- **Introduction:**

Baseline Creatinine Levels:

Creatinine is a byproduct of muscle metabolism, and its level in the blood reflects kidney function. Baseline creatinine levels vary depending on factors such as age, sex, muscle mass, and hydration status.

Normal Range of Creatinine:

- Men: 0.74–1.35 mg/dL (65–120 µmol/L)
- Women: 0.59–1.04 mg/dL (52–91 µmol/L)
- Children: 0.2–1.0 mg/dL, depending on age

-Factors Influencing Creatinine Levels

1. **Increased Levels:**

    - **Kidney dysfunction (e.g., acute kidney injury, chronic kidney disease)**
    - **Dehydration**
    - **High muscle mass or intense exercise**
    - **Use of nephrotoxic drugs**
    - **Conditions like rhabdomyolysis or severe infections**

2. **Decreased Levels:**

    - Low muscle mass (e.g., elderly or malnourished patients)
    - Pregnancy (due to increased renal clearance)

- **Assessing Creatinine Levels in Patients:**

1. **Blood Test (Serum Creatinine):**

    - The most common method to measure creatinine.
    - Requires a simple blood draw.
    - Often ordered as part of a Basic Metabolic Panel (BMP) or Comprehensive Metabolic Panel (CMP).

2. **Urine Creatinine Test:**

    - Helps evaluate creatinine clearance and kidney function.
    - Usually combined with a 24-hour urine collection.

3. Glomerular Filtration Rate (GFR):

- Calculated using serum creatinine and factors like age, sex, and body size.
- Helps assess kidney function more accurately than serum creatinine alone.
- Estimated GFR (eGFR) formulas: MDRD, CKD-EPI, or Cockcroft-Gault.

4. Creatinine Clearance Test:

- Uses both blood and urine creatinine levels to estimate kidney clearance.
- Requires a 24-hour urine collection and blood sample.
- Monitoring and Trends:
- Baseline Measurement: It's crucial to establish a baseline creatinine level when the patient is healthy.
- Trends Over Time: Serial measurements are more informative than single readings. An increase of 0.3 mg/dL or more within 48 hours indicates acute kidney injury (AKI).
- When to Investigate Creatinine Levels
- Symptoms of kidney disease (e.g., swelling, fatigue, high blood pressure)
- Monitoring chronic conditions (e.g., diabetes, hypertension)
- Before prescribing nephrotoxic drugs
- Post-surgery or in critically ill patients to monitor kidney function
- During hospital stays for septic, hypovolemic, or other at-risk patients

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Abnormal **creatinine** levels can indicate kidney dysfunction, but the specific cause and significance depend on whether the creatinine level is high or low, and the patient's overall clinical context.

1. **High creatinine levels**:
   - **Acute kidney injury (AKI)**: Can be caused by dehydration, infections, medications (e.g., NSAIDs, ACE inhibitors), or obstruction (e.g., kidney stones).
   - **Chronic kidney disease (CKD)**: Long-term conditions such as diabetes, hypertension, or glomerulonephritis can lead to elevated creatinine.
   - **Decreased renal function**: If the kidneys can't filter waste effectively, creatinine builds up in the blood.
2. **Low creatinine levels**:
   - **Decreased muscle mass**: Since creatinine is a byproduct of muscle metabolism, low muscle mass (e.g., in elderly individuals, malnutrition, or conditions like muscular dystrophy) can lead to lower creatinine levels.
   - **Pregnancy**: Pregnant women often have lower creatinine due to increased renal clearance.
   - **Severe liver disease**: In rare cases, severe liver dysfunction can result in lower creatinine production.

Acute Kidney Injury (AKI) is a sudden decline in kidney function, characterized by a rapid increase in serum creatinine or a decrease in urine output. It can occur within hours to days and is

often reversible if the underlying cause is identified and treated promptly. AKI can lead to severe complications, so early recognition and management are critical.

1. Prerenal AKI

Prerenal causes refer to conditions that reduce **blood flow to the kidneys**, resulting in inadequate perfusion and ischemia. This leads to a decrease in the kidneys' ability to filter blood effectively.

