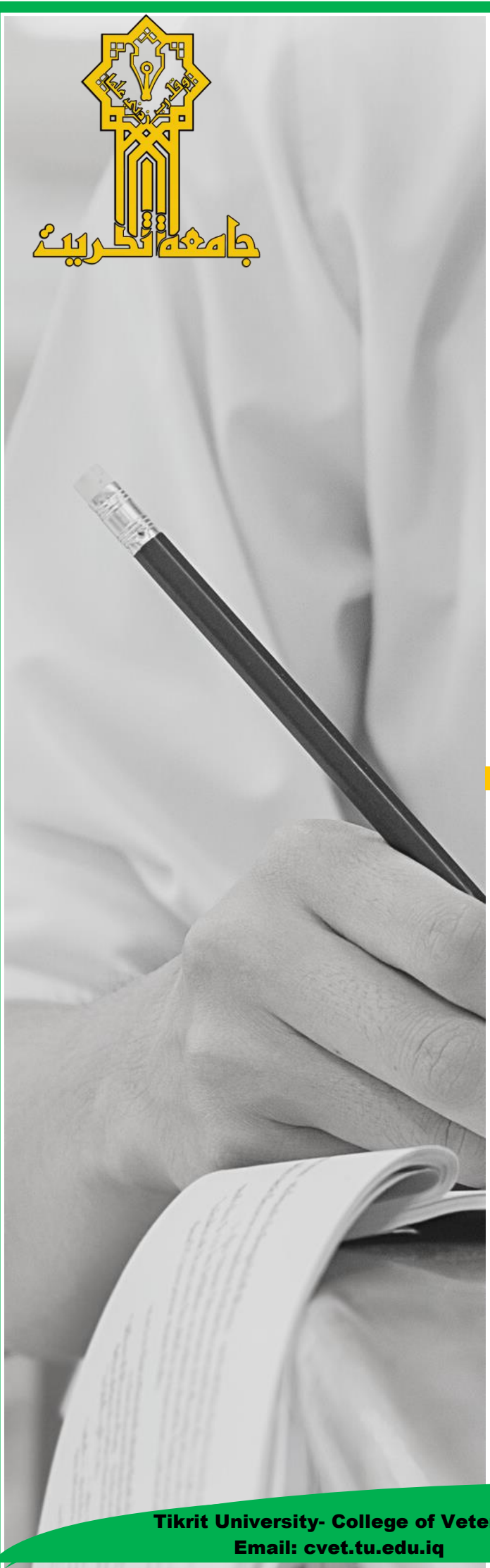

Tikrit University
College of Veterinary Medicine

Diseases of urinary diseases

Subject name: internal medicine

Subject year: 2024-2025

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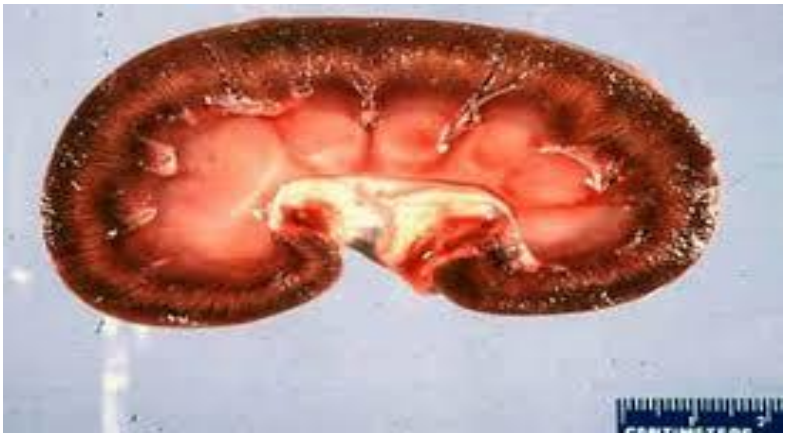
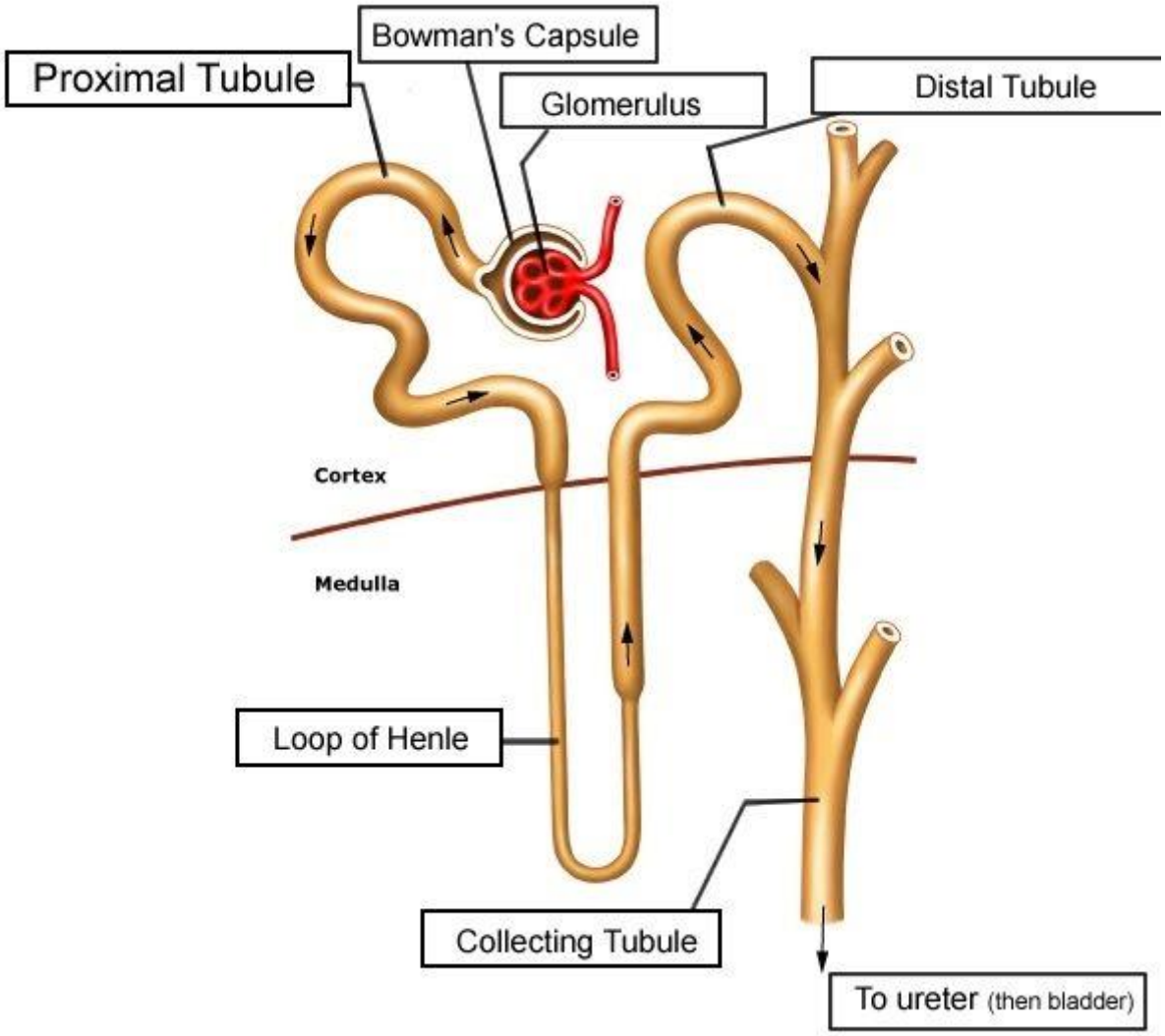
Lecturers link

Toxic nephrosis





Etiology

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

•Toxins

• Metals: mercury, arsenic, cadmium, selenium, and organic copper compounds.

• Antimicrobials, such as aminoglycosides, and overdosing with neomycin and gentamicin in the treatment of calves.

