

Diseases of the urinary system

Anatomy and physiology:

The composition of blood is kept constant mainly through the selective elimination of water and solutes by the kidneys rather than by these materials in food. This control involves balancing the body's input of ions and water with the amounts excreted. As Na^+ and Cl^- are the most abundant somatically active solutes in plasma, control of plasma volume and tonicity can be largely achieved by controlling the amounts of these ions and water excreted.

Functions of the kidney:

1- Excretory: Excretion of water products and drugs in the urine.

2- Regulatory: The kidney regulates the volume, osmotic pressure and reaction of the blood.

3-Endocrine: The kidney produces the following hormones.

a) Rennin- angiotensin system: It is produced by the juxtaglomerular apparatus which is made of specialized cells on the smooth muscle cells located on the afferent glomerular arteriole as it enters the glomerulus. These cells secrete rennin which converts angiotensinogen in the blood to angiotensin I. Angiotensin II is generated from angiotensin I by angiotensin converting enzyme. Angiotensin II is both a vasoconstrictor and the most important stimulus for release of aldosterone.

b) Erythropoietin: is a glycoprotein produced mainly by the kidney and is one of the major stimuli of erythropoiesis.

c) Prostaglandin: The kidney produces prostaglandin E_2 , a powerful vasodilator agent.

4-Metabolic:

a) Vitamin D metabolism: Naturally occurring vitamin D requires hydroxylation in the liver and again by the kidney to produce 1,25 dihydroxychole-calciferol. Loss of this metabolic activity in diseased kidney results in renal osteodystrophy.

b) Protein and polypeptide hormones: The kidney is the major site for catabolism of insulin, parathyroid hormone and calcitonin. In renal failure, the metabolic clearance of these hormones is reduced.

Gross structure of the kidney:

The basic unit of kidney function is the nephron of which there are about 1,000,000 nephrons in each kidney. Urine formed in the tubular part of the nephron collects in the renal pelvis and then flows through the ureter to the bladder for subsequent elimination via the urethra (Figs 1 and 2).

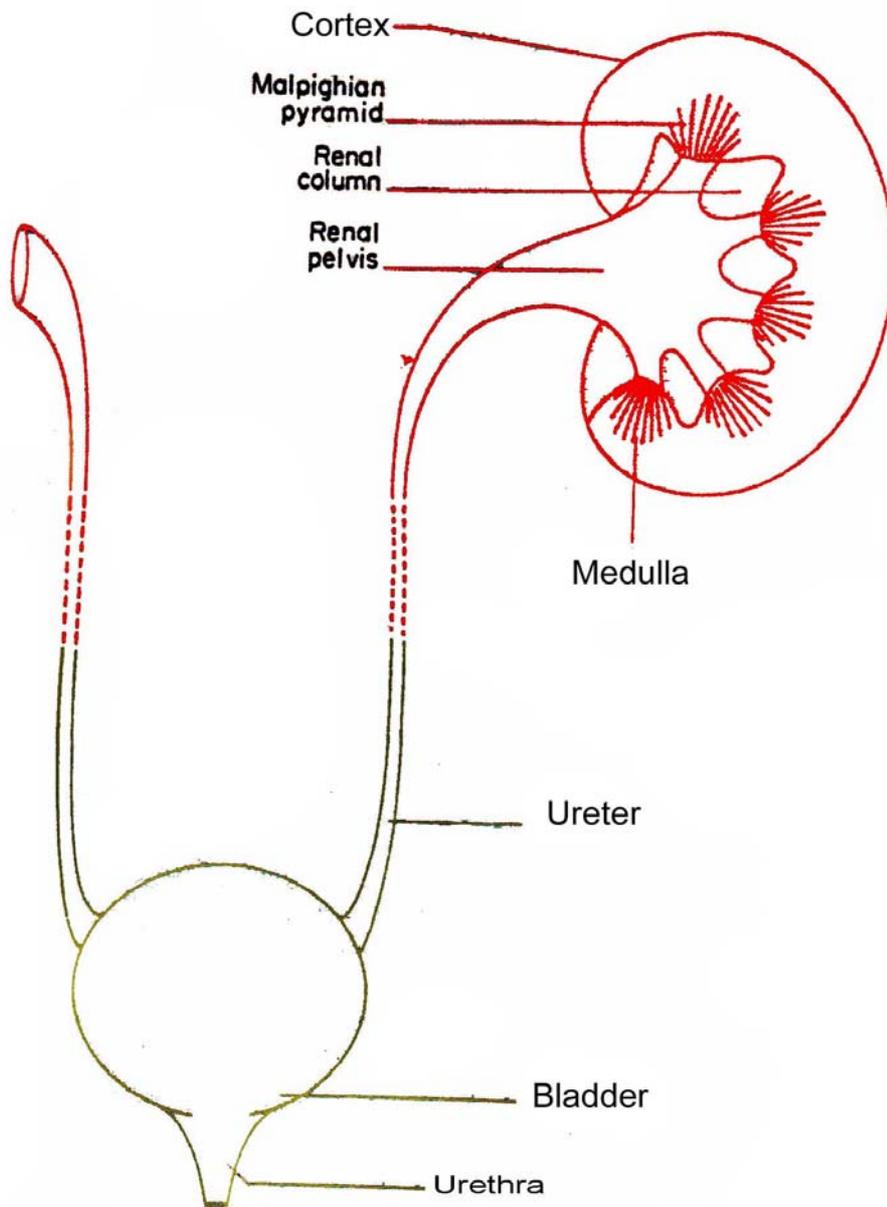


Fig. 1: Diagrammatic represent the normal anatomical arrangement of urinary system

The glomerulus (about 200 μm in diameter) is formed by invagination of a tuft of 50 anatomizing capillaries into the dilated blind end of the nephron (Bowman's capsule). The capillaries are supplied by an afferent arteriole & drained by a slightly smaller efferent arteriole. The glomerular membrane separates blood from the glomerular filtrate in Bowman's capsule, that is composed of three layers: -

- 1- Endothelial layer of the capillary.
- 2- Basement membrane.
- 3- Layer of epithelial cells.

