

INTRODUCTION.

Kidney failure :

- also known as renal failure is the partial or complete impairment of kidney function.
- It result in an inability to excrete metabolic waste products and water, and it contributes to disturbances of all body system.

UREMIC SYNDROME AND RENAL FAILURE:

- are used synonymously. The term uremic syndrome describes a set of manifestations that result from loss of renal function.

ACUTE RENAL FAILURE

- Acute kidney injury is a syndrome characterized by-
- Sudden decline in GFR(hours to day)
- Retention of nitrogenous wastes product in blood (Azotemia).
- Disturbance in extracellular fluid volume
- Disturbance in electrolyte and acid base homeostasis.



ACUTE RENAL FAILURE

- Sudden decrease in function (hrs-days)
- Often multifactorial.
- Pre-Renal and intrinsic renal cause-70%
- Oliguric, UOP-<400ml
- Associated with high mortality and morbidity



PRESENTATION.

- Often no sign or symptom
- Oliguria
- At risk group
- Patient with chronic renal disease, Diabetes, hypertension.
- Increased creatinine and urea.



ASSESSMENT.

- ARF vs CRF
- CRF more likely if Acute illness , long duration, of symptom, intra-abdominal mass, palpable bladder.
- If patient euvolaemic?
- Renal Parenchymal disease?
- Urine dipstick and microscopy
- Major vessel occlusion?



CAUSE

- Pre-renal 40-70 %
- Renal 10- 50 %
- Post- renal 10 %.

DIFFERENCE BETWEEN ACUTE AND CHRONIC RENAL FAILURE.

ACUTE RENAL FAILURE	CHRONIC RENAL FAILURE
1.ONSET-OVER DAYS TO WEEKS	OVER WEEK TO MONTH
2.INVARIABLY REVERSIBLE	USUALLY IRREVERSIBLE
3.CAUSE-PRE-RENAL OR RENAL	MOSTLY RENAL
4.URINARY VOLUME OLIGURIC AND ANURIA	POLYUREA AND NOCTURIA
5.RENAL FAILURE CAST -ABSENT	PRESENT
6.SPECIFIC gravity--HIGH	LOW AND FIXED.
7.DIALYSIS REQUIRED FOR SHORT PERIOD	REPEATEDLY.
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PRERENAL ARF.

- Cause of pre- Renal—Due to reduced blood delivery to the kidney.

Volume depletion-

hemorrhage

dehydration

GI fluid loss

Decrease effective circulatory blood volume

Hypotension.) Decrease cardiac output(CHF,MI,

Liver failure

Functional ‘

ACEIs, NSAID,ARBs

Correction of volume depletion can restore kidney function.

CAUSE OF POSTRENAL ARF.

- Due to obstruction of urinary outflow.

- Bladder outlet obstruction

Benign prostatic hypertrophy

Prostate cancer

Ureteral obstruction

Malignancy

Pelvis or Renal obstruction

Rapid resolution of Post-renal ARF without structural damage restore kidney function.



INTRINSIC RENAL DISEASES

Due to damage is within the kidney(structure of the nephron)

CAUSES—

Vascular damage(Renal thrombosis)

Glomerular damage(Nephrotic/Nephritic, Glomerulonephritis)

Acute tubular necrosis(ATN)- Account for 50% of all cases of ARF.

Ischemia(Hypotension)

Endogenous toxin(Hb, uric acid)

Exogenous toxin(Aminoglycosides, contrast induced nephropathy)

Acute interstitial nephritis

NSAID, Infection



Pre renal ARF can progress to intrinsic ARF if the the condition is not corrected.

CLINICAL FEATURES ACUTE RENAL FAILURE(ARF).

- *It depends upon the cause of ARF and the stage of the disease at which the patient presents.*
- *It includes one of these 3 major pattern-*
- **Syndrome of Acute nephritis**
- ***Syndrome accompanying tubular pathology***
 - **-Oliguric phase**
 - **-Diuretic phase**
 - **-Recovery phase**
- **Pre- Renal syndrome**



SYNDROME OF ACUTE NEPHRITIS

- *This is associated with acute glomerulonephritis (Inflammation of glomerulus)*
- *Results in increase glomerular permeability and decrease in glomerular filtration rate*
- *Features–*
 - mild proteinurea*
 - Haematuria*
 - oedema*



SYNDROME ACCOMPANYING PATHOLOGY

- ARF is caused by destruction of the tubule cells of nephrons—
- Disease progress in three stages—
- Oliguric phase—
 - Last for 7-10 days with urine output less 400ml/day.
- Accumulation of waste product of protein metabolism in the blood and resultant.
- Metabolic acidosis
- Azotaemia
- Hyperkalaemia
- Hyponatremia.
- Hypervolaemia

- *Diuretic phase—*
- Healing of tubules results in improving the urinary output
- Effect—
- *Dehydration and electrolyte imbalance.*
- *Recovery phase—*
- Full recovery of tubule cells occurs in half cases
- Time period—upto one year.



