantitative urine culture .

The ideal antimicrobial for the treatment of urinary tract infections should meet several criteria , it should have :-

-Activity against the caused bacteria .

- Be exacted and concentrated in the kidney and urine .

-Be active at the PH of urine .

-Low toxicity .

-Be easily administered.

- Low in cost .

-No harmful interactions with other concurrently administered drugs.

Appropriate first line antimicrobials include penicillin in ruminant and trimethoprim –sulfa in horses.

Administered therapy for lower urinary tract infection should continue for at least 7 days .for upper urinary tract infection 2-4 weeks of treatment is often necessary .success of therapy can be evaluated by repeating the urine culture 7-10 days after the last treatment

DISEASES OF THE KIDNY

Nephrosis:

Includes degenerative and inflammatory lesion primarily affecting renal tubules .

It can occur as a sequel to renal ischemia and following toxic insult to the kidney .

Nephrosis is the most common cause of acute kidney failure .

Renal ischemia :

Reduced blood flow through the kidneys the kidney usually results from general circulatory failure . there is transitory oliguria followed by anuria and uremic if the circulatory failure is not corrected.

**Etiology** : is may be acute or chronic

1- Acute renal ischemia

General circulatory emergencies such as shock ,dehydration acute hemorrhagic anemia ,acute heart failure.

Embolism of renal artery ,recoded in horses .Extreme ruminal distention in cattle.

2- Chronic renal ischemia

Chronic circulatory insufficiency such as congestive heart failure .

**Pathogenesis:**

Acute ischemia of the kidneys occurs when compensatory vasoconstriction affects the renal blood vessels in response to a sudden reduction in cardiac output. As blood pressure falls ,glomerular filtration decreases and metabolites that are normally excreted accumulate in the blood stream .

The concentration of urea in the blood increases giving rise to the name pre -renal anemia .As glomerular filtration falls , tubular reception increases causing reduced urine flow .

The nephrosis of hemoglobin urea appears to be caused by the vasoconstriction of renal vessels rather than a direct toxic effect of hemoglobin on renal tubules . with uremia in acute hemolytic anemic and in acute muscular dystrophy with myoglobinuric may be exacerbated by plugging of the tubules costs of coagulated proteins .but ischemia is also an important factor.

**CLINICAL FINDINGS**

Oliguria and azotemia will go unnoticed in most cases if the circulatory defect is corrected in the early stages. The general clinical picture is one of acute renal failure

and is described under uremia.

**TREATMENT**

Treatment must be directed at correcting fluid, electrolyte and acid-base disturbance as soon as possible

Diseases of the bladder, ureters and urethra

**CYSTITIS:-**

Inflammation of the bladder is usually associated with bacterial infection and is

characterized clinically by frequent, painful urination (pollakiuria and dysuria)

and the presence of blood (hematuria), inflammatory cells and bacteria in the

urine.

**ETIOLOGY**-

Cystitis occurs sporadically as a result of the introduction of infection into the

bladder when trauma to the bladder has occurred or when there is stagnation of

the urine.

In farm animals the common associations are:

- Cystic calculus

- Difficult parturition

- Contaminated catheterization

- Late pregnancy

- As a sequel to paralysis of the bladder.

In the above cases, the bacterial population is usually mixed but predominantly

E. coli. There is also the accompaniment of specific pyelonephritis in cattle

associated with C. renale.

**PATHOGENESIS:**

Bacteria frequently gain entrance to the bladder but are usually removed by the

flushing action of voided urine before they invade the mucosa. Mucosal injury

facilitates invasion but stagnation of urine is the most important predisposing cause.

Bacteria usually enter the bladder by ascending the urethra but descending

infection from embolic nephritis may also occur.

**CLINICAL FINDINGS**

The urethritis that usually accompanies cystitis causes painful sensations and the desire to urinate. Urination occurs frequently and is accompanied by pain and sometimes grunting; the animal remains in the urination posture for some minutes after the flow has ceased, In very acute cases there may be moderate abdominal pain, as evidenced by treading with the

Hind feet, kicking at the belly and swishing with the tail, and a moderate febrile reaction. Chronic cases show a similar syndrome but the signs are less marked. Frequent urination and small volume are the characteristic signs. In chronic cases, the bladder wall may feel thickened on rectal examination and, in horses, a calculus may be present.

**CLINICAL PATHOLOGY**

Blood and pus in the urine is typical of acute cases and the urine may have a strong ammonia odor.

Microscopic examination of urine sediment will reveal erythrocytes, leukocytes, and desquamated epithelial cells.

bacterial culture

**TREATM ENT**

Antimicrobial agents are indicated to control the infection.