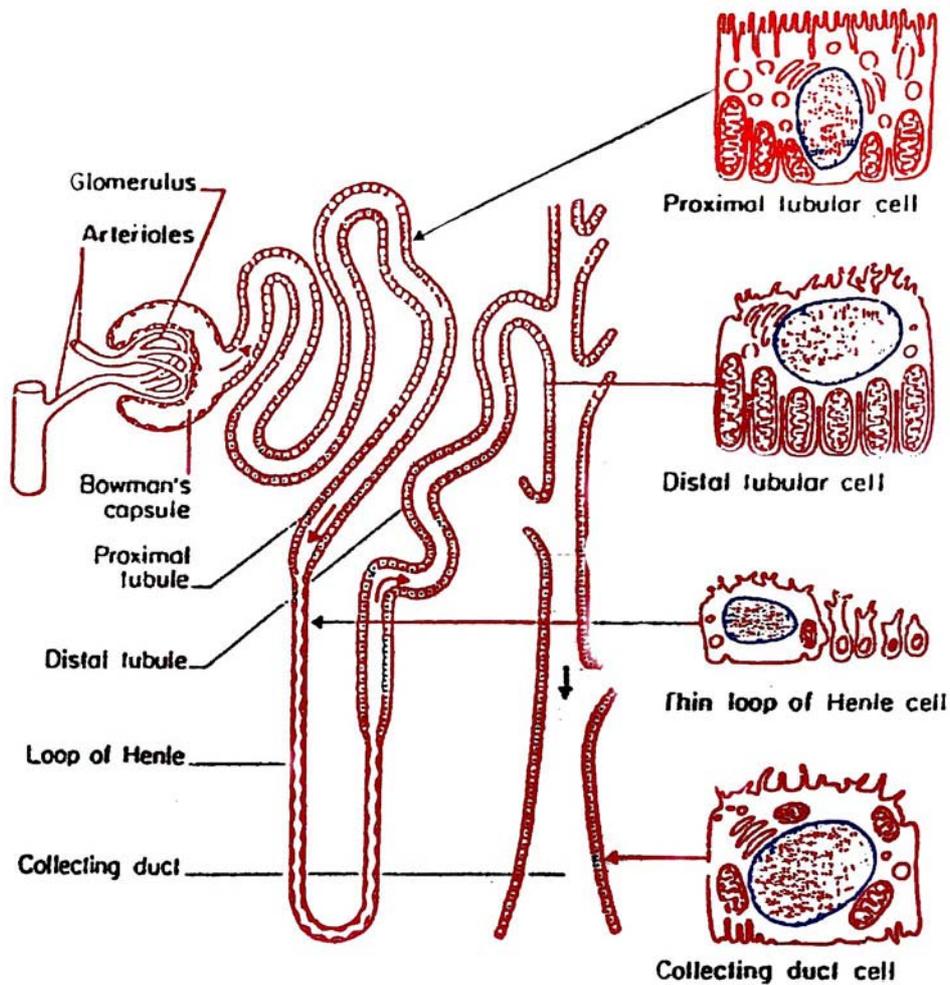


Fig. 2: Diagrammatic represent the normal histological structure of the nephron.

The permeability of the glomerular membrane is 100-500 times that of the usual capillary. The endothelial cells lining the glomerulus have thousands of small holes (fenestrae). Outside the endothelial cells is a basement membrane, composed of a meshwork proteoglycan fibrillae, which has also large spaces through which fluid can filter. A layer of epithelial cells lines the outer surface of the glomerulus. These cells consist mainly of finger like projections that cover the basement membrane. These fingers form slits called slit-pores through which the glomerular filtrate filters.

Functionally the glomerular membrane permits the passage of substances up to 4 nm in diameter & does not allow the passage of those with diameter greater than 8 nm.

The proximal convoluted tubules (PCT) is made of a single layer of cells which show on their luminal edges brush border due to the presence of numerous microvilli.

The structure of the loop of Henle differs according to its location in the kidney.

Cortical nephrons have short loops of Henle with a thin descending limb & a thick ascending limb. The cells lining the thick segment of the loop of Henle form a larger lumen & lack of prominent brush borders. In contrast, juxtamedullary nephrons have somewhat longer loops of Henle and thin segments on both sides of the loop.

The distal convoluted tubule (DCT) has a similar structure to the thick segment of the loop of Henle. DCT from large number of different nephrons drain into a common collecting duct & then via a papillary duct into the renal pelvis. The largest collecting ducts empty through the renal pelvis through the tips of the renal papillae, which protrude into the renal calyces. A kidney has about 250 large collecting ducts, each of which transmits the urine from 4000 nephrons. The epithelial lining of the collecting duct is composed of a layer of cuboidal cells, which gradually become taller as the collecting tubule merges into capillary duct. Nephrons are arranged into 10-15 groups called malpighian pyramids. The nephrons are all oriented so that the Bowman's capsules with related proximal & distal tubules are situated in the outer layers of the kidney cortex while the loops of Henle & Malpighian pyramids form the medullary rays.

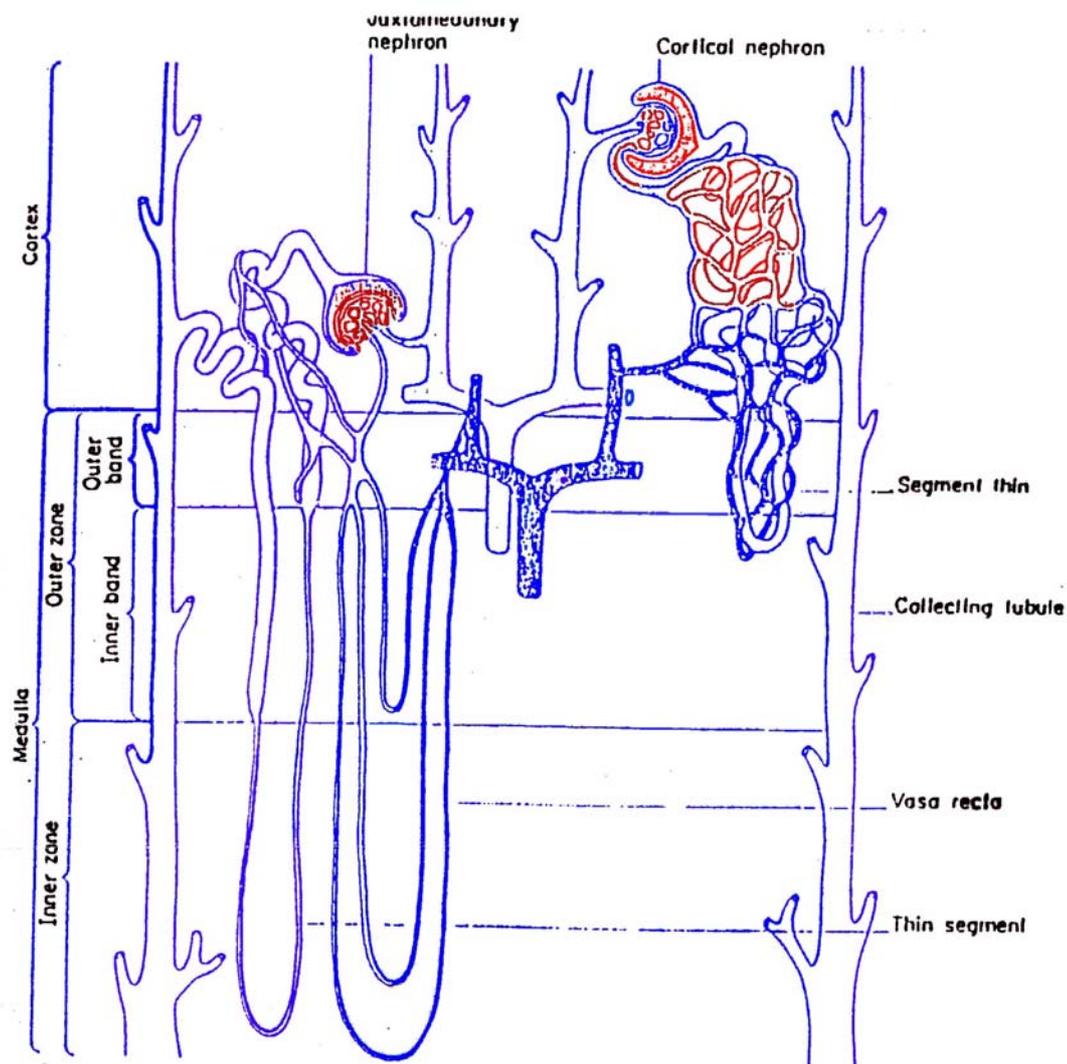


Fig 3: Diagrammatic representation of the blood supply to cortical and justamedullary Nephrons.

Diseases of the urinary system

Principles of renal insufficiency:

Disease of the kidneys, and in some instances of the ureters, bladder and urethra, reduce the efficiency of the kidney's functions, disturbances in protein, acid-base solute and water homeostasis and in excretion of the metabolic end-products will result.

It is noteworthy to mention that, diseases of the bladder and urethra are more common and more important in farm animals than the diseases of the kidneys; but some studies on renal insufficiency is necessary because many disease condition such as pyelonephritis, embolic nephritis, amyloidosis and nephrosis, all may eventually lead to renal insufficiency or renal failure.

Renal insufficiency:

It is defined as a degree or relative loss of renal function, but the animal can survive its state. Meanwhile, renal failure is defined as complete loss of renal tissue function and the animal can't survive or can't continue its existence.

Renal efficiency depends on the functional integrity of the individual nephrons. Renal insufficiency can occur because of:

- * Extrarenal causes - abnormalities in the rate of blood flow to the renal tissues.
- * Intrinsic factors from the kidneys such as:
 - a) Abnormalities of the glomerular filtration rate.
 - b) Abnormalities of tubular reabsorption .

