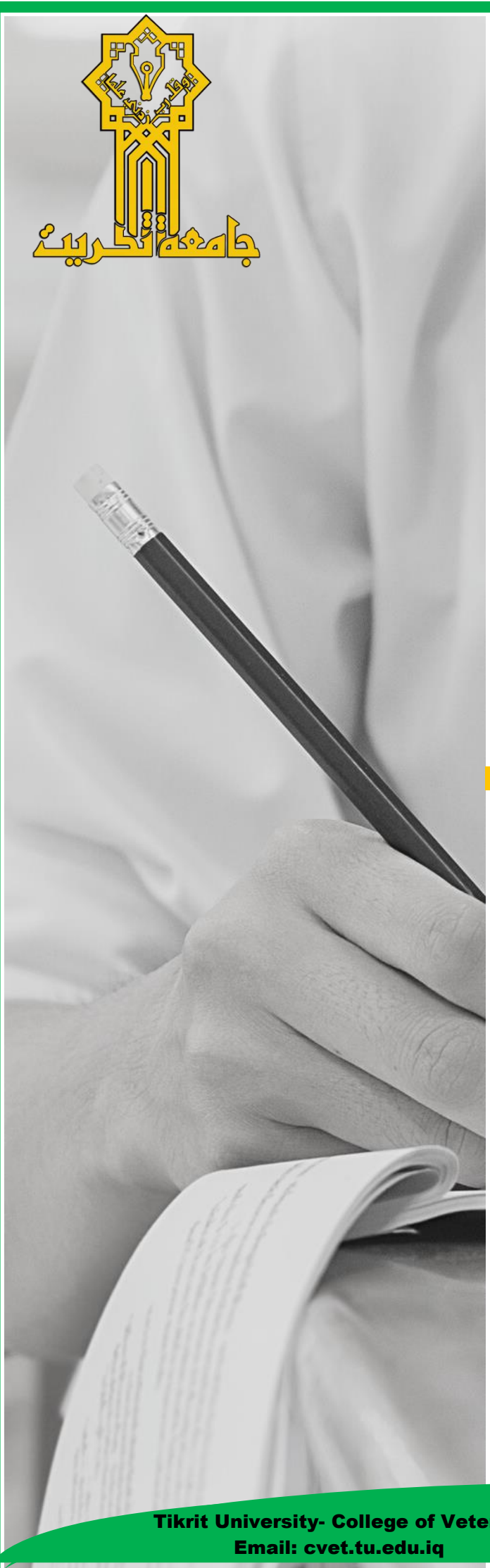

Tikrit University
College of Veterinary Medicine

Diseases of urinary diseases

Subject name: internal medicine

Subject year: 2024-2025

Lecturer name: Jassim Mohamed Suleiman

Academic Email: drjassimms1980@tu.edu.iq

Lecturers link

Renal insufficiency and renal failure

Renal function depends on the number and functionality of the individual nephrons.

*Renal insufficiency can occur from abnormalities in the

· Rate of renal blood flow

· Glomerular filtration rate

· Efficiency of tubular reabsorption.

the latter two are intrinsic functions of the kidney, whereas the first depends largely on vasomotor control, which is markedly affected by circulatory emergencies such as shock, dehydration, and hemorrhage.

Causes of renal insufficiency

The causes of renal insufficiency, and therefore of renal failure can be divided into prerenal, renal, and Postrenal groups.

1- Prerenal causes

include congestive heart failure and acute circulatory failure, either cardiac or peripheral, in which acute renal ischemia occurs in response to a decrease in renal blood flow. hypoxic damage because of the low oxygen tension in this tissue, and the high metabolic rate of this tissue. Renal medullary necrosis.

2- Renal causes

include glomerulonephritis, amyloidosis, pyelonephritis, embolic nephritis, and interstitial nephritis. The disease is also secondary to sepsis and hemorrhagic shock.

3- Postrenal uremia

complete obstruction of the urinary tract by vesicles or urethral calculus, or by transitional cell carcinoma located in the trigone

region of the bladder. Internal rupture of any part of the urinary tract, such as the bladder, ureters, or urethra.

Pathogenesis of renal insufficiency and renal failure

• Damage to the glomerular epithelium destroys its selective permeability and permits the passage of plasma proteins into the glomerular filtrate. The predominant protein is initially albumin, because of its negative charge and a lower molecular weight than globulins;

• Glomerular filtration may cease completely when there is extensive damage to glomeruli, particularly if there is acute swelling of the kidney.

•When renal damage is less severe, the remaining nephrons compensate to maintain total glomerular filtration by increasing their filtration rates.

