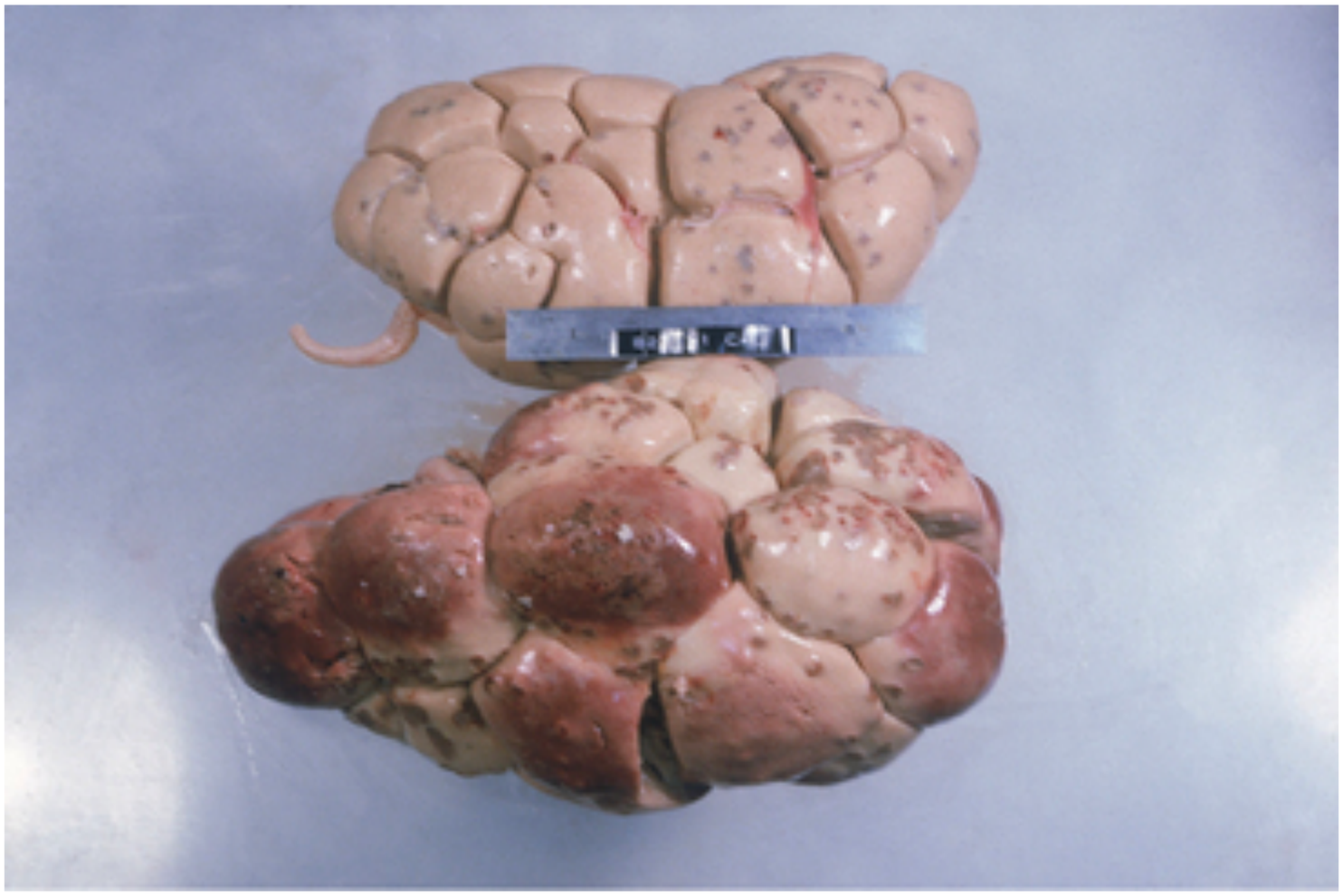
Diseases of the kidney



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1. Nephrosis

Nephrosis includes degenerative and inflammatory lesions primarily affecting the renal tubules. Nephrosis is the most, common cause of acute kidney failure and or uremia.