The first one (extrarenal) is depending upon the vasomotor and in animals is affected only by emergencies such as in hemorrhage, shock and dehydration which may lead to renal ischemia causing tubular necrosis with eventual development of renal insufficiency.

Glomerular filtration and tubular reabsorption can be affected independently of other such as in hemoglobinuric nephrosis, the glomerular filtration is unaffected while the tubular reabsorption is depressed. However, because of the common blood supply to the glomerulus and tubule, damage to any part of the nephron is usually followed by damage of the remaining part.

The development of renal dysfunction is depends on the degree of loss of renal function. If the degree of loss, and there fore the degree of dysfunction is such that the animal is not able to continue its existence, it is said to be a state of renal failure and the clinical syndrome of uremia is manifested.

Pathophysiology of the renal insufficiency:

Damage to the glomerular epithelium destroys its selective permeability and permits the passage of plasma proteins mainly albumin into the capsular fluid. Complete cessation of the glomerular filtration may occur when there is an extensive damage to the glomeruli, particularly if there is acute swelling of the kidneys, but in many instances the anuria of the terminal stages of acute renal disease is caused by back diffusion of all glomerular filtrate through the damaged tubular epithelium.

When the renal damage is less severe, the compensatory mechanism on the part of residual nephrons is to maintain the total glomerular filtration by increasing the glomerular filtration rate. Decreased glomerular filtration is also reflected by retention of the blood urea and other nitrogenous end products of metabolism. Although blood level of urea is not significant in the production of clinical signs, they are used as a measure of glomerular filtration rate.

When the glomerular filtration is reduced, phosphate and sulphate retention also occurs and may precipitate to renal metabolic acidosis. Phosphate retention may cause secondary hypocalcaemia due to increased calcium excretion in the urine. Variations of the potassium levels in serum also occur and usually depend on potassium intake. Hyperkalemia is a serious complication and may lead to myocardial asthenia and fatal heart failure.

Hyponatremia eventually occurs in all cases of renal failure and clinical dehydration may occur due to loss of large quantities of fluids caused by solute diuresis.

Renal failure:

Renal failure may be acute or chronic depending on the progress rate of the dysfunction and the degree of loss of renal function.

1) Acute renal failure:

Pathology of acute renal failure:

*Acute renal failure is characterized by a sudden decline in the glomerular filtration rate and concurrent development of uremia.

*Renal failure may result from complete loss of function of a large number of nephrons, partial loss of function of most nephrons, or any combination of them.

*The predominant pathophysiologic mechanisms causing this decline in the filtration rate are decreased glomerular hydrostatic pressure and/or increased Bowman's capsule pressure.

*Decreased glomerular hydrostatic pressure: is usually due to hemodynamic changes including:

1-Constriction of different arterioles.

2-Fibrin occlusion of renal vasculature.

3-Changes in renal blood flow.

4-Any reflex mechanism secondary to tubular obstruction in individual nephron.

*Increased Bowman's capsule pressure:

- Tubular collapse.

- Changes in glomerular permeability.

Causes of these tubular and vascular changes or factors are probably interdependent in acute renal failure.

The causes of acute renal failure can be dividing into: -

- Hemodynamic changes.

- Toxic tubular nephrosis.

- Immunologic disorders.

- Acute inflammation and / or obstruction.

- Any condition that predisposes the patient to marked hypotension and / or release of endogenous pressor agents has the potential to initiate hemodynamically-mediated acute renal failure .

- Systemic hypotension and / or arteriole constriction, along with tubular obstruction, causes the decrease in renal perfusion.
- Endotoxemia and complement-mediated coagulopathies are involved in hemodynamic factors.
- Nephrotoxins – tubular swelling and necrosis of proximal cortical tubular cells.
- Tubular epithelial diseases – lysosomal dysfunction, mitochondrial dysfunction, abnormal protein synthesis – resulting in tubular obstruction or epithelial swelling.
- Immunologically mediated acute renal failure is usually a result of interstitial nephritis or glomerulonephritis.

Causes of acute renal failure: -

In Horse	In Ruminants
-Hemodynamic causes.	-Hemodynamic causes.
-Toxic nephrosis.	-Toxic plants.
-Immunologic causes.	-Drugs, chemicals.
	-Endogenous.
	-Mycotoxins.
	-Septic.

2) Chronic renal failure:

Pathophysiology of chronic renal failure

It is a result of slow and often progressive loss of nephron function and / or population. Failure occurs when enough nephrons are lost or their function is sufficiently decreased to affect body condition and function.

-Chronic renal failure in large animals is usually the result of progressive glomerular disease, which may be immunologically mediated, or of tubular interstitial diseases caused by obstructive, unreversed or progressive, nephrotoxic septic, hemodynamic.

-With either acute or chronic renal failure, more than two-thirds of nephron function must be lost for clinical signs of uremia to be noted.

Causes: In Horse	Causes: In Ruminants
<i>Tubulointerstitial causes:</i>	
Any of toxic, whether vascular or septic causes	Chronic obstruction
Chronic or intermittent obstruction	Chronic pyelonephritis
Chronic pyelonephritis	
v	

Granulomatous infiltration	
Neoplasia	
Glomerular causes:	
Renal Hypoplasia	Amyloidosis
Amyloidosis	glomerulonephritis
Glomerulosclerosis	

Principal manifestations or clinical features of the urinary tract disease

The major manifestations of U.T. disease are:

- Abnormalities of the urine composition.
- Abnormalities of the daily urine flow.
- Pain and dysuria.
- Uremia (principal manifestation in renal failure).
- Rupture of the U.B., renal pelvis and urethra.
- Defect in nervous control of the urination.
- Urachal leakage of urine.

(1) Abnormal constituents of urine:

(a) Proteinuria:

It means the presence of protein in the urine . Normal urine in all animal species contains little or small amount of protein from desquamation of epithelial cells and other sources, but the amount is insufficient to produce a positive reactions to the standard test . One exception is the urine of newborn calves 40 hrs old after receiving colostrums and sheds urine of high protein contents. Also, urine of equines have an increased protein level, and consequently their urine appears turbid.

Proteinuria is usually associated the following disease conditions:

- Hemoglobinuria, myoglobinuria, hematuria.
- Glomerulonephritis, renal infarction, nephrosis, amyloidosis, congestive heart failure.

Excessive excretion of protein in the urine leads to hypoproteinemia and manifested clinically as muscular weakness, general depression and reduced work capability of the animal. The clinical significance of proteinuria as an indication of renal diseases is much greater when the formed elements including casts and cells are present in the urine of the diseased animal.

Chronic proteinuria or massive acute proteinuria may cause hypoproteinemia as in case of oglomerulonephritis, acute tubular nephrosis in horses and in amyloidosis in cattle.

(b) Casts and cells:

Casts are organized, tubular structures which vary in appearance depending on their composition. They occur only when the kidneys are involved in the disease process. They present as an indication of inflammatory or degenerative changes in the kidney where they are formed by agglomeration of desquamated cells and protein. R.B.Cs, W.B.Cs and epithelial cells may originate at any part of the urinary tract.

(c) Hematuria:

It is the presence of intact blood cells in the urine. It may appear as gross blood clots passed at the beginning, during, or at the end of urination or as more uniformed discoloration of the urine throughout the urination without clots. If large clots are present, obstruction of U.T. may occur, resulting in stranguria and dysuria.

Hematuria may result from prerenal, renal or postrenal causes as following:

Prerenal causes of hematuria:

When vascular damage occurs such as:

- Trauma to kidneys.
- Septicemia.
- Purpura hemorrhagica in horses.

Renal causes of hematuria:

- Acute glomerulonephritis.
- Pyelonephritis.
- Tubular damage due to toxic insults such as sulphonamide toxicity.
- Embolism or Renal A.
- Renal infarction.

Post renal causes of hematuria:

- Urolithiasis.
- Urethritis, cystitis.
- Enzootic hematuria in cattle – tumor in U.B., which is usually accompanied by blood losses.