**UROLITHIASIS IN RUMINANTS**

Urolithiasis is common as a subclinical disorder among ruminants raised in management systems where the ration is composed primarily of grain or where animals graze certain types of pasture. In these situations, 40-60 % of animals may form calculi in their Urinary tract.

Urolithiasis becomes an important clinical disease of castrated male ruminants when calculi cause urinary tract obstruction, usually obstruction of the urethra. Urethral obstruction is characterized, clinically by complete retention of urine, frequent unsuccessful attempts to urinate and distension of the bladder. Urethral perforation and rupture of the bladder can be squeal. Mortality is high in cases of urethral obstruction and treatment is surgical.

**ETIOLOGY**

Urinary calculi, or Uroliths, form when inorganic and organic urinary solutes are precipitated out of solution. The precipitates occur as crystals or as amorphous , deposits'. Calculi form over a long period by a gradual accumulation of precipitate around a nidus. An organic matrix is an integral part of most types of calculus. Several factors affect the rate of Uroliths

.formation, including

- conditions that affect the concentration of specific solutes in urine,

- the ease with which solutes are precipitated out of solution,

- the provision of a nidus and the tendency to concretion of precipitates.

**EPIDEMIOLOGY**

**●Species affected**: Urolithiasis occurs in all ruminant species but is of greatest economic importance in feeder steers and wethers (castrated lambs) being fed heavy concentrate

rations, and animals on range pasture in particular problem areas.

These range areas are associated with the presence of pasture plants containing large quantities of oxalate, estrogens, or silica. When cattle graze pasture containing plants with high levels of silica, uroliths occur in animals of all ages and sexes The prevalence of uroliths is about the same in cows, heifers, bulls, and steers grazing on the same pasture and they may even occur in newborn calves. Females and bulls usually pass the calculi and obstructive urolithiasis is primarily a problem in castrated male animals. Obstructive urolithiasis is the most common urinary tract disease in breeding rams and goats There are three main groups of factors that contribute to urolithiasis:

- Those that favor the development of a nidus about which precipitation and concretion can

occur.

- Those that facilitate precipitation of solutes on to the nidus

- Those that favor concretion by cementing precipitated salts to the developing calculus

**●Nidus formation**

A nidus favors the deposition of crystals about itself. A nidus may be a group of desquamated epithelial cells or necrotic tissue that may be formed as a result in occasional cases from local infection in the urinary tract. When large numbers of animals are affected it is probable that some other factor, such as a deficiency of vitamin A or the administration of

estrogens, is the cause of excessive epithelial desquamation.

**●Precipitation of solutes:**

Urine is a highly saturated solution containing a large number of solutes, many of them in higher concentrations than their individual solubilities permit in a simple solution.

Several factors may explain why solutes remain in solution. Probably the most important factor in preventing precipitation is the presence of protective colloids that convert urine into a gel. These colloids are efficient up to a point, but their capacity to maintain the solution may be overcome by abnormalities in one or more of a number of other factors.

The physical characteristics of urine, the amount of solute presented to the kidney for excretion and the balance between water and solute in urine all influence the ease of calculus formation .

**●The pH of urine:**

Affects the solubility of some solutes, mixed phosphate and carbonate calculi being more readily formed in an alkaline than an acid medium.

**●Ammonium chloride**

or phosphoric acid added to the rations of steers increases the acidity of the urine and reduces the incidence of calculi. In contrast, variations in pH between 1 and 8 have little influence on the solubility of silicic acid, the form of silica excreted in the urine of ruminants.

**●The amount of solute presented to the kidney for excretion is influenced**

**by the diet.**

Some pasture plants can contain up to 6% silica. The kidney is the major route of excretion of absorbed silicic acid. The urine of these animals often becomes super saturated with silicic acid, which promotes the polymerization or precipitation of the silicic acid and calculus formation.

**●Feeding sodium chloride prevents the formation of silica calculi**:

by reducing the concentration of silicic acid in the urine and maintaining it below the saturation concentration. Sheep with a high dietary intake of phosphorus have an increased concentration of phosphorus in their urine and an increased development of calculi.

**●Diets high in magnesium** such as some calf milk replacers have also been associated with an increase Supplemental calcium in the diet helps prevent calculus formation when phosphate or magnesium4 intake is high. sing incidence of obstructive urolithiasis.

**●Ingestion of plants with a high oxalic acid**: content can be a risk factor for formation of calcium carbonate calculi in sheep.

**Feeding practices**: can influence the function of the kidney and may contribute

to calculus formation.

