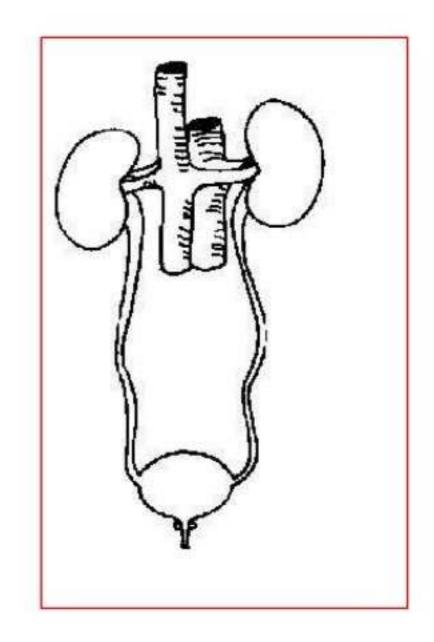
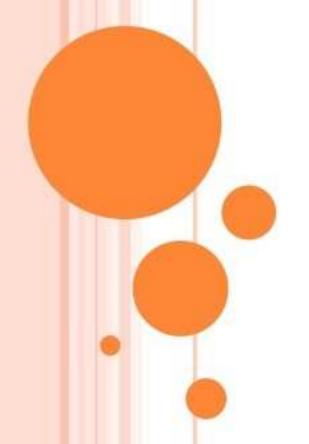
PRESENTATION ON ACUTE AND CHRONIC RENAL FAILURE





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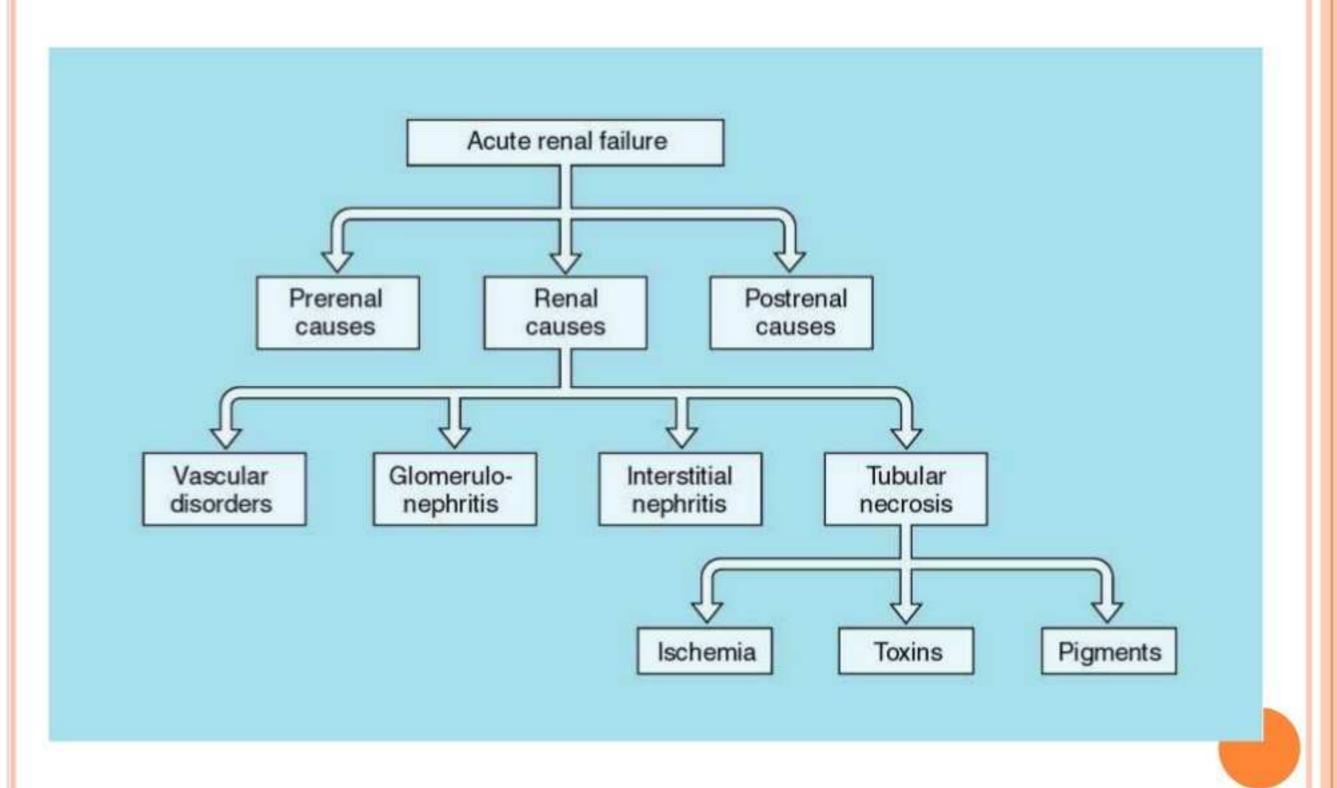
INTRODUCTION TO ACUTE RENAL FAILURE