Causes of hematuria

	In Horse	In Ruminants
Urethra	Calculi	Calculi
	Urethritis	Urethritis
	Habranemiasis	Trauma
	Idiopathic in males (Proximal dorsal uretral HO.)	
Bladder	Calculi	Calculi

	Cystitis	Cystitis
	Neoplasia	Papilloma
	Bleeding diathesis	Bleeding diathesis
	Amorphous debris	Polyps
Kidney	Calculi	Pyelonephritis
	Trauma	Trauma
	-Nephritis	-Infarction

	-Vascular Anomaly	-MCF
	-Parasitic migration	
	-Neoplasia	
	-Glomerulopathy	
	-Papillary necrosis	

- If the blood is equally distributed in the urine – This could be kidney damage.
- Hematuria at the beginning of urination – This could be urethral damage.
- Hematuria at the end of urination – This could be vesicle damage.
- Hematuria after exercise, especially, in horse – This could be cystic calculi.

Uniform discoloration of urine without clots, you got to make sure the discoloration is hematuria and not Hburia, bilirubinuria or myoglobinuria.

(d) Crystalluria:

It is the presence of crystals in the urine the presence of crystals in urine of herbivorous animals have no special significance unless they occur in large numbers and one associated with irritation of U.T. It may occur with no clinical signs or may indicate a severe problem in renal tissues or U.T. infection.

Calcium carbonate and triple phosphate crystals are more common as urine. If they are observed, the possibilities of urinary tract infection and / or a potential for obstruction should be considered.

If they congregate into large masses, they may obstruct either renal pelvis, ureters, urethra or the urethral opening while smaller formations may obstruct the individual nephrons.

(e) Pyuria:

It is the presence of purulent debris in the urine. Pyuria indicates an inflammatory exudation at any point of the urinary tract, usually renal pelvis and bladder. This purulent debris may appear in the form of grass clots or shreds or only detectable by microscopic examination.

Pyuria is usually accompanied by the presence of bacteria in the urine. Also, dysuria, stranguria and crystalluria are evident. A fever may or may not be present in such cases, but urine scalding of the perineum is usually present.

(f) Hemoglobinuria:

It is defined as presence of hemoglobin in the urine. False hemoglobinuria occurs with cases of hematuria when the R.B.Cs are destroyed and liberate their contents of hemoglobin into urine. Meanwhile, true hemoglobinuria is manifested by deep red discoloration of the urine caused by lysis of R.B.Cs due to many disease such as:

- Bacillary hemoglobinuria.
- Babesiosis.
- Copper intoxication.
- Water intoxication.

(g) Myoglobinuria:

It is the presence of myoglobin in the urine. Myoglobinuria is a good evidence of severe muscular destruction such as in azoturia in horses. It may be observed in enzootic muscular dystrophy but the amount of myoglobin in such young animals is insufficient to cause the problem.

Because the molecule of myoglobin is much smaller than that of hemoglobin, it is usually excreted in the urine in the diseased cases without staining of blood serum.

(h) Glucosuria and Ketonuria:

Glucosuria is not common in large animals but occurs usually in pet animals such as in diabetes mellitus. Glucosuria in large animals is usually associated with the following disease conditions:

- Enterotoxaemia is caused by clostridium peferinges type D.
- Rabred cattle.
- Administration of dextrose solution, treatment by using adrenocorticotrophic hormones, cortisone.
- Cases of nephrosis.
- Meanwhile, ketonuria is more common in cattle and sheep as in cases of starvation, pregnancy toxemia and acetonemia in cattle.

(i) Indicanuria:

It is the presence of Indican substance in the urine (potassium indoxyl sulphonate). Presence of excessive amount of this substance in the urine indicates an increased absorption of the detoxified end – products from gastrointestinal tract. Such condition is usually associated the cases of constipation and obstruction of the intestine.

(j) Creatinuria:

Excessive breakdown of muscular tissues leads to increase amount of creatinine in the urine and considered a good indication for muscular dystrophy.

(2) Abnormalities of the daily urine flow Polyuria:

It is the passage of abnormally large amounts of urine.

This may be normal response when excessive fluid and / or electrolytes are presented to tubules of healthy kidney.

Evaluation of an animal with polyuria should begin with inquiry about any history of drug or fluid administration or laboratory evidence of renal disease.

Osmolality or specific gravity:

If the osmolality is close to isoenuric range (similar to plasma concentration regardless of patient's fluid volume status) – primary renal disease should be considered.

Determination of GFR would be indicated:

- If the basic measurements of GFR (e.g. serum creatinine, and 12-or 14hr creatinine clearance) are not abnormal – water deprivation test may be needed to determine the ability of tubules to concentrate urine.
- An alternative to this test is to measure the fractional excretion of sodium in the urine.
- Sodium excretion is greater the 1 % is highly suggestive of primary tubular disease.

This measurement is performed on collected urine and serum samples by measuring the creatinine and sodium in both the serum and the urine. Fractional excretion of sodium is than determined by the formula:

- If the specific gravity or osmolality of the urine is less than that of the plasma, - diabetes insipidue should be considered.
- It may be the result of secretion of vasopressin or ↓ response of kidney tubules and receptors to ↓ vasopresin.

Causes of polyuria in Horses and Ruminants:

- Acute and chronic renal failure.
- Hyperglycemia – Diabetes mellitus.
- Psychogenic water intoxication.
- Fluid steroid administration.
- Diuretic.
- Salt deficiency or toxicity.
- Diabetes insipidus.
- Severe deficiency of Cl-K- or urea.

(b) Oliguria and Anuria:

Oliguria means reduction in the daily urine output.

Oliguria usually results from:

- Terminal stages of nephritis.
- Congestive heart failure.

- Peripheral circulatory failure.
- Renal ischemia.
- Dehydration and hemorrhage.
- Partial obstruction or stenosis of the urinary passages.
- Hot weather and excessive salivation, and excessive sweating.

Meanwhile, anuria means complete absence of urine. Such case is usually observed in many disease problems such as complete bilateral obstruction of urinary passages, acute form of tubular necrosis.

3) Dysuria and stranguria:

- Dysuria is difficult or painful urination.
- Stranguria is slow and painful urination.

It is difficult to differentiate between dysuria and stranguria in large animals and they are usually caused by:

- Urethral obstruction.
- Inflammation of the urethra, bladder or both.
- Neurologic conditions that prevent normal emptying of urine, from the bladder.

The horse voids urine very actively and forcefully. Both male and female adult horses normally may groan and strain on urination. In other species of animals urination is passive, and straining or groaning associated with urination normally could not be observed.

Clinical signs in horses:

- Straining, urination of shorter duration than normal or frequent attempts at urination that produce only small amounts of urine .
- The urine may be discolored e.g. blood urine. (Normal horse urine is very cloudy because it contains large quantities of calcium carbonate and mucons).

Clinical signs in ruminants:

- The clinical signs are similar to those in horses, except that urine dribbling is more common in male goat and sheep with calculi.
- Vocalization is more common in male goats with calculi.
- Urine scalding of the perineal region or rear legs may be noted in either ruminants or horses.
- Intermittent prolapse of the prepuce is marked in .
- Dysuria or stranguria in large animals may be confused with tenesmus.

4) Urachal leakage of urine:

The clinical signs of incomplete urachal closure in newborn animals are visible dripping of urine from the ventral abdomen and / or swelling of umbilical area. Improper closure may be the causes by inflammatory diseases or incomplete closure.

Diagnosis is based on:

1. History.
2. Clinical findings (pain, fever, exudate...).
3. Percutaneous palpation and / or ultrasonography.

Treatment:

Surgical removal of infected urachus and / or umbilical vessels.

5) Uremia:

This term is used to describe the clinical syndrome, which occurs in the terminal stage of renal insufficiency. It may be also applied to the symptoms mainly gastrointestinal and nervous which could result from various types of advanced renal diseases.