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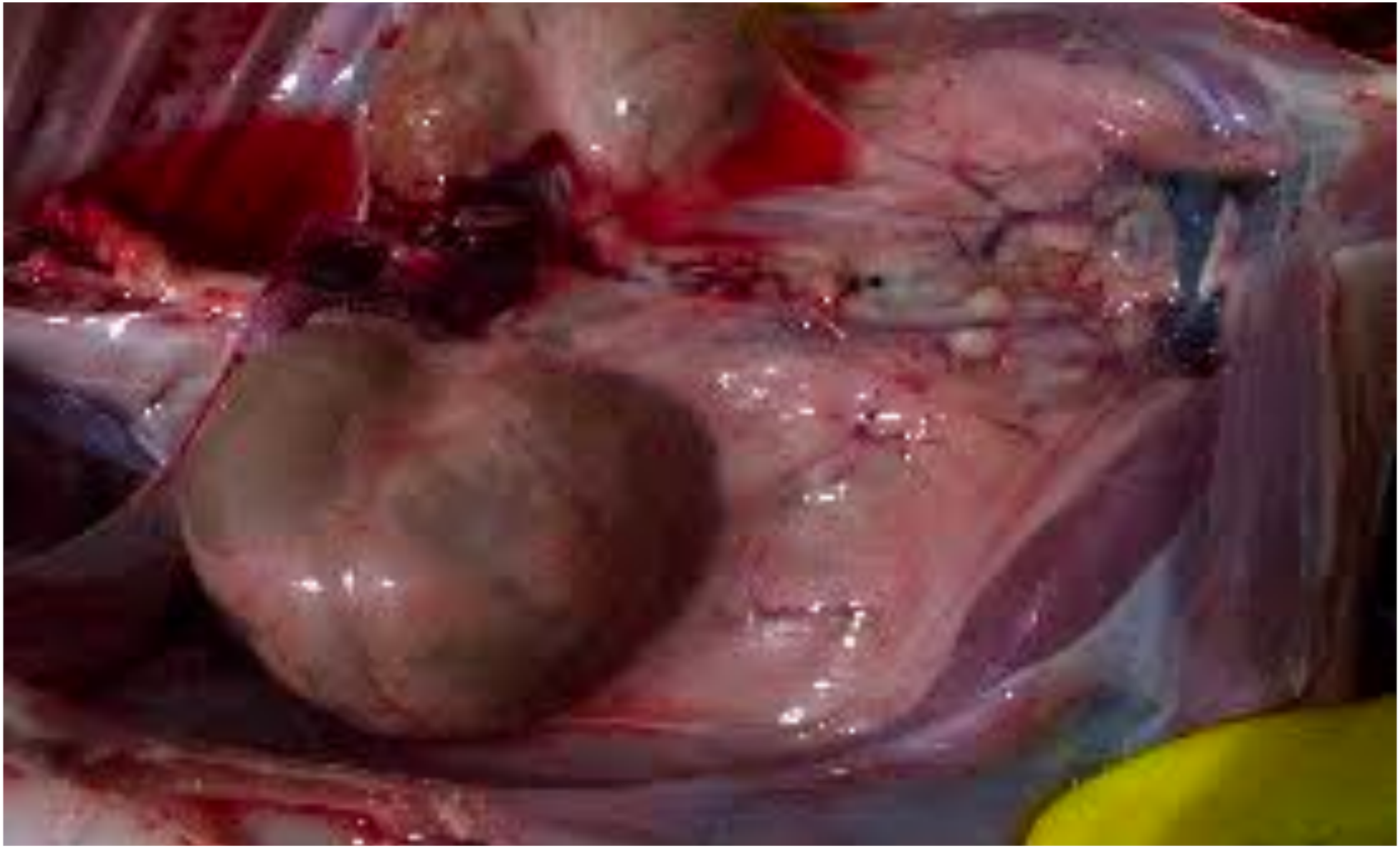
•Toxic nephrosis:

The kidneys are susceptible to endogenous and exogenous toxins from blood and during urine excretion.