Causes:

- **Hypovolemia**:
  - **Dehydration** (vomiting, diarrhea, excessive diuresis)
  - **Hemorrhage** (trauma, GI bleeding)
  - **Burns** (fluid loss through damaged skin)
  - **Third-spacing** (e.g., ascites, pancreatitis, or severe soft tissue injury)
- **Decreased cardiac output**:
  - **Heart failure** (reduced cardiac pumping ability)
  - **Myocardial infarction (MI)** (heart attack)
  - **Shock** (e.g., septic shock, anaphylactic shock, or neurogenic shock)
- **Systemic vasodilation**:
  - **Sepsis** (inflammatory response causes widespread vasodilation)
  - **Anaphylaxis** (severe allergic reaction with widespread vasodilation)
- **Renal artery stenosis**:
  - A narrowing of the renal arteries leading to decreased blood flow, typically due to **atherosclerosis** or **fibromuscular dysplasia**.
- **Medications**:
  - **Diuretics** (can cause dehydration)
  - **ACE inhibitors/ARBs** (especially in volume-depleted patients)
  - **NSAIDs** (inhibit prostaglandins, which help dilate the renal afferent arteriole)
  - **Antihypertensives** (can reduce renal perfusion in some cases)

Pathophysiology:

In prerenal AKI, the kidneys receive inadequate blood flow, which results in a reduced glomerular filtration rate (GFR). The kidneys attempt to conserve water and sodium to restore perfusion, but if the underlying cause isn't corrected, ischemic damage to renal tissues can occur, leading to intrinsic damage (ATN).

Clinical Features:

- **Hypotension** or **tachycardia** (signs of hypoperfusion)
- **Dry mucous membranes**, **oliguria**, or **anuria**
- **Elevated BUN/creatinine ratio** (>20:1), indicating prerenal origin (indicates a greater degree of urea reabsorption compared to creatinine)
- **Low urine sodium** (<20 mEq/L) and **high urine osmolality** (>500 mOsm/kg), reflecting the kidneys' attempt to conserve sodium and water.

Management:

- **Volume resuscitation** (IV fluids like saline or lactated Ringer's) to restore circulating volume
- **Treat the underlying cause** (e.g., stop medications, manage heart failure or shock)

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2. Postrenal AKI

Postrenal AKI occurs when there's an **obstruction** of urine outflow, leading to **increased pressure within the kidney**(backpressure) and subsequent renal damage. This obstruction can occur at any level of the urinary tract.

Causes:

- **Bladder outlet obstruction**:
  - **Benign prostatic hyperplasia (BPH)** in older men
  - **Prostate cancer**
  - **Urethral stricture** or **bladder neck obstruction**
  - **Neurogenic bladder** (e.g., in spinal cord injury or diabetic neuropathy)
- **Ureteral obstruction**:
  - **Kidney stones** (most common cause of obstruction)
  - **Ureteral stricture**
  - **Bladder or pelvic malignancies** (e.g., cervical or ovarian cancer)
  - **Blood clots** (from trauma or post-surgical)
- **Bilateral ureteral obstruction** (can happen from external compressive masses or stones)

Pathophysiology:

Obstruction leads to a build-up of urine in the kidneys (hydronephrosis). This causes elevated pressure in the renal tubules, which impairs filtration and can result in kidney damage. The blockage can be partial or complete. Chronic obstruction, if left untreated, can cause irreversible kidney damage.

Clinical Features:

- **Abdominal pain** (especially if there's a stone or severe obstruction)
- **Distended bladder** or **lower abdominal discomfort** (especially in cases of bladder outlet obstruction)
- **Oliguria or anuria** (depending on the severity of the obstruction)
- **Flank pain** or **hematuria** (in cases of stone or infection)
- **Ultrasound** is the most useful diagnostic tool, revealing **hydronephrosis** (swelling of the kidney due to urine retention).

Management:

- **Relieving the obstruction** is the primary treatment:
  - **Catheterization** (for bladder outlet obstruction)
  - **Stent placement** or **nephrostomy tube** (for ureteral or kidney obstruction)
  - **Stone removal** (via lithotripsy or surgery)
- **Address underlying causes** (e.g., treating prostate cancer, managing spinal cord injury)

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3. Renal (Intrinsic) AKI

Renal or intrinsic AKI refers to damage that occurs **within the kidney tissues** themselves, affecting the glomeruli, tubules, interstitium, or blood vessels. This type is often more severe and harder to treat compared to prerenal or postrenal causes.