- Acute kidney failure is the rapid loss your kidneys' ability to remove waste and help balance fluids and electrolytes in your body. In this case, rapid means less than 2 days. And it may progress to end stage renal disease, uremic syndrome, and death without treatment.
- ✓ In most cases ARF is usually asymptomatic and is diagnosed when screening of hospitalized patients reveals a recent increase in serum blood urea nitrogen and creatinine.

Recent Research's-

- The term acute renal failure (AKI) has largely replaced acute renal failure (ARF), reflecting the recognition that smaller decrements in kidney function that do not result in overt organ failure.
- The term ARF is now reserved for severe AKI, usually implying the need for renal replacement therapy.

ETIOLOGY OF ARF



1. PRERENAL CAUSES

- Any condition that significantly reduces renal perfusion pressure and causes a decreased glomerular filtration rate and azotemia* may cause prerenal kidney failure.
- Clinical conditions that may result in prerenal kidney failure include but are not limited to: extracellular fluid losses secondary to burns, prolonged vasoconstriction (hypertension), and reduced cardiac output as seen in patients with shock syndrome or congestive heart failure.
- If prerenal failure is identified early and treated correctly, the kidney dysfunction may be reversible.
- If the underlying cause continues to affect renal perfusion, it may lead to ischemic damage to the nephron and acute tubular necrosis (ATN).