Etiology

(1) Direct action of metals toxins (Mercury, arsenic, cadmium, selenium, and organic copper compounds).
(2) Oral administration of potassium dichromate and mercurid chloride includes topical blistering agents containing mercuric chloride.
(3) Antimicrobials like aminoglycosides, and overdosing with neomycin and gentamicin in treatment of calves.

(4) Prolonged treatment with or overdosing of sulfonamides, turpentine oil, Benzimidazole compound (Thiabendazole)
(5) Overdosing with vitamin K, D2 or D3 (injection) in Horses.
(6) Treatment of horses with non-steroidal anti-inflammatory drugs (Phenylbutazone and flunixin meglumine).
(7) Monensin in ruminants.
(8) Oxalate in plants
(9) Mycotoxins

Pathogenesis

(1) In acute nephrosis there is obstruction to the flow of glomerular filtrate through the tubules as a result of interstitial edema and intraluminal casts.

(2) If there is sufficient tubular damage, there may be back leakage of glomerular filtrate into the interstitial.

(3) There may also be a direct toxic effect on glomeruli which decreases glomerular filtration.

(4) The combined effect is oliguria and uremia.

(5) In subacute cases, impaired tubular resorption of solutes and fluids may lead to polyuria.

NB: Many systemic diseases such as septicemia cause temporary tubular nephrosis.

Clinical findings

(1) Clinical signs may not be referable to the urinary system.
(2) Per acute cases (In vitamin K3 injection) there may be colic and stranguria.
(3) In acute nephrosis there is depression, anorexia, hypothermia, a slow or an elevated heart rate, pulse, diarrhea, later on dehydration occurs.

Clinical pathology

(1) Presence of proteinuria, glucosuria and haematuria.
(2) Elevation of Gamma-glutamyl transferase in urine.
(3) Elevation of SUN and creatinine although hypoproteinemia may be present.
(4) In acute renal disease of horses, hypercalcemia and hypophosphatemia may be present but in vitamin D intoxication serum calcium and phosphate are increased.
(5) Azotemia occurs when uremia is present.

Necropsy finding:

(1) Kidneys are swollen and wet in acute cases.

(2) Necrosis and desquamation of tubular epithelia and hyaline casts in the dilated tubules.

(3) Renal medullary necrosis in phenylbutazone poisoning.

(4) Ulcers in all or any part of the alimentary tract.

- Treatment:
- (1) Specific antitoxins with treatment of the real cause.
- (2) Renal and hepatic wash using 10-25% IV glucose.
- (3) Purgative to get rid of GIT toxins.
- (4) Treatment the complication (Diarrhea, uremia).
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(2) Renal ischemia

Definition
It is a reduction of blood flow through the kidneys due to general circulatory failure causing transitory oliguria followed by anuria and uremia if the circulatory failure is not corrected.

Etiology
Any condition of hypotension or release of endogenous pressor agents.

Etiology of acute renal ischemia

(1) General circulatory emergencies such as shock, diarrhea, dehydration, acute hemorrhagic anemia, heart failure.
(2) Embolism of renal artery, recorded in horses.
(3) Extreme ruminal distension in cattle.

Human Pig Rodent

Etiology of chronic renal ischemia:
Chronic circulatory insufficiency e.g. CHF.

Pathogenesis

(1) As blood pressure falls, a sudden reduction in cardiac output occur lead to compensatory vasoconstriction of renal blood vessels and consequently acute ischemia, glomerular filtration decreases and metabolites that are normally excreted accumulate in the blood stream.
(2) The concentration of urea in the blood increases, giving rise to prerenal uremia.