- Pre- Renal syndrome
- Because of secondary disorders like ischaemia(Decreased blood flow to tissue) and not due to glomerular or tubular damage.
- Cause of ischaemia—
 - Renal arterial obstruction
 - hypovolaemia
 - hypotension
- Due to decrease renal flow, there is decrease in GFR causing oliguria, azotemia(elevation of BUN and creatinine) and oedema.



SIGN AND SYMPTOMS OF ACUTE RENAL FAILURE

- Decreased urine output—Anuria / Oliguria.
- Acidic breathing.
- Electrolyte imbalance—Hyperkalemia.
- Nausea, vomiting -----Dehydration.
- Hypertension
- Hematuria
- Ascitis
- Pale skin
- Poor appetite



DIAGNOSIS OF ACUTE RENAL FAILURE

- *Routine laboratory test*
- *(Creatinine and blood urea nitrogen)*
- *Ultrasound of kidney - Ultrasonography helps to see the presence of two kidney, for evaluating kidney size and shape, and for detecting hydronephrosis or hydroureter, renal calculi and renal vein thrombosis.*
- *Kidney biopsy.*



ACUTE RENAL FAILURE DIAGNOSTIC.

○ Laboratory Evaluation

- Blood urea and creatinine - Both are raised due to diminished renal blood flow.

○ URINE ROUTINE EXAMINATION

- If Glomerulonephritis—

- Hematuria
- Proteinuria
- RBC cast.

- Serum Creatinine reliable marker of GFR

- BUN- generally follows Serum Creatinine

- **BUN/ Creatinine** -- helpful in classifying cause of ARF—

- **Ratio > 20 : 1--** suggest pre-renal cause

- **Ratio 10–15 : 1--** suggest intrinsic renal cause.



LAB. INVESTIGATION

- **BLOOD EXAM :**

- Anemia
- Leucopenia
- Thrombocytopenia

- **SERUM ELECTROLYTES:-**

- *HYPONATREMIA*
- *HYPERKALEMIA*
- *HYPOCALCEMIA*
- *HYPERPHOSPHATEMIA*
- *METABOLIC ACIDOSIS.*

ACUTE RENAL FAILURE DIAGNOSTIC

- Urinolysis
- *Unremarkable in pre- renal and post-renal cause.*
- *Differentiates ATN vs AIN vs AGN.*
- *Muddy brown cast in ATN*
- *WBC cast in AIN .*
- *RBC cast in AGN.*



INVESTIGATION.

- *RBCs and RBC cast in Glomerular disease.*
- *Crystals , RBC and WBC in post-renal ARF.*
- *HB % ↓-- Haemolysis, GI bleeding*
- *↑/↓ Total Leucocyte count---Infection.*
- *LFT: ↓Albumin imply proteinurea—GN.*
- *Elevated Blood urea, Serum creatinine ratio indicate Pre-renal ARF.*
- *↓HCO₃: Metabolic acidosis.*



OTHER BIOMARKER.

- *Cystatin C*
- *Neutrophil gelatinase- associated lipocalin(NGAL).*
- *Interleukin*
- *Kidney injury molecule-1*
- *N-acetyl-D-glucosaminidase.*



CYSTATIN C

- *Superior to serum creatinine, as a surrogate marker of early and subtle changes of kidney function.*
- *It identifies kidney injury while creatinine levels remain the normal range.*
- *Allows detection of AKI, 24-48 hours earlier than serum creatinine.*



KIDNEY INJURY MOLECULE-1

- *KIM-1 is a type 1 trans membrane glycoprotein.*
- *Served as a marker of severity of AKI.*
- *NEUTROPHIL gelatinase- associated lipocalin(NGAL).*
- *NGAL can be detected in the plasma and urine within 2 hrs of cardiopulmonary bypass-associated AKI.*
- *Considered equivalent to troponin in acute coronary syndrome.*



ACUTE RENAL FAILURE
DIAGNOSTIC

TEST	FAVOURS PRERENAL DISEASE	FAVOURS ATN
BUN/CREATININE RATIO	>20:1	10-15:1
Uosm	>500mosol/kg	<350 mosol/kg
Una	<20 meq/L	>40 meq/L
FEna	<1 percent	>2 percent