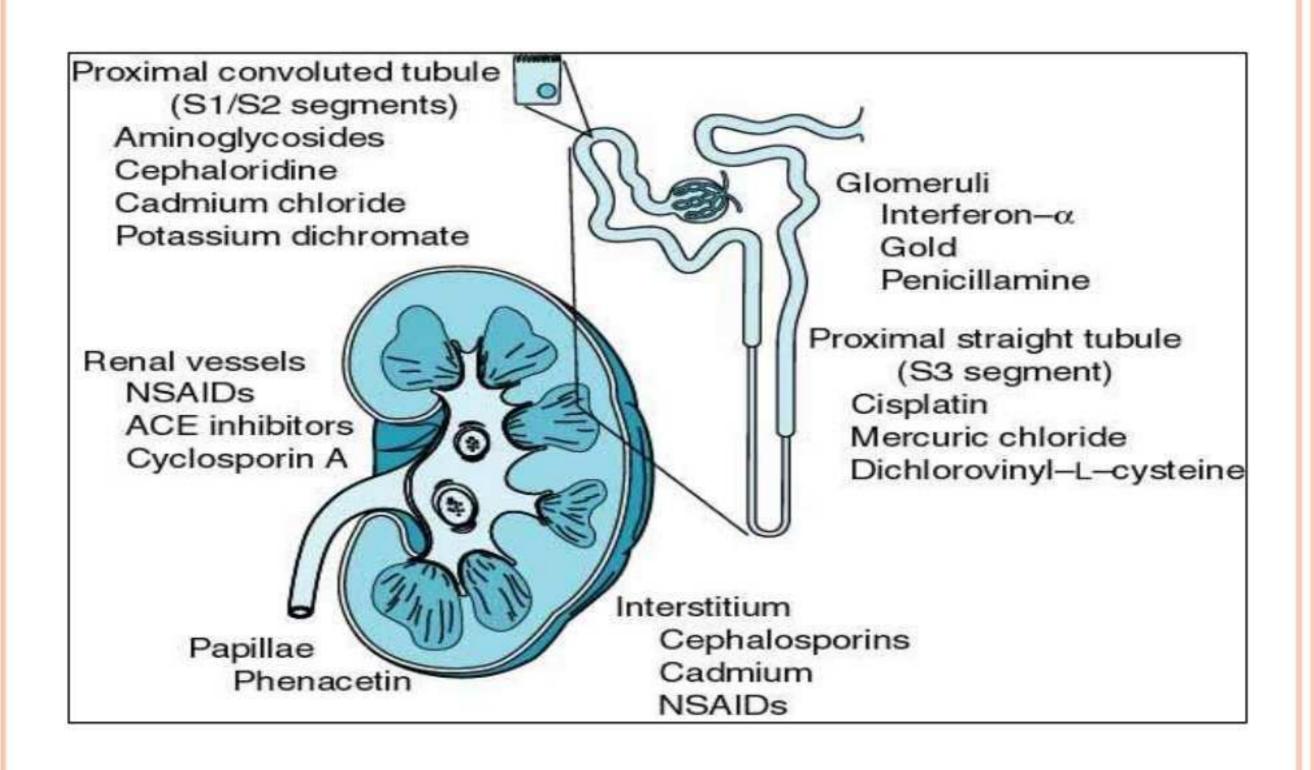
*Azotemia is a medical condition characterized by abnormally high levels of nitrogencontaining compounds (such as urea, creatinine, various body waste compounds, and other nitrogen-rich compounds) in the blood.

RENAL CAUSES

- Actual damage to the nephrons and renal parenchyma characterize intrarenal failure.
- Clinical conditions that result in intrarenal damage can be categorized under kidney disease or acute tubular necrosis.
- ATN is a common type of acute renal failure in the critically ill patient.
- The use of nephrotoxic drugs (streptomycin, penicillin, and amphotericin) in older patients or in individuals with underlying renal insufficiency place patients at a higher risk of developing ATN.
- The risk for ATN is also higher in patients with prolonged prerenal factors. ATN is a potentially reversible type of renal failure but it may take weeks or months before adequate renal function returns.

POST-RENAL CAUSES

- Post-renal failure is caused by clinical conditions that cause obstruction to urine flow.
- Any problem that stops the excretion of urine may cause this type of ARF.
- Common conditions associated with post-renal failure are tumors, benign prostatic hypertrophy, kidney stones and bladder neck obstruction.
- If post-renal failure is untreated it may result in actual nephron damage and intrarenal failure.



Nephrotoxicants may act at different sites in the kidney, resulting in altered renal function. The site of injury by selected nephrotoxicants are shown.



PATHOPHYSIOLOGY OF ACUTE RENAL FAILURE

The clinical course of ARF is characterized by the following three phases:

Phase 1. Onset

 ARF begins with the underlying clinical condition leading to tubular necrosis, for example hemorrhage, which reduces blood volume and renal perfusion. If adequate treatment is provided in this phase then the individual's prognosis is good.

Phase 2. Maintenance

- A persistent decrease in GFR and tubular necrosis characterizes this phase.
 Endothelial cell necrosis and sloughing lead to tubular obstruction and increased tubular permeability.
- Efficient elimination of metabolic waste, water, electrolytes, and acids from the body cannot be performed by the kidney during this phase. Therefore, azotemia, fluid retention, electrolyte imbalance and metabolic acidosis occurs.