Clinical findings

(1) Renal ischemia does not appear as a distinct disease and its signs are associated with the primary disease.
(2) Oliguria and azotemia.
(3) Later on, acute renal failure and uremia may occur.

Clinical pathology

Proteinuria as well as elevation of SUN and creatinine.

Necropsy findings

(1) Renal cortex is pale and swollen.
(2) Necrosis is visible at the corticomedullary junction.
(3) Necrosis of tubular epithelium, (glomeruli in severe cases).
(4) In hemoglobinuria and myoglobinuria hyaline casts are present in the tubules.

Diagnosis

Symptoms including oliguria and azotemia in the presence of circulatory failure.

Treatment

(1) Correcting fluid, electrolyte and acid-base disturbance.
(2) Supportive treatment with treatment of the real cause.
(3) Treatment of acute renal failure.

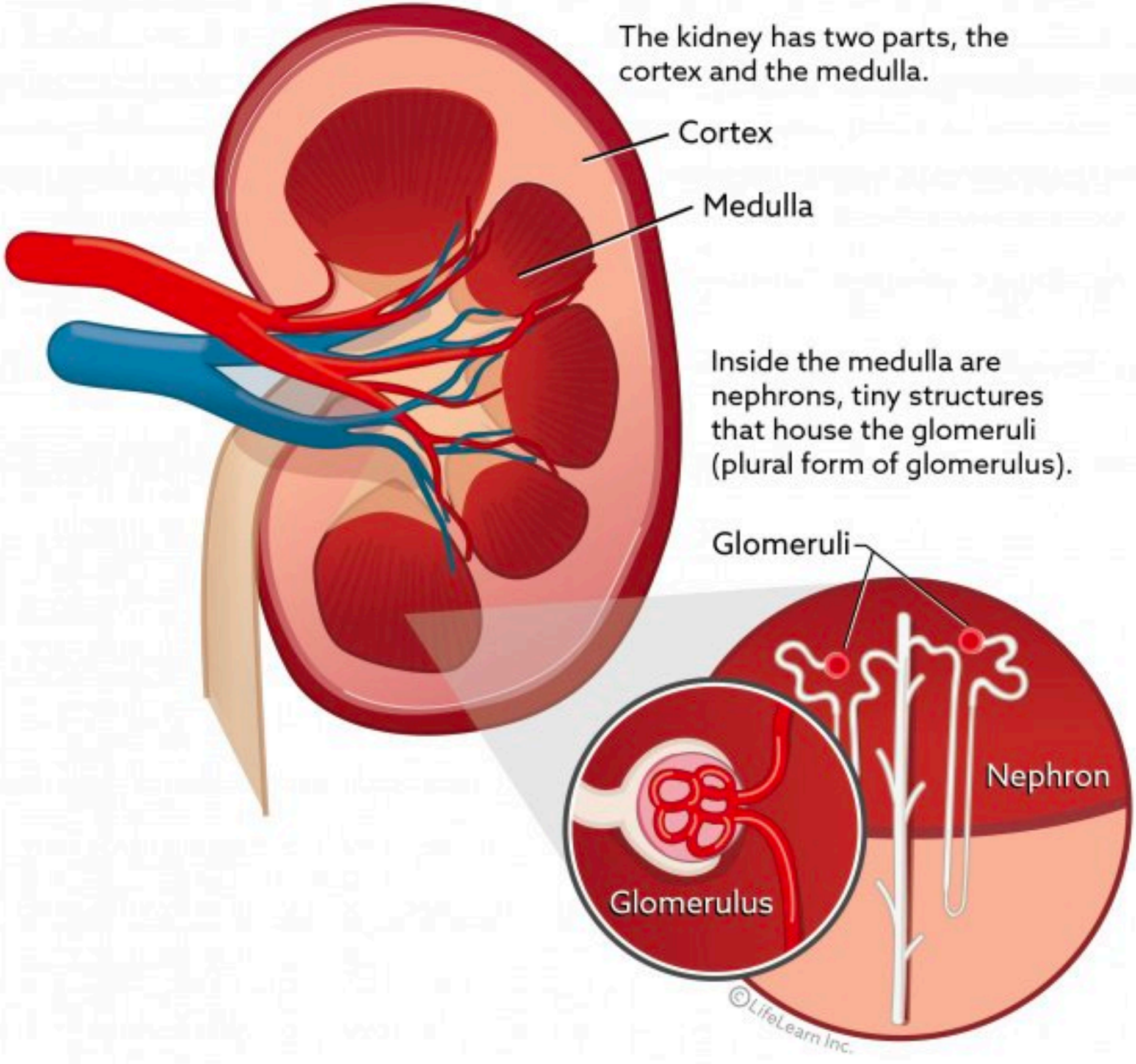
(3) Glomerulonephritis

(1) It occur as a primary diseases or as a component of diseases affecting several body systems, such as Equine infectious anemia
(2) It affects the renal glomeruli although the inflammatory process extends to affect the surrounding interstitial tissue and blood vessels. It is rare in ruminants.

Pathogenesis

Glomerular injury, circulating antigen-antibody complexes (Immunity) may be deposited in the glomerulus.


2.0 cm


The kidney has two parts, the cortex and the medulla.
Cortex
Medulla
Inside the medulla are nephrons, tiny structures that house the glomeruli (plural form of glomerulus).
Glomeruli
Nephron
Glomerulus
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Symptoms and Diagnosis

They depend on the primary causes:
(1) Many affected animals are asymptomatic until found dead.
(2) Some have signs of tachycardia, edema of the conjunctiva, nystagmus, walking in circles and convulsions.
(3) There is severe proteinuria and low plasma albumin.