•Decreased glomerular filtration also results in retention of metabolic waste products such as urea and creatinine. the serum urea nitrogen (SUN) concentration can be used to monitor glomerular filtration rate.

•In contrast, creatinine is excreted almost entirely by the kidney, creatinine originates from the breakdown of creatine phosphate in muscle, and serum concentrations of creatinine are a useful marker of glomerular filtration rate.

•Loss of tubular resorptive function is evidenced by a continued loss of sodium and chloride; hyponatremia and hypochloremia eventually occur in all cases of renal failure. The continuous loss of large quantities of fluid from solute diuresis can cause clinical dehydration.

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

•The renal tubules respond to ADH by conserving water and returning serum osmolality to normal, producing concentrated urine.

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

•acute renal ischemia might result, leading to acute renal failure. Prolonged hypoproteinemia results in rapid loss of body condition and muscle weakness. Acidemia secondary to metabolic acidosis and hyponatremia can also be a contributing factor to muscle weakness and mental attitude. All these factors play some part in the production of clinical signs of renal failure.

Glomerulonephritis

•Glomerulonephritis can occur as a primary disease or as a component of diseases affecting several body systems, such as equine infectious anemia and chronic swine fever.

•In primary glomerulonephritis, the disease involves only the kidney, predominantly affecting the glomeruli, although the inflammatory process extends to affect the surrounding interstitial tissue and blood vessels.

•The disease is sometimes associated with other chronic, systemic illness such as in cows with Johne's disease, bovine virus diarrhea, fascioliasis,or leptospirosis;

•The immune system plays a major role in the pathogenesis of glomerular lesions.

•Glomerular injury can be initiated by an immune response in which antibodies are directed against intrinsic glomerular antigens or by foreign antigens planted in the glomerulus.

•As the complexes accumulate, they stimulate an inflammatory response that damages the glomerular filtration system.

•Glomerulonephritis is a common cause of chronic renal failure in horses. Several forms of glomerulonephritis are recognized in horses: membranous glomerulonephritis, poststreptococcal

glomerulonephritis, membranoproliferative glomerulonephritis, and focal glomerulosclerosis.

Nephrosis

•Nephrosis includes degenerative and inflammatory lesions primarily affecting the renal tubules, particularly the proximal convoluted tubules.

•Nephrosis is classified into two main groups:

(1) tubular injury caused by ischemic insult .

(2) cell death or damage to the tubules caused by nephrotoxins .

ISCHEMIC NEPHROSIS

•Reduced blood flow through the kidneys usually is caused by general circulatory failure.

Etiology

•Any condition that predisposes the animal to marked hypotension and release of endogenous pressor agents potentially can initiate hemodynamically mediated acute renal ischemia and renal failure.

•Ischemia may be acute or chronic.

1-Acute Renal Ischemia

• General circulatory emergencies such as shock, dehydration, acute hemorrhagic anemia, and acute heart failure; renal failure secondary to calf diarrhea .

• Embolism of renal artery, recorded in horses

2-Chronic Renal Ischemia

· 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 mean arterial blood pressure decreases below 60 mm Hg glomerular filtration decreases, and metabolites that are normally excreted accumulate in the bloodstream.

•The concentration of urea nitrogen in plasma or serum increases, giving rise to the name prerenal uremia.

•As glomerular filtration falls, tubular resorption increases, causing reduced urine flow.

•Up to a certain stage, the degenerative changes are reversible by restoration of renal blood flow, but if ischemia is severe enough and of sufficient duration, the renal damage is permanent.

•Proximal tubules are highly sensitive to ischemia because they are one of the energetically most active cells in the body.

Clinical findings

•Renal ischemia does not appear as a distinct disease, and its signs are masked by the clinical signs of the primary disease.

•Oliguria and azotemia will go unnoticed in most cases if the circulatory defect is corrected in the early stages.

•renal insufficiency may cause a poor response to treatment with transfusion or the infusion of other fluids in hemorrhagic or hemolytic anemia, in shock or dehydration.

Clinical pathology

•Laboratory tests can be used to evaluate renal function once the circulatory condition has been corrected.

•Urinalysis as well as SUN and creatinine concentrations are common indices.

•Serum biochemistry on serially collected samples may also be used to monitor the response to therapy.

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

Necropsy findings

Lesions of renal ischemia are present primarily in the cortex, which is pale and swollen. There may be a distinct line of necrosis visible at the corticomedullary junction.

Treatment

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

•If renal damage has occurred, supportive treatment as suggested for the treatment of acute renal failure should be instituted.