• repeated daily dosing with long-acting oxytetracycline preparations may induce toxicity;

• treatment with sulfonamides

• Horses treated with vitamin K3 (menadione sodium bisulfite)

• Horses treated with vitamin D2 (ergocalciferol) and cholecalciferol (D3)

•Treatment of horses with nonsteroidal antiinflammatory drugs (NSAIDs), including phenylbutazone and flunixin meglumine; dose rates of more than 8.8 mg/kg BW of phenylbutazone per day for 4 days are likely to cause nephrosis.

•Ketoprofen in sheep at 30 mg/kg IV,once2; renal toxicity may be facilitated

Pathogenesis

•In acute nephrosis there is obstruction to the flow of glomerular filtrate through the tubules caused by interstitial edema and intraluminal casts.

• there may be back leakage of glomerular filtrate into the interstitium. decreases glomerular filtration.

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

Clinical findings

•Clinical signs may not be preferable to the urinary system.

• In Peracute cases, such as those caused by vitamin K3 administered by injection, there may be colic and stranguria.

•In acute nephrosis there is oliguria and proteinuria with clinical signs of uremia in the terminal stages.

•These signs include depression, dehydration, anorexia, hypothermia, a slow or an elevated heart rate, and weak pulse.

•Diarrhea may be present that is sufficiently intense to cause severe clinical dehydration.

Clinical pathology

• Proteinuria, glucosuria, enzymuria, and hematuria are initial changes on urinalysis in experimental toxic nephrosis.

•The earliest indication is the detection of the proximal tubule enzyme GGT in urine.

•Hypoproteinemia may be present.

•In horses, hypercalcemia and hypophosphatemia can be present.

Necropsy findings

In acute cases the kidney is swollen and wet on the cut surface and edema, especially of perirenal tissues, may be apparent.

Treatment

• supportive care .

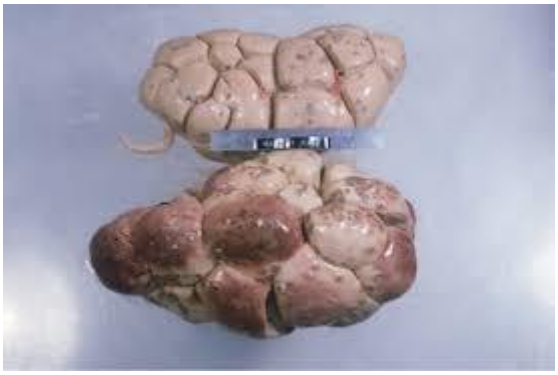
• If the toxin can be identified, it should be removed.

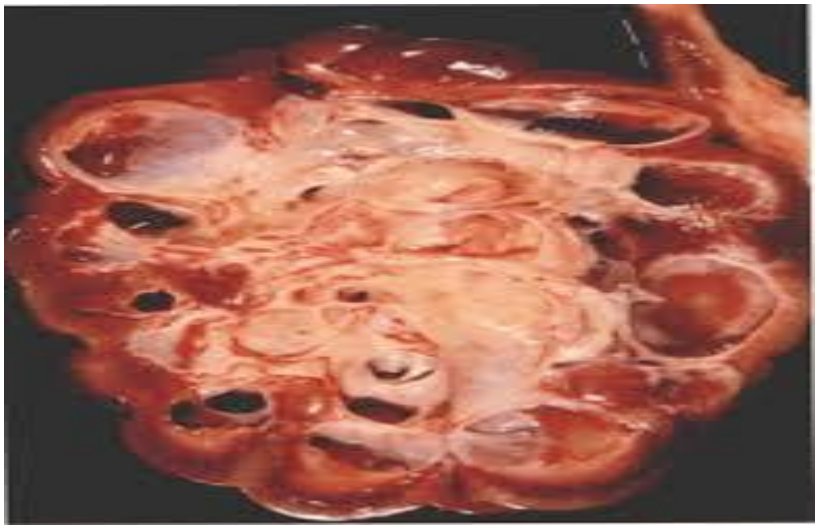
Differential diagnosis

• Diabetes mellitus is rare in horses and extremely rare in ruminants.