(4) Blood urea is increased in lamb (greater than 35 mmol/L) with hyperphosphatemia and hypocalcemia.
(5) At postmortem the kidneys are large and pale and have multifocal pinpoint yellow and red spots throughout the cortex.
(6) On histopathological examination there are severe vascular lesions in the choroid plexuses and the lateral ventricles of the brain.

(4) Interstitial nephritis(1) It is rarely recognized in animals. It is seen in PM findings.
(2)It may be diffuse or have a focal distribution in calves, white-spotted kidney) due to destruction of nephrons.
(3) The kidney is an important reservoir for leptospira sp., particularly cattle. The disease begins with acute tubular nephrosis. Horses with chronic interstitial nephritis have the clinical syndrome of chronic renal failure with uremia.

5. Embolic nephritis

Clinical signs occur when embolic lesions are very extensive, in which septicemia may be followed by uremia as well as transient proteinuria and pyuria

Etiology

(1) Septicemia or bacteremia when bacteria lodge in renal tissue lead to embolic suppurative nephritis or renal abscess.
(2) Localized septic processes from vulvar endocarditis, suppurative lesions in uterus, udder, navel, peritoneal cavity in cattle. Be associated with systemic infections such as septicemia in neonatal animals including shigellosis in foals and E. coli septicemia in calves Erysipelas in pigs Septicemic or bacteremic strangles in horses.

Pathogenesis

(1) Bacterial emboli localize in renal tissue and causing focal suppurative lesions.
(2) Emboli can block larger vessels and cause infarction of portions of kidney,
(3) Presence of proteinuria, casts, and microscopic hematuria.
(4) The gradual enlargement of focal embolic lesions leads to toxemia and gradual loss of renal function.
(5) Clinical signs usually develop only when multiple emboli destroy much of the renal parenchyma, or when there is one or larger infected infarcts.