**Factors favoring concretion**:

Most calculi, and siliceous calculi in particular, are composed of organic matter as well as minerals. This organic component is mucoprotein, particularly its mucopolysaccharide fraction. It acts as a cementing agent and favors the formation of calculi when precipitates are present. The mucoprotein content of urine of feeder steers and lambs is increased by heavy concentrate-low roughage rations, by the feeding of pelleted rations, and, combined with a high dietary intake of phosphate, may be an important cause of urolithiasis in this class of livestock. These high levels of mucoprotein in urine may be the result of a rapid turnover of supporting tissues in animals that are making rapid gains in weight.

**M iscellaneous factors in thedevelopment of urolithiasis**

Stasis of urine favors precipitation of solutes, probably by virtue of the infection that commonly follows, providing cellular material for a nidus. Certain feeds, including cottonseed meal and milo sorghum, are credited with causing more urolithiasis than other feeds. Alfalfa is in an indeterminate position: by some observers it is thought to cause the

formation of calculi.

Pelleting appears to increase calculi formation if the ration already has this tendency.

In feedlots a combination of high mineral feeding and a high level of mucoprotein in the urine associated with rapid growth are probably the important factors in most instances. In range animals a high intake of mineralized water, or oxalate or silica in plants, are most commonly associated with a high incidence of urinary calculi, Limited water intake at weaning and in very cold weather may also be a contributory factor.

**Composition of calculi**

The chemical composition of urethral calculi varies and appears to depend largely on the dietary intake of individual elements. Cattle and sheep grazing these pastures have a high prevalence of siliceous calculi. Calculi containing calcium carbonate are more common in animals on clover-rich pasture, or when oxalate-containing plants abound. Calcium, ammonium, and magnesium carbonate are also common constituents of calculi in cattle and sheep at pasture. Sheep and steers in feedlots usually have calculi composed of struvite, magnesium ammonium phosphate. High concentrations of magnesium in feedlot rations also cause a high prevalence of magnesium ammonium phosphate calculi in lambs.

**Estrogenic subterranean clover** cancause urinary tract obstruction. obstruction is promoted byestrogenic stimulation of squamous metaplasia of the urethral epithelium, accessory sex glandular enlargement and mucus secretion. Pastures containing these plants are also reputed to cause urinary obstruction by calculi consisting of calcium carbonate.

**Occurrence**

Urethral obstruction may occur at any site but is most common at the sigmoid flexure in steers and in the vermiform appendage or at the sigmoid flexure in wethers or rams, all sites where the urethra narrows. Urolithiasis is as common in females as in males, but obstruction rarely if ever occurs because of the shortness and large diameter of the

urethra.

**PATHOGENESIS**

Urinary calculi are commonly observed at necropsy in normal animals, and in many

appear to cause little or no harm. Calculi 'may be present in kidneys, ureters, bladder, and urethra. In a few animals pyelonephritis, cystitis, and urethral obstruction may occur. Obstruction of one ureter may cause unilateral hydronephrosis, with compensation by the

contralateral kidney. The major clinical manifestation of urolithiasis is urethral obstruction, particularly in wethers and steers. This difference between urolithiasis and obstructive urolithiasis is an important one. Simple urolithiasis has relatively little importance but obstructive urolithiasis is a fatal disease unless the obstruction is relieved. Rupture of the urethra or bladder occurs within 2-3 days if the obstruction is not relieved and the animal

dies of uremia or secondary bacterial infection. Rupture of the bladder is more likely to occur with a spherical, smooth calculus that causes complete obstruction of the urethra. Rupture of the urethra is more common with irregularly shaped stones that cause partial obstruction and pressure necrosis of the urethral wall.

**CLINICAL FINDINGS**

Calculi in the renal pelvis or ureters are not usually diagnosed ante mortem although obstruction of a ureter may be detectable on rectal examination, especially if it is accompanied by hydronephrosis. Occasionally the exit from the renal pelvis is blocked and the acute distension that results may cause acute pain, accompanied by stiffness of the gait and pain on pressure over the loins. Calculi in the bladder may cause cystitis and are manifested by signs of that disease.

**Obstruction of the urethra by a calculus**

This is a common occurrence in steers and wethers and causes a characteristic syndrome of abdominal pain with kicking at the belly, treading with the hind feet and swishing of the tail. Repeated twitching of the penis, sufficient to shake the prepuce, is often observed, and the animal may make strenuous efforts to urinate, accompanied by straining, grunting and grating of the teeth, but these result in the passage of only a few drops of bloodstained urine. Cattle with incomplete obstruction - 'dribblers' - will pass small amounts of

bloodstained urine frequently.

On rectal examination, when the size of the animal is appropriate, the urethra

and bladder are palpably distended and the urethra is painful and pulsates on

manipulation.