• Cushing’s syndrome (chronic hyperadrenocorticism pituitary pars intermedia dysfunction) is more common in horses

Hydronephrosis





•Hydronephrosis 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.

•In cases of acute complete obstruction the clinical picture is dominated by signs of anuria, dysuria, or stranguria.

•Chronic or partial obstructions cause progressive distension of the renal pelvis and pressure atrophy of the renal parenchyma.

• If the obstruction is unilateral, the unaffected kidney can compensate fully for the loss of function and the obstruction may not cause kidney failure.

•Unilateral obstruction may be detectable on palpation per rectum of a grossly distended kidney.

•Hydronephrosis and chronic renal failure have been recorded in a steer suffering from chronic partial obstruction of the penile urethra by a urolithiasis.

•Partial obstruction of the ureters by papillomas of the urinary bladder has been recorded in a series of cows.

•Compression by neoplastic tissue in cases of enzootic bovine leukosis may also cause hydronephrosis.

•Ultrasonography can be used as an aid to diagnosis.

Interstitial nephritis

Chronic interstitial nephritis – cow

•Interstitial nephritis may be diffuse or have a focal distribution.

•In calves, focal interstitial nephritis (white-spotted kidney) is a common incidental finding at necropsy but does not present as a clinical urinary tract disease.

•In pigs, diffuse interstitial nephritis is observed following infection by *Leptospira* sp. and is important clinically because of the resultant destruction of nephrons that occurs.

•Chronic interstitial fibrosis is a common postmortem finding in horses suffering from chronic renal failure.

•Horses with chronic interstitial nephritis have the clinical syndrome of chronic renal failure with uremia.

Embolic nephritis

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

•Even though embolic nephritis may not be clinically evident, transient proteinuria and pyuria may be observed if urine samples are examined at frequent intervals.

Etiology

•Emboli may originate from localized septic processes such as

· Valvular endocarditis, in all species

· Suppurative lesions in uterus, udder, navel, and peritoneal cavity in cattle or be associated with systemic infections.

· Septicemia in neonatal animals, including *Actinobacillus equuli* infection in foals and *E. coli* septicemia in calves

· Erysipelas in pigs and *C. pseudotuberculosis* in sheep and goats

· Septicemic or bacteremic *Streptococcus equi* infection in horses

Pathogenesis

•Bacterial emboli localize in renal tissue and cause the development of focal suppurative lesions.

•Emboli can block larger vessels and cause infarction of portions of kidney, with the size varying with the caliber of the occluded vessel.

•The gradual enlargement of focal embolic lesions leads to the development of toxemia and gradual loss of renal function.

•Clinical signs usually develop only when multiple emboli destroy much of the renal parenchyma, or when there are one or more large infected infarcts.

Clinical findings

•Signs of toxemia and the primary disease are usually present.

•The kidney may be enlarged on rectal examination.

•Repeated showers of emboli or gradual spread from several large, suppurative infarcts may cause fatal uremia.

• Spread to the renal pelvis may cause signs similar to pyelonephritis.

•Large infarcts may cause bouts of transient abdominal pain.

Clinical pathology

•Hematuria and pyuria are present in embolic nephritis.

•Hematology usually reveals evidence of an acute or chronic inflammatory process.

Necropsy findings

• small gray spots in the cortex.

•Fibrous tissue may surround longstanding lesions, and healed lesions consist of areas of scar tissue in the cortex.

Treatment

•Antimicrobials should be selected on the basis of quantitative urine culture and susceptibility testing.

•Antimicrobial treatment should be continued for a fairly lengthy period (7‑14days).

• In neonatal animals this may involve treatment for septic shock.

Differential diagnosis

--Differentiation from pyelonephritis is difficult unless the latter is accompanied by signs of lower urinary tract infection such as cystitis or urethritis.

--Severely dehydrated neonatal animals may experience prerenal uremia and are susceptible to ischemic tubular nephrosis.

--The presence of other signs of sepsis should increase suspicion of the presence of embolic nephritis.