Clinical findings

(1) Signs of toxemia.
(2) The kidney may be enlarged on rectal examination.
(3) Repeated showers of emboli or gradual spread from several large, suppurative infarcts may cause fatal uremia.
(4) Spread to the renal pelvis may cause signs similar to pyelonephritis.
Clinical pathology:
(1) Hematuria and pyuria.
(2) Proteinuria is present but is also normally present in neonatal animals in the first 30-40 hours of life.
(3) Culture of urine at the time when proteinuria occurs may reveal the identity of the bacteria infecting the emulous.
(4) Hematology reveals acute or chronic inflammatory process.

Necropsy findings

(1) Small gray spots in the renal cortex in early stage.
(2) Large abscesses in later stages may be extending into pelvis.
(3) Fibrous tissue may surround long standing and healed lesions, consist of areas of scar tissue in cortex, causing depressed surfaces due to destruction of cortical tissue.
(5) Extensive scaring may cause an obvious irregular reduction in the size of the kidney.

Differential diagnosis

(1) Pyelonephritis.
(2) Prerenal uremia and ischemic tubular nephrosis in severely dehydrated neonatal animals. The presence of other signs of sepsis can reveal the presence of embolic nephritis.
(3) The sudden acute abdominal pain in renal diseases suggest acute intestinal obstruction but defecation is not affected and rectal examination of intestine is negative.