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- Immune function is impaired and the patient may be anemic because of the suppressed erythropoietin secretion by the kidney and toxin-related shorter RBC life.

Phase 3. Recovery

• Renal function of the kidney improves quickly the first five to twenty-five days of this phase. It begins with the recovery of the GFR and tubular function to such an extent that Blood urea nitrogen level (BUN) and serum creatinine stabilize. Improvement in renal function may continue for up to a year as more and more nephrons regain function.

SYMPTOMS

- Bloody stools
- Breath odour and metallic taste in mouth
- Changes in mental status or mood
- Decreased appetite
- Decreased sensation, especially in the hands or feet
- Flank pain (between the ribs and hips)
- High blood pressure
- Nausea or vomiting, may last for days
- Nosebleeds
- Persistent hiccups
- Prolonged bleeding
- Seizures
- Shortness of breath
- Slow, sluggish movements
- Swelling due to the body keeping in fluid usually in the ankles, feet, and legs
- Urination changes:
 - Little or no urine
 - Excessive urination at night
 - Urination stops completely

DIAGNOSTIC TESTS

- H&P (History & Physical test)
- BUN, creatinine, sodium, potassium. pH, bicarbontae, Hgb (haemoglobin) and Hct (hematocrit).
- Urine studies
- US of kidneys
- KUB (Kidney, Ureters, Bladders radiography).
- Renal CT/MRI
- Retrograde pyelogram- Retrograde Pyelogram is a urologic procedure where the physician injects contrast into the ureter in order to visualize the ureter and kidney. The flow of contrast (up from the bladder to the kidney) is opposite the usual flow of urine, hence the retrograde name.

MANAGEMENT OF ARF

Medical treatment

- Fluid and dietary restrictions
- Maintain Electrolytes
- May need dialysis to jump start renal function
- May need to stimulate production of urine with IV fluids, Dopomine, diuretics, etc.
- Hemodialysis
- Peritoneal dialysis
- Continous renal replacement therapy (CRRT)
- Nursing interventions.

CONCLUSION FOR ARF

- Acute renal failure is common in hospitalised patients and is most often due to ATN. It is associated with a significant mortality and morbidity and is expensive to treat. Drugs play an important role in the pathogenesis of ARF.
- The pharmacist can play an important role in identifying possible pathogens, ensuring that the patient does not receive any further nephrotoxic compounds during the recovery phase.

CHRONIC RENAL FAILURE

Introduction-

- Chronic kidney disease (CKD), also known as chronic renal disease (CRD), is a progressive loss in renal function over a period of months or years.
- Chronic kidney disease is diagnosed as a result of screening of people known to be at risk of kidney problems, such as those with high blood pressure or diabetes and those with a blood relative with chronic kidney disease.
- It is differentiated from acute kidney disease in that the reduction in kidney function must be present for over 3 months.
- Chronic kidney disease is identified by a blood test for creatinine. Higher levels of creatinine indicate a lower glomerular filtration rate and as a result a decreased capability of the kidneys to excrete waste products.

ETIOLOGY OF CRF

Causes of chronic kidney disease (CKD) include the following:

- Diabetic kidney disease.
- Hypertension.
- Vascular disease (Angina & MI).
- Glomerular disease (primary or secondary).
- Cystic kidney diseases.
- Tubulointerstitial disease (nephritis affecting the interstitium of the kidneys)
- Urinary tract obstruction or dysfunction
- Recurrent kidney stone disease
- Congenital (birth) defects of the kidney or bladder
- Unrecovered acute kidney injury

SYMPTOMS

- Tiredness
- Weakness
- Not sleeping well
- Less desire to eat than usual
- Nausea
- Itching
- Shortness of breath
- Altered taste
- Altered mental state

STAGES

• All individuals with a glomerular filtration rate (GFR) <60 mL/min/1.73 m² for 3 months are classified as having chronic kidney disease, irrespective of the presence or absence of kidney damage.