Renal failure is manifested by the clinical state of uremia, which is characterized by an increase in the blood levels of total protein and urea nitrogen and by retention of other solutes, and urinary constituents in the blood.

Uremia may be the result of a acute or chronic renal failure. It seems most reasonable that several toxins are associated with the uremic process, including azotaemia. However, the etiology of uremia is the same of acute and chronic renal failure.

The predominant clinical signs of uremia seen in large animals are depression, anorexia, seizures and encephalopathy, which may occur, in severe episodes. Weight loss, oral erosions, gastro intestinal ulcers, melena and accumulation of excessive dental tarter are also observed in the diseased cases.

Abnormalities, which interact to produce signs of uremia:

(a) Auto intoxication:

a. 1. Electrolytes (excess or deficiency):

Increased sodium → overhydration.

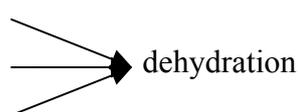
Reduced sodium → dehydration.

Increased potassium → myocardial asthenia and fatal heart failure.

Reduced potassium → muscular weakness .

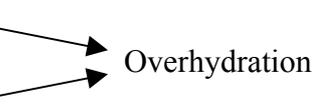
a.2.Fluid:

Polyuria
Vomiting
Diarrhea



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graph LR; A1[Polyuria] --> B[dehydration]; A2[Vomiting] --> B; A3[Diarrhea] --> B;
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Oliguria
Anuria



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graph LR; A4[Oliguria] --> B[Overhydration]; A5[Anuria] --> B;
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a.3.Acid-base imbalance:

Renal metabolic acidosis is evidenced.

a.4. Hormones:

Reduced production of erythropoietin hormone usually results in non-regenerative anemia.

a.5. Accumulation of waste products of metabolism.

(b) Body compensation:

- Polyuria – results in compensatory polydipsia.

- Hypocalcaemia – results in compensatory release of parathormone.
- Metabolic acidosis – results in increased rate and depth of respiration (hyperpnoea) aiming to eliminate the excess CO₂.

Assessment of the urinary system and diagnosis of the urinary tract (UT) diseases:

(1) History:

It is very important to make a few remarks on the history, before or prior to discussing physical examination findings, due to its importance in diagnosing the UT diseases. In selected cases, the history may be more important than the clinical examination findings in diagnosis of a UT disease, e.g. a castrated male goat with pain and stranguria should be assumed to have urinary calculi unless other cause can be proven.

As a general rule, diseases of the lower tract are often very “noisy” (easily recognized by the owners), whereas diseases of the upper tract are of then more subtle; an exception to this is subacute contagious bovine (CB) pyelonephritis in cattle.

(11) Assessment of gross and microscopic structure:

- Rectal palpation: Refer to the practical note vet. Clinic diagnosis.
- Percutaneous biopsy of the kidney.

The causes of renal failure usually can be determined by history, urine analysis, renal function tests, and physical examination for obstructive causes, negating the need for a renal biopsy.

- Radiography:
Enlargement of the renal pelvis found in pyelonephritis or hydronephrosis, urethral enlargement or aberrant locations, or defects in continuity of the excretory urinary tract can be determined using I/V injection of 1 mL / kg. B.wt. of Renografin.
- Urinary tract endoscopy:
Direct visualization of most of the lower urinary tract is possible with rigid and flexible endoscopes. Cystoscopic examination can be valuable in determining suspected abnormalities such as site of hematuria, pyuria and ureteral obstruction.
- Ultrasonography:
Ultrasonography is useful for assessing location of individual structures of the urinary system and lesions within those structures.

(111) Catheterization (Refer to practical note of clinical examination) .

(1V) Urinalysis:

Urinalysis should be completed within 15-30 minutes of collection. If this is not possible it should be refrigerated, as prolonged exposure to room temp before analysis can result in growth of bacterial cells, dissolution of delicate casts, cellular degeneration resulting in loss of cellular details, and change in pH. Urinalysis

includes examinations of all the physical, biochemical and microbiological of findings of the urine.

- Urine specific gravity.
- Color smell.
- Viscosity.
- pH of urine in herbivorous animals is alkaline 7.9 to 0.05 .
- Biochemical findings. ,
- Microbiological of examination.

(V) Renal function tests:

◀ The simplest and most important tests of urinary functions are the determination of whether or not the urine is being voided.

◀ Renal function tests evaluate the functional capability of the renal tissues (kidneys). In general these tests assist to know:-

- Blood flow to the kidneys.
- Glomerular filtration.
- Tubular function.

We can classify these tests into three groups depending on whether they are based on the exam of blood or urine, or both.

(1) Tests conducted on urine:

Specific gravity: is the simplest test to measure the capacity of renal tubules to conserve fluids and excrete solutes. In most species of animals, specific gravity ranges from 1.028 to 1.095.

In chronic renal diseases, sp. gr. falls down to up to 1.010.

Osmololity of fluid is an alternative to sp. gr. and it gives a more accurate assessment of tubule's capacity to conserve fluid and excrete solutes.

(2) Tests conducted on blood:

These tests depend on either:

- ◀ The accumulation of metabolites normally excreted by the kidneys (in cases of insufficiency).
- ◀ Excretion of endogenous substances by the kidney.
- ◀ Creatinine and BUN are essential components of evaluation of renal function. Creatinine and BUN do not rise significantly above the normal range until 60-75% of nephrons are destroyed.
- ◀ Glomerular filtrations rate has been estimated by: I /V injection of sodium sulphanyl, then to follow the disappearance of this substance.
- ◀ Renal Blood flow may be evaluated by measuring the half-time of disappearance of dye such as phenolsulphthalein (PSP) as given by I /V injection .

(3) Tests conducted on urine and blood:

- ◀ Water deprivation test. It can be used to assess the renal concentrating ability in horse.
- ◀ Fractional excretion of sodium.

General principles of treatment of urinary tract diseases

(Basic Idea of Treatment)

- ◀ The initial treatment of acute renal failure (ARF) in all animals aimed to remove the predisposing factors, removing the primary cause, and restoring normal fluid balance by correction of dehydration, electrolytes and acid- base abnormalities.
- ◀ Serum concentration of sodium, chloride, potassium and bicarbonate should be monitored daily and replaced or corrected as needed.
- ◀ After correction of any volume and electrolyte deficits, an attempt should be determine disorder of ARF as oliguric or polyuric, by using the following one of the followings:
 - 1-Estimation total plasma proteins.
 - 2-Estimation central venous pressure, and / or by measuring fluid input compared to its output .

In oliguric animal:

- ◀ I/V injection of Mannitol 20% solution at a dose rate of 0.25-2 gm /kg B.wt.
- ◀ Furosemide (Lasix) at a dose rate of 1-2 mg / kg B.Wt. every 2 hours until diuresis.
- ◀ Dopamine at a dose rate of 1-5 Mg /Kg /min in 5% dextrose solution I/V with Furosemide may be useful to improve urine output.

In Non- oliguric animal:

- ◀ I/V injection of 0.9 NaCl or balanced electrolyte solution at a dose rate of 40-80 ml / kg / day until there is a precipitous drop in serum creatinine.
- ◀ Continuation with 10-20 ml / Kg / day.
- ◀ Sedation could be obtained by using xylazin at a dose rate of 0.4-0.8 mg / Kg. by I .V injection.

* The most frequently used antimicrobial drugs that are recommended for UT infection are:

- Trimethoprim / sulfadiazine.
- Nitrofurantain.
- Ampicillin, Amoxicillin,
- Penicillin – Gentamycin.
- Cephalosporins.

Antimicrobial therapy for infection of lower tract should be continued for 7-10 days, and it should be continued for 2-4 week for upper tract infection. Bacterial culture should be made 7-10 day post treatment, and second antimicrobial dose may be needed.

Use of Antiemetic (in dogs) in cases of uremia, such as:

- Cimetidine (Tagamet):
10 mg /Kg. B.wt I / V injection on 2nd .day.
you can give it orally to the animal when vomiting stops .
- Ranitidine.
2-4 mg /kg. B.wt twice / day.