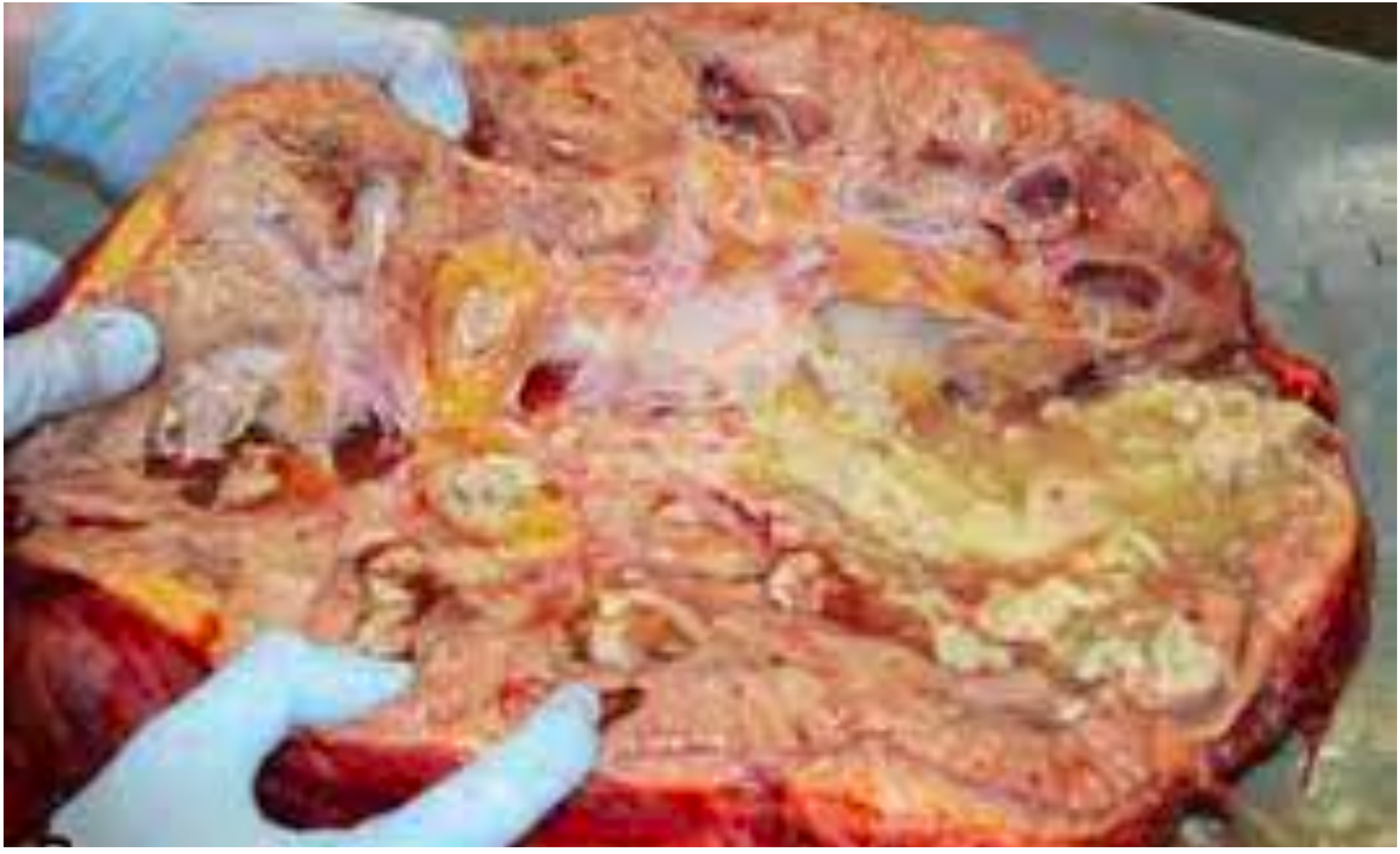
Treatment

(1) Treat the real cause with renal wash and antiseptic.
(2)Antimicrobials after urine culture and sensitivity. It should be continued for long period (7-14 days) or more.
(3) Avoid the use of nephrotoxic drugs.
(3) Treatment the septic shock in neonatal animals.

6. Pyelonephritis

Definition

It is a suppurative infection of kidney caused by ascending infection from the lower urinary tract. It is characterized by enlarged kidney, pyuria, suppurative nephritis, cystits and ureteritis as well as abnormal urine contents.



Etiology

(1) Secondary to bacterial infections of the lower urinary tract

(2) Spread from embolic nephritis of hematological origin such as septicemia in cattle caused by Pseudomonas aeruginosa.

(3) Specific pyelonephritis in cattle caused by *Corynbacterium renale* alone or mixed with *C.pilosum, C.cystitidis, C.pseudotuberclosis, Actinomyces (Coiyncbacterium) pyogens, Actinobacilins equuli, Ecoli and Staph aureus*.

Occurrence

(1) It is widespread in all countries in cattle but rare in sheep.
(2) Cows are more susceptible than bulls.
(3) All ages of mature cows are susceptible.

Source of infection

(1) Urine of affected or carrier animals and C. renale can also be isolated from the vagina or vaginal vestibule of urinary tract of healthy or carrier animals.

Transmission

(1) Infection can be transmitted by direct contact or by the use of contaminated brushes.
(2) Careless use of catheters.
(3) Venereal (The organism was isolated from the prepuce, urethra, and the semen of bulls).

Risk factors

(1) In cows, It is more common in early lactation and rare in second lactation.
(2) Cold seasons of the year and heavily fed, high-producing dairy herds.
(3) It is low in cows that have post parturient uterine disease that treated with antibiotics.
(4) Obstructive urinary abnormalities in bulls.
(5) Technique or infection of urinary catheterization.

Pathogenesis

(1) Pyelonephritis usually develops as an ascending infection from bladder, ureters, and kidneys.

(2) Trauma to the urethra, or urine stasis, may facilitate ascending infection. The destruction of renal tissue obstruction of urinary outflow ultimately result in uremia and the death of the animal.

(3) C. renale have a greater ability to attach to urinary tract epithelium, are more resistant to phagocytosis and make the initial ascending infection.

Clinical findings

(1) The first sign may be the passage of blood-stained urine.
(2) In other cases, the first sign may be an attack of acute colic, (raising of the tail, and kicking at the abdomen) and straining to urinate, the attack passing off in a few hours due to obstruction of ureter or renal calyx by pus or tissue debris.
(3) The onset is gradual with a fluctuating temperature (about 39.5°C), decreases appetite, loss of condition, and fall in milk yield over a period of weeks.
(4) Presence of blood, pus, mucus, and tissue debris in the urine, particularly in the last urine part.
(5) Dribbling and painful urination.