Stage 1

• Slightly diminished function; kidney damage with normal or relatively high GFR (≥90 mL/min/1.73 m²). Kidney damage is defined as pathological abnormalities or markers of damage, including abnormalities in blood or urine test or imaging studies.

Stage 2

 Mild reduction in GFR (60–89 mL/min/1.73 m²) with kidney damage. Kidney damage is defined as pathological abnormalities or markers of damage, including abnormalities in blood or urine test or imaging studies.

Stage 3

Moderate reduction in GFR (30–59 mL/min/1.73 m²). British guidelines distinguish between stage 3A (GFR 45–59) and stage 3B (GFR 30–44) for purposes of screening and referral.

Stage 4

 Severe reduction in GFR (15–29 mL/min/1.73 m²) Preparation for renal replacement therapy.

Stage 5

 Established kidney failure (GFR <15 mL/min/1.73 m², permanent renal replacement therapy (RRT), or end stage renal disease (ESRD)

CKD Stage	GFR level (mL/min/1.73 m ²)
Stage 1	≥ 90
Stage 2	60 - 89
Stage 3	30 – 59
Stage 4	15 – 29
Stage 5	< 15

NDD-CKD vs. ESRD

- The term non-dialysis dependent CKD, also abbreviated as NDD-CKD, is a designation used to encompass the status of those persons with an established CKD who do not yet require the life-supporting treatments for renal failure known as renal replacement therapy (including maintenance dialysis or renal transplantation).
- The condition of individuals with CKD, who require either of the 2 types of renal replacement therapy (dialysis or transplantation), is referred to as the end-stage renal disease (ESRD).

TREATMENT

Although chronic kidney disease cannot be cured, it is possible to slow the damage to the kidney in most patients. Doctor may recommend any of the following:

- Controlling protein in the urine by restricting the amount of protein in the diet or medication.
- Taking ACE inhibitors or angiotensin II receptor antagonists to slow the progression to chronic renal failure.
- Reducing the use of and the dosages of drugs that may be toxic to the kidneys.
- Managing the complications of chronic renal disease such as fluid overload, high blood phosphate or potassium levels, low blood level of calcium, and anemia.

- Lowering high blood pressure.
- Controlling blood sugar and lipid levels.
- Staying hydrated.
- Controlling salt in the diet.
- Quitting smoking.
- Undergoing dialysis, a medical process that cleans the blood.
- Having a kidney transplant.
- Counseling for you and your family about dialysis and/or transplant options.

PREVENTION

To help reduce your chance of chronic kidney failure, take the following steps:

- Get a physical exam every year that includes a urine test to monitor your kidney's health.
- Do not smoke. Stop smoking if you are a smoker.
- Maintain a healthy weight.
- Drink water and other fluids to stay hydrated.
- People who have diabetes, previously known kidney disease, high blood pressure, or are over the age of 60 should be screened regularly for kidney disease.
- People with a family history of kidney disease should also be screened regularly.

DISTINGUISHING ACUTE FROM CHRONIC RENAL IMPAIRMENT

- Comparing any previous measurements of serum creatinine with the patient's current biochemistry. Pre-existing chronic renal impairment can be excluded if a relatively recent previous measurement of renal function was normal.
- A history of several months' vague ill-health, nocturia or pruritus, and findings of skin pigmentation, anaemia, long-standing hypertension or neuropathy suggest a more chronic disease.
- Renal ultrasonography to determine size and echogenicity of the kidneys. It is noteworthy that renal size is normal in most patients with ARF.
- Anaemia is a major feature of CRF, but it may occur early in the course of many diseases that cause ARF.
- Bone disease evidence of long-standing renal bone disease (e.g. radiological evidence of hyperparathyroidism, greatly elevated parathyroid hormone (PTH) levels) is diagnostic of CRF, but hypocalcaemia and hyperphosphataemia may occur in both ARF and CRF.