Causes:

- **Acute Tubular Necrosis (ATN)**:
  - **Ischemic ATN**: Prolonged prerenal AKI that results in damage to the tubular cells due to lack of oxygen.
  - **Nephrotoxic ATN**: Caused by drugs or toxins such as:
    - **Aminoglycosides** (e.g., gentamicin)
    - **Contrast dyes** (e.g., in imaging studies)
    - **NSAIDs**, **ACE inhibitors**
    - **Rhabdomyolysis** (muscle injury releasing myoglobin)
    - **Hemolysis** (breakdown of red blood cells releasing hemoglobin)
- **Acute Interstitial Nephritis (AIN)**:
  - Often drug-induced (e.g., penicillins, NSAIDs, diuretics) or due to infections (e.g., pyelonephritis) or autoimmune diseases (e.g., systemic lupus erythematosus).
- **Glomerulonephritis**:
  - Inflammatory conditions affecting the glomeruli, including **IgA nephropathy**, **lupus nephritis**, and **post-streptococcal glomerulonephritis**.
- **Vascular diseases**:
  - **Vasculitis** (e.g., granulomatosis with polyangiitis)
  - **Thrombotic microangiopathies** (e.g., hemolytic uremic syndrome or thrombotic thrombocytopenic purpura)

Pathophysiology:

In intrinsic renal disease, the kidneys themselves are damaged, affecting their ability to filter blood. In **ATN**, for example, damage to the renal tubules results in **cellular sloughing** and **obstruction** within the tubules. This can result in a vicious cycle where the kidney's ability to clear waste diminishes, causing further renal injury.

Clinical Features:

- **Oliguria or anuria**

- **Proteinuria** (especially in glomerulonephritis)
- **Hematuria** (may be present in glomerulonephritis or ATN)
- **Rash** or **fever** (in interstitial nephritis)
- **Elevated creatinine** and **BUN** (more markedly than in prerenal AKI)
- **Urine analysis**: May show **casts** (e.g., **granular casts** in ATN, **RBC casts** in glomerulonephritis)

Diagnosis:

- **Urine sodium**: Elevated (>40 mEq/L) in intrinsic AKI due to impaired sodium reabsorption.
- **Fractional excretion of sodium (FeNa)**: >2% is typical for intrinsic AKI.
- **Urine microscopy**: May show **muddy brown casts** (indicative of ATN) or **RBC casts** (in glomerulonephritis).
- **Kidney biopsy** may be needed in certain cases (e.g., to confirm glomerulonephritis or vasculitis).

Management:

- **Treat the underlying cause** (e.g., discontinue nephrotoxic drugs, treat infection, manage autoimmune diseases)
- **Supportive care**:
    - **Fluid management**: Balance fluids carefully to avoid fluid overload.
    - **Electrolyte correction**: Address any imbalances, like hyperkalemia or metabolic acidosis.
    - **Dialysis**: Indicated in severe cases of intrinsic AKI with life-threatening complications (e.g., refractory hyperkalemia, volume overload).

Investigations for AKI and CKD

AKI Investigations:

- **Blood tests**: Serum creatinine, BUN, electrolytes, ABG.
- **Urine tests**: Urinalysis, FeNa, urine output.
- **Imaging**: Renal ultrasound, CT scan (with contrast if needed).
- **Kidney biopsy**: If the cause of AKI is unclear.

CKD Investigations:

- **Blood tests**: Serum creatinine, eGFR, electrolytes, BUN.
- **Urine tests**

Albert Syndrome is a rare, inherited genetic disorder that affects the **eyes**, **ears**, and **brain**. It is primarily characterized by **progressive vision loss** and **hearing impairment**, with some

associated neurological features. While not a widely recognized or commonly known condition, it is thought to be a **mildly progressive** syndrome.

However, to clarify, **Albert Syndrome** is very obscure, and there isn't a huge amount of readily available information on it in mainstream clinical resources. This could be because it may not be formally recognized in all classifications or may be included under other, more widely known syndromes with similar features. Sometimes, rarer syndromes or newly identified disorders are initially described by the last name of the person who first identified the condition, but those names don't always become widely adopted.

Key Features of Albert Syndrome:

The core features of Albert syndrome often involve:

1. **Visual impairment**:
    - Often includes **retinal degeneration**, a condition that leads to the gradual loss of vision.
    - May present in early childhood and progressively worsen with age.
2. **Hearing loss**:
    - A progressive **sensorineural hearing loss** is a common feature. This means that the problem lies in the inner ear or auditory nerve.
3. **Neurological problems** (may include):
    - **Cognitive decline** or other signs of **neurological involvement**.
    - Potential for problems with **coordination**, **balance**, or **movement** in some cases.
4. **Other symptoms**:
    - There can be **genetic factors** involved, and it may follow an **autosomal dominant inheritance** pattern (though this is not always the case).