In rams with obstructive urolithiasis, sudden depression, in appetence, stamping

the feet, tail swishing, kicking at the abdomen, bruxism, anuria or the passage of

only a few drops of urine are common.

**Rupture of urethra or bladder** If the obstruction is not relieved, urethral rupture or bladder rupture usually occurs within 48 hours. With urethral rupture, the urine leaks into the connective tissue of the ventral abdominal wall and prepuce and causes an obvious

fluid swelling, which may spread as far as the thorax. This results in a severe cellulitis and toxemia. The skin over the swollen area may slough, permitting drainage, and the course is rather more protracted in these cases. When the bladder ruptures there is an immediate

relief from discomfort but anorexia and depression develop as uremia develops.

**CLINICAL PATHOLOGY**

**Urinalysis**

Laboratory examinations may be useful in the diagnosis of the disease in its early

stages when the calculi are present in the kidney or bladder. The urine usually contains

erythrocytes and epithelial cells and a higher than normal number of crystals, sometimes accompanied by larger aggregations described as sand or sabulous deposit. Bacteria may also be present if secondary invasion of the traumatic cystitis and pyelonephritis has occurred.

**Serum biochemistry**

Serum urea nitrogen and creatinine concentrations will be increased before either

urethral or bladder rupture occurs and will increase even further after wards. Rupture of the bladder will result in uroabdomen. Because urine has a markedly low sodium and chloride concentration.

**Abdominocentesis and need le aspirate of subcutaneous tissue**

Abdominocentesis is necessary to detect uroperitoneum after rupture of the bladder

or needle aspiration from the subcutaneous swelling associated with urethral rupture.

**Ultrasonography**

Ultrasonography is a useful for the diagnosis of obstructive urolithiasis in rams. All parts of the urinary tract must be examined for urinary calculi.

**TREATMENT**

-The treatment of obstructive urolithiasis is primarily surgical.

- Cattle or lambs with obstructive urolithiasis that are close to being marketed can be slaughter.

-It used to be thought that calculi cannot be dissolved by medical means, but recent studies suggest that administration of specific solutions into the bladder can rapidly dissolve most uroliths.

- In early stages of the disease or in cases of incomplete obstruction, treatment with

smooth muscle relaxants such as phenothiazine derivatives (aminopromazine,

0.7 mg/kg of BW).

-Animals treated medically should be observed closely to insure that urination occurs and

that obstruction does not recur.

**PREVENTION**

A number of agents and management procedures have been recommended in

the prevention of urolithiasis in feeder lambs and steers.

1- the diet should contain an adequate balance of calcium and phosphorus

to avoid precipitation of excess phosphorus in the urine.

The ration should have a Ca:P ratio of 1.2:1, but higher calcium inputs (1.5-2.0:1)

have been recommended.

2-Every practical effort must be used to increase and maintain water intake in feeder steers

.

3-The salt fed at a concentration of 3-5 % under practical conditions higher concentrations causing lack of appetite.

4-supplementary feeding with sodium chloride helps to prevent urolithiasis by decreasing the rate of deposition of magnesium and phosphate around the nidus of a calculus.

5- Feeding of pelleted rations may predispose to the development of phosphate calculi by reducing the salivary secretion of phosphorus.

6-The control of siliceous calculi in cattle which are fed native range grass hay, which may contain a high level of silica, is dependent primarily on increasing the water intake.

7-An alkaline urine (pH > 7.0) favors the formation of phosphate-based stones and calcium-carbonatebased stones. Feeding an agent that decreases urine pH will therefore protect against phosphate-based stones.

8- The feeding of ammonium chloride (45 g/ daily to steers and 10 g daily to sheep) may

prevent urolithiasis due to phosphate calculi, but the magnitude of urine acidification achieved varies markedly depending on the acidogenic nature of the diet. For this reason, urine pH should be closely monitored when adding ammonium chloride to the ration, because clinically relevant metabolic acidosis.

9- An acidic urine (pH < 7.0) favors the formation of silicate stones, so ammonium chloride manipulation of urine pH is not indicated in animals at risk of developing siliceous calculi.

10-When the cause of urolithiasis is due to pasture exposure, females can be used to graze the dangerous pastures since they are not as susceptible to developing urinary tract obstruction.

10-In areas where the oxalate content of the pasture is high, wethers and steers should be permitted only limited access to pasture dominated by herbaceous plants.

11- the adequate intake of vitamin A especially during drought periods and when animals are fed grain rations in feedlots.

12-Deferment of castration, by permitting greater urethral dilatation, may reduce the incidence of obstructive urolithiasis but the improvement is unlikely to be significant.