Gastrointestinal coating agents:

Kaolin, Pectin 3 times / day orally.

Restriction of food for at least 24 hrs during therapy

Diseases of the kidney

1- Renal ischemia:

It means reduction or decline of the blood flow through the kidneys, which is usually the result of circulatory failure. There is transient oliguria followed by anuria and uremia if the failure is not corrected.

Pathogenesis:

- Sudden reduction in cardiac output due to any cause.
- Lowering of blood pressure.
- Accumulation of the metabolites that are normally excreted such as BUN and creatinine, giving rise to prerenal Azotemia.
- Reduction of the glomerular filtration, which increases the tubular resorption and reduced urine flow (oliguria).
- Up to a certain level, the degenerative changes are reversible by restoration of renal blood flow; but if the ischemia is severe enough and of sufficient duration, the renal damage is permanent.
- The parenchymatous lesions vary from tubular necrosis to diffuse cortical in which both tubules and glomeruli are affected.
- Plugging of the tubules with casts of coagulated protein may exacerbate uremia acute hemolytic anemia and acute muscular dystrophy with myoglobinuria, but ischemia is also an important factor.

Clinical findings:

- Renal ischemia does not appear as a distinct disease and the clinical signs of the primary disease mask its signs.
- The general clinical picture is one of acute renal failure.

Clinical pathology:

1. Urinalysis.
2. Monitoring BUN, creatinine level in serum.

Passage of large volume of low specific gravity urine after a period of oliguria is usually a good indication of a return of normal glomerular and tubular function.

On urinalysis, proteinuria is an early indication of damage to the renal parenchyma.

Treatment:

- Correction of fluid, electrolytes and acid – base balance.
- Supportive treatment as suggested for ARF (if there is renal damage).

(2) Pyelonephritis:

Definition:

It is bacterial inflammation of kidney and renal pelvis. Pyelonephritis develops most commonly from ascending infection through the lower urinary tract. It is

characterized clinically by pyuria, dysuria or stranguria, suppurative nephritis, cystitis and ureteritis.

Etiology:

- In most cases it results following bacterial infection of the lower urinary tract.
- Spread from embolic nephritis of hematological origin (descending infection) such as septicemia of cattle caused by *Pseudomonas aeruginosa*.
- Specific pyelonephritis caused by *Corynebacterium renale* in cattle and *Eubacterium suis* in pigs is also recorded.

Pathogenesis:

The development of pyelonephritis is depending up on:

- Common presence of infection in U.T.
- Stagnation of urine.
- Reflux of urine from the bladder.

Urine stasis can occur as a result of:

Blocking of the ureters by inflammatory swelling or debris, by pressure from the uterus in pregnant females, and by obstructive urolithiasis.

Infection ascends the ureters → invades renal pelvis causing pyelitis → and involves the papillae causing diptheroid destruction → then it extends to involve the medulla and cortex.

Signs of toxemia and fever, usually accompany extensive bilateral infection, uremia may develop if the lesions are sufficiently extensive. Pyelonephritis is always accompanied by hematuria because of the inflammatory lesions of the ureters and bladder.

Clinical findings:

- The first sign observed may be the passage of blood stained urine in some cases.
- In other cases, the first sign may be an attack of colic, manifested by swishing of the tail, kicking at the abdomen and treading of the feet. Straining to urinate; such attacks are caused by obstruction of the ureter or renal calyx by pus or tissue debris.
- The onset is gradual with fluctuating temperature (39.5° C).
- Capricious appetite, loss of condition and fall in milk yield over a period of weeks.
- The most obvious sign is the presence of pus, blood, mucous and tissue debris in the urine, particularly at last portion voided.
- Urination is frequent, painful and may occur in a dribble rather than a stream.
- Rectal examination may be negative in the early stages, but later there is usually detectable thickening and contraction of bladder wall and enlargement of one or both ureters.
- Left kidney may show enlargement, absence of lobulation and pain on palpation.
- In many cases (chronic) there are no distinct clinical signs referable to the U.S. and may have, history of chronic weight loss and in such cases urinalysis is important in diagnosis.

Clinical pathology:

- Urine analysis for blood and protein.
- Urine pH is greater than 8.5 (Alkaline).
- Urine specific gravity is 1.008-1.212.
- Microscopic exam – pyuria, debris, blood cells.
- Bacteriological culture
- Hypoalbuminemia and Hypergammaglobulinemia.
- Elevations of BUN 1.5 mg / dL and cr. 100 mg / dL. Which carry a grave prognosis.

Treatment:

- General principles for treatment of U.T. infections.
- Acidification of urine by the administration of monobasic sodium phosphate (100 gm daily for several days).
- Antibiotic therapy; penicillin remains the antibiotic of choice in this cases. 15.000 IU / Kg B.Wt of procaine penicillin for at least 3 weeks.
- Unilateral Nephrectomy may be carried out in valuable animals.
- Improving appetite, milk yield and clearing of urine expect good prognosis.

(3) Glomerulonephritis:

This form of nephritis involves primarily the glomerulus then may extend to involve the interstitial tissues (secondarily) and rarely the blood vessels. Glomerulonephritis is an immunologic disease or disorder that results in deposition of Ag-Ab complexes in the GBM or anti-GBM antibody to the capillary walls, with consequent impairment of GF, ↓ GF and enhanced permeability to plasma proteins.

Glomerulonephritis is an uncommon clinical renal disease in sheep and occasionally as a clinical entity in cattle, but the cattle are usually not examined until the disease process is advanced.

The three types are seen include:

- Spontaneous proliferative glomerulonephritis.
- Glomerulonephritis of pregnancy toxemia in sheep.
- Mesangiocapillary glomerulonephritis.

Clinical and lab. Findings:

- Weight loss, chronic diarrhea and generalized edema.
- Marked oliguria or even anuria in some cases.
- Proteinuria, hypoalbuminemia and anemia.
- Serum Cr. And BUN are elevated in advanced cases – develop signs of azotemia.

Treatment:

- Is usually unrewarding.
- Plasma transfusion.
- Anabolic steroids.

(4) Embolic nephritis:

Embolic lesions in the kidney cause no clinical signs unless they are extensive in which case toxemia followed by terminal uremia. Transitory periods during which

proteinuria and pyuria occur may be observed if urine samples are examined at frequent intervals.

Causes:

Embolic suppurative nephritis or renal abscess may occur after any septicemia and bacteremia when bacteria lodge in renal tissues the origin of emboli may be: -

(A) Sporadic cases:

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

(B) Associated with systemic infections such as: -

1. Shigellosis in foals.
 2. Erysipelas in pigs.
- Septicemic or bacteremic strangles in horses.

Pathogenesis: -

- Localization of single bacterial cell or bacteria in small clumps in renal tissues development of embolic suppurative lesions.
- Emboli block larger vessels than capillaries infarction, local ischemia in the affected portion, and if this infarction is small there is no clinical signs.
- Clinical signs only develop when emboli are multiple and destroy much of renal parenchyma.
- If the urine checked repeatedly, the sudden appearance of proteinuria, casts, and microscopic hematuria without other signs of renal disease it suggests infarction.

Clinical signs:

- Embolic nephritis may pass without clinical signs due to insufficient renal damage.
- Enlargement of the kidney may be palpated by rectal examination.
- Fatal uremia and toxemia may be occurring.
- Large infarcts may cause bouts of transient abdominal pain.

Diagnosis :

- Clinical signs.
- Clinical pathology as that of pyelonephritis accompanied with cystitis or urethritis.
- P.M. lesions:
 - Enlargement of kidney with abscess formation.
 - Much fibrous tissue surrounding the lesions.

Treatment:

- Sensitivity tests to chose proper antibiotics.
- Antibiotics for about 7-10 days and may be injected with specific enzymes.

(5) Interstitial Nephritis:

It is a common disease of the dog, it may be acute “diffuse” or chronic “focal” but is always non-suppurative. Focal interstitial nephritis white – spotted kidney. Acute type is usually caused by leptospirosis in dogs. Chronic poisoning with caustic-treated roughages in cows leads to chronic form of the disease.