(6) Rectal palpation reveals an enlargement of kidney, loss its lobulation and pain. Later on, thickening of bladder wall and enlargement of one or both ureters (Cord-like).
(7) In many cases there is only weight loss and suspected gastrointestinal disease so urine analysis is essential.
(8) Unless early treatment, the disease is highly fatal.
(9) The course is usually several weeks or even months and the terminal signs are uremia then death.

Clinical pathology

(1) Urine analysis and examination:
1) Proteinuria and hematuria.
2) Urine pH is greater than B.5 (More alkaline).
3) Higher specific gravity vary between 1.008 and 1.021
4) Microscopic examination will show pyuria and presence of C.renale in urine culture (Gram +ve, Chinese-like litter or palisade-like cluster).
(2) Blood and serum contents:
1) Hypoalbuminemia and hyper gamma globulinemia.
2) Neutrophilia may be present.
3) Elevation of serum creatinine and urea (above 1.5 mg/dL and 100 mg/dL respectively, this means bad prognosis).
(3) Renal biopsy and ultrasound reveal dilated renal collecting system.
(4) Endoscopic examination reveals enlarged urethra and thick bladder.

Necropsy findings

(1) The kidneys are usually enlarged and loss of lobulation.
(2) The renal calyces and enlarged ureters contain blood, pus, and mucus.
(3) Light colored necrotic areas on the kidney surface.
(4) Changes visible on the cut surface include excavation of papillae, abcessation, and necrosis which extend from the distal medulla into the cortex.
(5) The bladder and urethra are thick-walled and their mucous membranes are hemorrhagic, edematous, and eroded.
(6) Histologically, the renal lesions are a confusing mixture of acute suppurative changes and various degrees of fibrosis with mononuclear cell infiltration.

Differential diagnosis

(1) Acute colic in acute intestinal obstruction: normal kidney and urine.

(2) Chronic cases may be confused with traumatic reticulitis, normal kidney and urine.

(3) Sporadic cases of non-specific cystitis can only be differentiated by culture of the urine.

(4) Polypoid cystitis is a non-specific result of bladder inflammation and may be a cause of dysuria and obstructive uropathy.

Treatment

(1) Large doses of procaine penicillin G 15000 IU/kg BW, IM, daily for at least 3 weeks.
(2) Monobasic sodium phosphate or sodium acid phosphate (125 gm dissolved in drinking water daily for several days to acidify urine).
(3) IV glucose (Urinary lavage).
(4) Urinary antiseptic, diuretic, sedative.
(5) Sufficient quantity of water, less nitrogenous food.
NB: Improvement of appetite and milk yield and clearing of the urine indicate good prognosis.

Control

(1) Isolation of affected animals and destruction of infected litter and bedding.

(2) Hygienic measures for parturition, catheterization, etc.

(3) Excess water intake, vitamin A and C.

Hydronephrosis

Definition

It is a dilatation of the renal pelvis with progressive atrophy of the renal parenchyma. It occurs as a congenital or an acquired condition following obstruction of the urinary tract.

Causes

(1) Any urinary tract obstruction.
(2) Urolithiasis in ruminants.
(3) If the obstruction is unilateral, the unaffected kidney can compensate fully for the loss of function and the obstruction may not cause kidney failure (Unilateral obstruction may be detectable on palpation per rectum of a grossly distended kidney).
(4) Chronic partial obstruction of the penile urethra by a urolith causes hydronephrosis and chronic renal failure in a steer.
(5) Papillomas of the urinary bladder causes partial obstruction of the ureters in cows.

Symptoms, diagnosis and treatment: Depend on the main cause.

(8) Renal neoplasms

Primary tumor of the kidney are uncommon. Carcinoma occur in cattle and horses. Enlargement of the kidney is the characteristic sign, in cattle and horses neoplasm should be considered in differential diagnosis of renal enlargement.