

MODULE 3

RENAL SYSTEM

Kidney purpose:

- regulate fluid balance and acid base
- excrete waste via urine
- excrete drugs
- secrete hormones including erythropoietin (RBC production), Vit D3 and renin, aldosterone, ADH.
- blood pressure regulation
- filter blood to produce urine

To function kidneys need:

- adequate blood flow and pressure through kidneys
- a clear urinary tract to excrete urine
- blood goes through afferent to glomerulus and out efferent, blood goes through bowmans capsule and through proximal tubule and proximal collecting tubule which Na⁺ and some urea are reabsorbed, then through loop of henle and out through distal tubule.
- Creatine (large molecule) is filtered by glomerular and further secreted into urine by the distal tubule.
- Urea is filtered and then 50% is reabsorbed (it's inhibits water reabsorption)

Acute kidney injury

- An acute and sudden deterioration in kidney function that presents in hours or days.
- common causes are sepsis and low blood pressure.
- 3 stages of damage to kidneys measured by amt of creatinine increase and urine output decrease, dehydration
- detected by low urine output, or rise in serum creatinine
- Causes of AKI can be pre-renal, Renal or Post renal.
- creatine is a waste product produced by muscles which is removed from the body by the kidneys, if a persons kidney function is reduced the levels of creatine in blood will be higher. Normal range is 60-120micromol/L
- pt has AKI if there output is less than 0.5ml/kg/hr for 6+ hours.
- Symptoms include decreased urine output, thirst, poor fluid intake, diarrhoea & vomiting, raised temp
- Treatment- removal of obstruction, Renal Replacement Therapy RRT (dialysis), haemofiltration.
- Diuretic not used as they can worsen outcomes (unless there is APO in relation to AKI).
- Prevention: avoidance of hypovolemia (low body fluid volume), nephrotoxic drugs and contrast.
- More common amongst elderly due to more medications being taken and co morbidities
- The urea and electrolyte blood test is used to assess kidney function, elevated levels indicate reduced kidney function
- Urea is a waste product of protein, elevated levels indicate that the kidneys are unable to excrete it
- The ratio for urea to creatinine is 25:1 a rise in either is indicative of AKI

Pre-renal:

- blood flow through kidney is reduced= hypoperfusion of kidney,
- commonly caused by prolonged hypotension, heart failure, arrhythmias & anti hypertensive medications.
- Decreased CO= Decreased glomerular filtration rate= kidneys release rennin which releases angiotensin, aldosterone and ADH which cause vasoconstriction to increase BP, Aldosterone and ADH also increase reabsorption of Na⁺, H₂O and urea which results in less urine output, more concentrated urine (higher specific gravity), and higher levels of urea and creatinine in the blood circulation.
- Decreased GFR=Less urea is filtered but urea is more effectively reabsorbed by kidneys due to slow filtration rate and more time to reabsorb in the proximal tubule.
- Decreased GFR= less creatinine filtered but still a lot secreted
- (creatinine and urea levels both rise but urea more so)
- Normal levels of urea to creatinine is 15:1, in someone with decreased GFR it could be 40:2.
- Urinalysis would reveal less Na⁺ and a higher Specific gravity

Renal:

- Pathology within the kidney (pts may be well hydrated with normal BP).
- Damage to the nephrons or surrounds
- Caused by kidney disease, medications- NSAIDS, aminoglycosides (antibiotics), ACE inhibitors, toxins- iodinated contrast used in imaging & systemic disease eg lupus, myeloma, vasculitis.

Types:

Acute Glomerulonephritis: inflammation of glomerulus (blood vessels) within the nephron which can cause:

- decreased GFR,
- increased capillary permeability (larger components getting into filtrate eg protein, RBC),
- decreased blood flow through efferent arteriole which provides blood to cells surrounding nephron .
- Decreased GFR causes retention of Na⁺, H₂O, K⁺, Cl⁻ ect.
- This leads to hypertension, azotaemia (retention of creatinine and urea in blood), malaise, N&V.
- Causes: infection of glomerulus, damage from distance infections, immunological disorders- lupus, drugs.
- Can cause permanent damage and cause progressive loss of kidney function

Acute tubular necrosis (death of tubular cells)

- These cells secrete and reabsorb substances
- causes: drop in efferent blood flow which causes lack of perfusion to these cells=cells become ischemic= death,
- trauma, sepsis, anaphylaxis, burns, major surgery, nephrotoxic agents, drugs, contrasts, immuno suppressants, blocked tubules caused by skeletal muscle breakdown which releases myoglobin.
- Urea gets filtered into filtrate but does not get reabsorbed so passes into urine.
- Creatinine is filtered into filtrate but no more is secreted.
- Levels of both still increase in the blood but the ratio stays the same and they increase the same amount.

- Obstruction from dead cells can occur causing decreased GFR = RAAS= further damage.

Post-renal:

- obstruction to urinary flow beyond the kidney.
- Obstruction leads to back pressure and swelling within the kidney (hydronephrosis) which leads to AKI.
- Causes; enlarged prostate, stones, structure of ureters, pelvic/bladder masses, blocked IDC.
- most rare type of AKI
- urine output decreases, pain, ultrasound to diagnose, catheter to relieve pressure.
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