Clinically is characterized by:

- Polyuria with low specific gravity urine.
- Vomiting – polydipsia and depression.
- Arched back in severe cases and temperature may be elevated.

Treatment:

- Restoration of water and electrolyte balance is important.
- Parenteral administration of antibiotics.

(6) Nephrosis:

Nephrosis includes degenerative and inflammatory lesions of the renal tubules uremia may develop acutely or as terminal stages after a chronic illness manifested by polyuria, dehydration and loss of weight.

Etiology:

Most cases of nephrosis caused by toxins.

Toxins:

- Mercuric compounds, selenium and organic copper compounds.
- Oxalates in plants and fungi.
- Thiabendazole anthelmintics.
- Over dose w sulphonamides.

Haemodynamic factors:

- (1) Dehydration leading to concentration of toxins in the tubules.
- (2) Severe renal ischemia.
- (3) Hemoglobinuria causing hemoglobinuria nephrosis.

Pathogenesis:

- In acute nephrosis there is obstruction to glomerular filtrate flow through the tubules obstructive oliguria and uremia.
- In chronic cases there may be impairment of tubular reabsorption of solutes and fluids.

Clinical signs:

In acute stage there is oliguria, proteinuria and other symptoms of uremia as :

- Anorexia, hypothermia, depression, weak pulse.
- In cow there is continuous mild hypocalcemia.
- Polyuria in chronic cases.

Diagnosis:

- Clinical signs.

- Clinical pathology.
- Necropsy findings.

Treatment:

- Correct the primary cause.
- Water and electrolytes therapy.
- Antibacterial agents.

(7) Hydronephrosis:

Cystic enlargement of the kidney due to obstruction of the ureter, caused usually by urolithiasis and congenital anomalies in ureters it is seldom detected clinically in farm animals.

(8) Renal Amyloidosis:

It is usually accompanied general amyloids in the body it occurs as a result of long standing metabolic disorders of proteins and clinically manifested by profuse-water diarrhea, signs of dehydration, hypoproteinuria.

Causes:

1. Suppurative lesions due to strept infection.
2. Pyogenic membranes, Abscess formation in liver-lung.

Pathogenesis:

Amyloid substances deposits in the glomeruli interfere the process of glomerular filtration and permeability. This disturbance in permeability leads to passage of protein causing proteinuria. Absorption of toxic amyloids causes nephritic changes and edema in interstitial wall and renal insufficiency.

Clinical signs:

- Gradual loss of body weight.
- Profuse watery diarrhea.
- Urine with low specific gravity.
- Depressed appetite, signs of dehydration.
- Edema in intermaxillary space.

Diagnosis:

- Clinical signs.
- Clinical pathology.

Differential diagnosis from:

1. Chronic enteritis.
2. Paratuberculosis.
3. Pyelonephritis.
4. Leukosis.

Diseases of the urinary bladder

1) Cystitis:

Inflammation of the bladder is usually caused by bacterial infection and is characterized clinically by frequent, painful urination and the presence of blood, inflammatory cells and bacteria in the urine.

Etiology:

Cystitis occurs sporadically due to the introduction of microorganism into the bladder, which is usually associated with trauma to the bladder, paralysis of the bladder and stagnation of urine due to any cause.

The most common predisposing factors or the common association are:

1. Cystic calculus.
2. Dystocia or difficult parturition.
3. Late pregnancy.
4. Contaminated catheterization.

Cystitis can also occur because of ascending infection and accompanying pyelonephritis caused by *corynebacterium renal* in cattle, *Eubacterium suis* in pigs *corynebacterium cystitidis*, *Escherichia coli*, *Streptococcus* and *Pseudomonas* .

Pathogenesis:

- Bacteria frequently gain entrance to the bladder, but are usually removed by the flushing action of voided urine before they invade the mucosa.
- Stagnation of the urine with mucosal injury, facilitate invasion of the mucosa.
- Irritation of stretch receptors in the bladder wall, as a result of this irritation the animal is constantly stimulated to urinate and the U.B. remains empty.

Hence, signs relate to changes in urination behavior (dysuria, pollyuria, stranguria and apparent incontinence).

Clinical findings:

- Frequent and painful urination, accompanied by grunting.
- Animal adopted the urination posture some minutes after the act of urination has been completed.
- The volume of passed urine each time is very small.
- Moderate abdominal pain as manifested by treading with hind feet, kicking at the belly, swishing with the tail.
- A moderate febrile reaction.
- Acute retention may develop if the urethra has been blocked by blood, or pus.
- Painful or palpation.

In chronic cases, the signs are less marked but frequent urination and small volume of urine are the signs. The bladder wall may feel thickened in rectal exam.

Diagnosis :

1. It is mainly rests on observation of clinical signs.
2. Clinical pathology findings:
 - Cellular findings in the urine, such as blood and pus.
 - Urine may have a strong ammonia smell.
 - Urine may be turbid having much mucous.

- Bacterial culture from the urine commonly shows microorganisms.
3. Necropsy findings:
- Hyperemia, hemorrhage and edema of the mucosa.
 - Thickening of the wall in chronic cases.
 - Erosion of the papillary projections.
4. Differential Diagnosis:
- (A) Pyelonephritis:
- It is difficult to be differentiated, but the prognosis in case of cystitis is much better.
 - Thickening of the bladder wall.
- (B) Cystic urolithiasis:
- It is usually detected by rectal palpation by ultrasonographic and radiographic examinations.
 - Urine flow is greatly restricted.
 - Distended bladder can be felt on rectal palpation.

Treatment:

- The earlier treatment, is commended, the better the prognosis, and return to normal production.
- Antibiotic treatment for at least 10-14 days is highly recommended.
- Water and salt should be always available.
- Use of urine acidifiers such as ammonium chloride at a dose rate of 50-100 mg / kg twice / day.
- Urinary antibiotic.

2) Paralysis of the bladder or Vesical paralysis:

Vesical paralysis is uncommon in large animals caused mainly by the lesions of lumbosacral part of the spinal cord. In the early stages of vesical paralysis due to neurogenic injury, the bladder remains full with urine and dribbling of urine occurs especially during movement; good flow of urine can be obtained by compression of the bladder through rectal examination.

However, regular catheterization is essential in conjunction with a prophylactic dose of antibiotic to prevent the development of cystitis. Nerve tonics, strychnine, belladonna and *nux vomica* are useful in such cases.

Obstruction of the urinary tract:

Calculi, blood, neoplasia, smegma, edema and / or cellulites may result in obstruction of the urethra.

- Calculi are most often CaCO₃ stones that lodge in the pelvic urethra in the stallions and geldings.
- Blood clots resulting in obstruction of the urethra are rarely seen, but may be found in association of renal trauma.
- The most common neoplasia causing obstruction of the urethra is a squamous cell carcinoma of the penis.
- Many inflammatory and / or edematous diseases of the penis and urethra may cause urinary obstruction.

3) Urolithiasis:

Urinary calculi or uroliths include: Nephroliths, ureteral, cystic and urethral calculi.

Nephroliths and ureteral calculi:

- Ureteral calculi and renal stones may result in obstruction of one or both ureters and renal pelvis.
- The obstruction may be complete or incomplete and may be chronic or intermittent.
- Most commonly both kidneys and / or ureters are affected.
- It is more common in young adult thoroughbred horses.
- The stones are usually composed of CaCO₃ or Ca. phosphate.
- Because of the bilateral involvement and insidious nature of the disease, most cases have progressed to chronic renal failure (CRF) before they are noticed. Weight loss and / or poor performance are the most common presenting complaint.

Cystic Calculi:

- Cystic calculi occur sporadically in horses. They are usually singular and either large and smooth or smaller and rough.
- The most common clinical signs are hematuria after exercise, stranguria and dysuria . Urinary incontinence usually occurs in most cases.