Physical Examination of the Renal System

A **renal system examination** is an essential part of a comprehensive physical examination, particularly when evaluating a patient for kidney disease or related conditions (e.g., nephropathy, electrolyte imbalances, fluid retention). The exam can help identify signs of **acute kidney injury (AKI)**, **chronic kidney disease (CKD)**, **nephritis**, **urinary tract infections (UTIs)**, or other renal issues.

Key Areas of Focus in Renal Physical Examination:

1. **General Inspection:**
    - **General appearance**: Look for signs of **fluid retention** (e.g., edema, weight gain) or **malnutrition** (in cases of chronic renal failure).
    - **Vital signs**: Check **blood pressure** (hypertension is common in kidney disease), **respiratory rate**, **heart rate**, and **temperature**.

- **Edema**: Inspect for swelling, which may be seen in the **ankles**, **legs**, **feet**, or **face** (especially the eyelids). **Generalized edema** could suggest kidney or heart failure.
- **Signs of uremia**: Look for **pale skin**, **bruising**, or **yellowing** (jaundice) which could suggest kidney dysfunction (uremic toxicity).

2. **Inspection of the Abdomen:**
   - **Abdominal distension**: This could be a sign of **fluid retention** (e.g., ascites) or **hydronephrosis**.
   - **Scars**: Check for any previous surgical scars, such as those from **kidney transplant** or **nephrectomy** (kidney removal).
   - **Visible masses**: A mass in the lower abdomen or flank could suggest **polycystic kidney disease**, **tumors**, or an enlarged **hydronephrotic kidney**.
3. **Palpation of the Abdomen and Flanks:**
   - **Palpate for kidney enlargement**: Gently palpate both **flanks** (lower back, just below the rib cage) to check for any enlarged kidneys, which may indicate **polycystic kidney disease** or **hydronephrosis** (swelling of the kidney due to urine retention). Normally, the kidneys are not palpable unless they are enlarged or tender.
   - **Kidney tenderness**: Lightly palpate the area over the kidneys. **Flank tenderness** could indicate **pyelonephritis** (kidney infection), **renal stones**, or **renal infarction**.
   - **Costovertebral angle (CVA) tenderness**: Tap gently over the **CVA** (where the spine meets the ribs, at the lower back) to assess for kidney tenderness. **CVA tenderness** is a hallmark of **pyelonephritis** or other kidney infections.
   - **Palpate the bladder**: In cases of **urinary retention** or **bladder distention**, the bladder may be palpable above the pubic symphysis.
4. **Percussion:**
   - **Percuss the abdomen** for signs of **fluid accumulation** (ascites) or to identify a **distended bladder**.
   - **Percussion of the kidneys**: Gently percuss the kidneys (at the CVA) to assess for tenderness or any abnormal sounds, such as **renal enlargement** or **pain**.
   - Percussion may also help assess the **lower abdomen** for **distended bladder** (in cases of urinary retention).
5. **Auscultation:**
   - **Abdominal auscultation**: Listen for bowel sounds (which may be normal or diminished). In severe renal failure, there could be **peritoneal friction rubs** if there is peritonitis.
   - **Auscultate for bruits**: If there is suspicion of **renal artery stenosis**, listen with a stethoscope over the **abdomen** (just above the umbilicus) and the **flanks** for any **bruit**. This suggests turbulent blood flow due to narrowing of the renal arteries.

Key Signs of Renal System Disorders:

- **Edema**: Indicative of kidney failure or fluid retention (especially in **legs**, **ankles**, **face**, or **lungs** in cases of congestive heart failure).
- **Hypertension**: Elevated blood pressure is common in chronic kidney disease (CKD) and contributes to its progression.
- **Proteinuria**: May suggest **diabetic nephropathy**, **glomerulonephritis**, or **nephrotic syndrome**.
- **Hematuria**: Blood in the urine can be a sign of **glomerulonephritis**, **renal stones**, **infections**, or **trauma**.
- **Flank pain**: Pain in the back or sides could suggest **kidney infection (pyelonephritis)**, **stones**, or **hydronephrosis**.

#Note: The doctor talked about Diabetes type 1and 2 but we will discuss them in detail in the Endocrine system.

Done by your favourite colleagues :Mira'alnaser & Boshra Rbaihat