Urolithiasis :

- Urinary calculi or urolithiasis occur sporadically in ruminants. It becomes of clinical importance when obstruction of the U.T. occurs.
- Obstruction urolithiasis is almost exclusively a disease of males and primarily affects steers, rams, bucks & wethers, especially castrated animals.
- Three common clinical entities occur in urolithiasis syndrome which are:
 - ◀ Urethral obstruction.
 - ◀ Rupture of the urethra.
 - ◀ Rupture of the bladder.

As a result of these entities, obstructive urolithiasis are characterized clinically by:

- ◀ Complete retention of urine.
- ◀ Unsuccessful efforts to urinate .
- ◀ Distention of the U.B.
- ◀ And eventually ruptured of the urethra or bladder .

Causes and Epidemiology :-

Urolithiasis is a disease of all animal species, but of greater importance in fattening steers and rams which fed high concentrate diet . Also, it occurs among animals grazing an pasture that contains high levels of oxalate, and silica , The incidence of urolithiasis varies greatly with the season, and of high value at winter season .

There are groups of factors that contribute to urolithiasis :-

- ◀ Those factors which favor the development of a nidus about which precipitation and concretion can occur .
- ◀ Those factors which facilitate precipitation of solutes on to the nidus and .

- ◀ Those which favor concretion and cementing precipitated salts to the developing calculus .

(1) Nidus Formation :-

A Nidus is usually a group of desquamated epithelial cells or necrotic tissues that favor the deposition of crystals and other salutes around it .

Nidus or calculus may result from:

- Local infection of the U.T.
- Herd problem due to vitamin A deficiency.
- Fattening problem e.g. Administration of massive doses of anabolic or growth promoting such as estrogen preparation, (estradiol, stilbesterol) which lead to massive desquamation of epithelium.

(2) Precipitation of solutes: -

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

Normally, urine is highly saturated solution containing large quantities of solutes. These salts are not precipitated, normally, because of the colloidal state mechanism in the urine. This protective colloid prevents precipitation of salutes and converts the urine into a gel.

This mechanism is so efficient up to certain point and its capacity to maintain normal solution could be overcome by a number of factors. The degree of super saturation of urine depends on the diet, PH of urine, a mount of water intake and the presence of crystalloids inhibitors in the urine.

A- Diet: Plays an important role in solute ppt in different ways :

- Highly concentration diet rich in phosphate and oxalate increases the incidence of metabolic defects that consequently increases the incidence of urine solutes.
- Hypervitaminosis – D for example increases ca level in urine.
- Cattle raised an grass pastures that contain high levels of silica (6%) have high conc. of silicon dioxide in the reticulorumen fluid, which is converted into salicylic acid, absorbed and then excreted in the urine until its values increase above the supersaturation of urine, especially with reduced water intake.

Salicylic acid may be polymerized to form large micelles, which may then coalesce and precipitate in the urine protein to form a nidus, which continues to grow in the presence of supersaturated urine.

B- pH of the urine:

PH of the urine has an effect on the solubility of some salts. Although formation of siliceous calculi unaffected by changes in the urine PH, the acidification of urine favors the dissolution of stones and reduces the incidence of such calculi.

Mixed phosphate and carbonate calculi usually formed in alkaline urine than in acid urine. So addition of ammonium chloride or phosphoric acid decreases the incidence of calculi in fattening animals. This mainly due to the effect of pH on urinary calcium citrate, which acts as buffer in urine maintaining calcium in a soluble state by formation of soluble calcium citrate complex which is not dissociated.

Deposition of citrate contents favors the precipitate of solutes. Concentration of urine favors the precipitate of solutes.

C- Water intake:

Obstructive urolithiasis occur an most frequently in winter season, and reduced water intake is thought to result in increase concentration of crystalloids in the urine .

(3) Factors favoring concretion and cementing: -

Mucoprotein, especially mucopolysaccharide fraction may act as cementing agent favor the formation and development of urinary calculi. This may be the fact in the fattening steers that feed on:

- ▶ High concentration diet with low roughage.
- ▶ High phosphorus contents in the diet.
- ▶ High level of mucoprotein in urine of fattening steers may result as rapid turnover of supporting tissues in those animals making rapid gain in body weight
- ▶ High concentration diet – rapid gain in body weight .
- ▶ Rapid turnover of supporting tissues.
- ▶ High level of mucoprotein in the urine.

Chemical composition of calculi:

It varies and depends mainly on the dietary intake of individual elements. Calcium, ammonium, and magnesium carbonate are the common calculi in cattle and sheep. Calcium carbonate and phosphate are more common in the Horse. Silicon calculi are common in grazing cattle and sheep.

Calculi may be found as one stone, or more than one or as amorphous sediment. They may be large and smooth or small and rough.

Clinical findings:

- Calculi in the renal pelvis and ureters are not usually diagnosed clinically; but rectal examination may help in diagnosis of urethral calculi especially if they accompanied by hydronephrosis .
- The animal is in state of restlessness, switches its tail, may kick at the abdomen and strains in attempts to urinate.
- Frequent, painful urination with straining and grunting (in case of partial obstruction).
- Dribbing of Blood-Stained urine if complete anuria is not present.
- Pulsation of the urethra at the ischial arch, which could be palpated rectally.
- Swelling and hypersensitivity at the sigmoid flexure may be noted.
- Rectal examination reveals distended bladder, which usually ruptures within 48 hours in complete obstruction.
- In steers, calculi usually lodge at the proximal sigmoid flexure, while in rams, bucks and withers, calculi are usually at the urethral process.
- Pulse, respiration and temperature may be elevated in diseased sheep at goats.

- Finally, Azotemia develops.

Three common clinical entities occur in urolithiasis syndrome:

1. Urethral obstruction.
2. Rupture of the bladder.
3. Rupture of the urethra.

Urethral obstruction could be confused with many disease conditions and must be differentiated from:

- Bowel obstruction.
- Tenesmus with enteritis, coccidiosis.
- Acute indigestion.

Rupture of the urethra:

In the early stages, ruminant often go undetected resulting in necrosis with eventually perforation of the urethra at the site of obstruction.

The most prominent clinical findings include:

- ◀ Urine leaks through the defect and accumulates in the subcutaneous tissues along the sheath and ventral abdominal wall resulting in ventral swelling or what is called water belly.
- ◀ Depressed appetite.
- ◀ Rectal examination reveals either collapsed or partially distended bladder.
- ◀ Rupture of the urethra must be differentiated from subcutaneous abscessation, umbilical abscessation, umbilical hernia, hematoma of penis and infected of the prepuce.

(4) Rupture of the Bladder (Cystorrhaxis) :

This is the most serious sequel to urethral obstruction. When the bladder ruptures, an initial relief occurs and clinical signs of that case may not appear for 1-2 days later. By the next few days, uremia becomes severe, progressive to severe depression; anorexia and dehydration could be observed.

Ventral abdominal swelling may be detected by ballottement and urinary bladder may not be palpated rectally. Rupture of the bladder is usually on the dorsal aspect; it may consist of multiple holes or of single large tear.

Congenital defects of the urinary tract:

- ◀ Previous or patent urachus = Urachal leakage.
- ◀ Urethral Arteria: It is recorded in calves, and is manifested by failure to pass urine and distension of the patent portion of the urethra.
- ◀ Hypospadias: It means imperfect closure of the external male urethra in newborn animals mainly in lambs. It is usually accompanied with other neonatal defects including atresia ani, and diaphragmatic hernia. The cause is unidentified.
- ◀ Ectopic ureters: The ectopic ureter opens into the urogenital tract at a place other than the such as cervix, urethra or vagina. The condition may be unilateral with urinary incontinence from birth as major clinical sign.
- ◀ Renal hypoplasia and dysplasia: Bilateral hypoplasia is recorded in piglets and horses. Affected animal may die at birth, few days or months after that. In diseased animals, there were gradual onset of anorexia, weight loss and lethargy.

- ◀ Polycystic kidneys.
- ◀ Rupture of the bladder.