ACUTE KIDNEY INJURY(AKI) Previously known as Acute Renal Failure(ARF)

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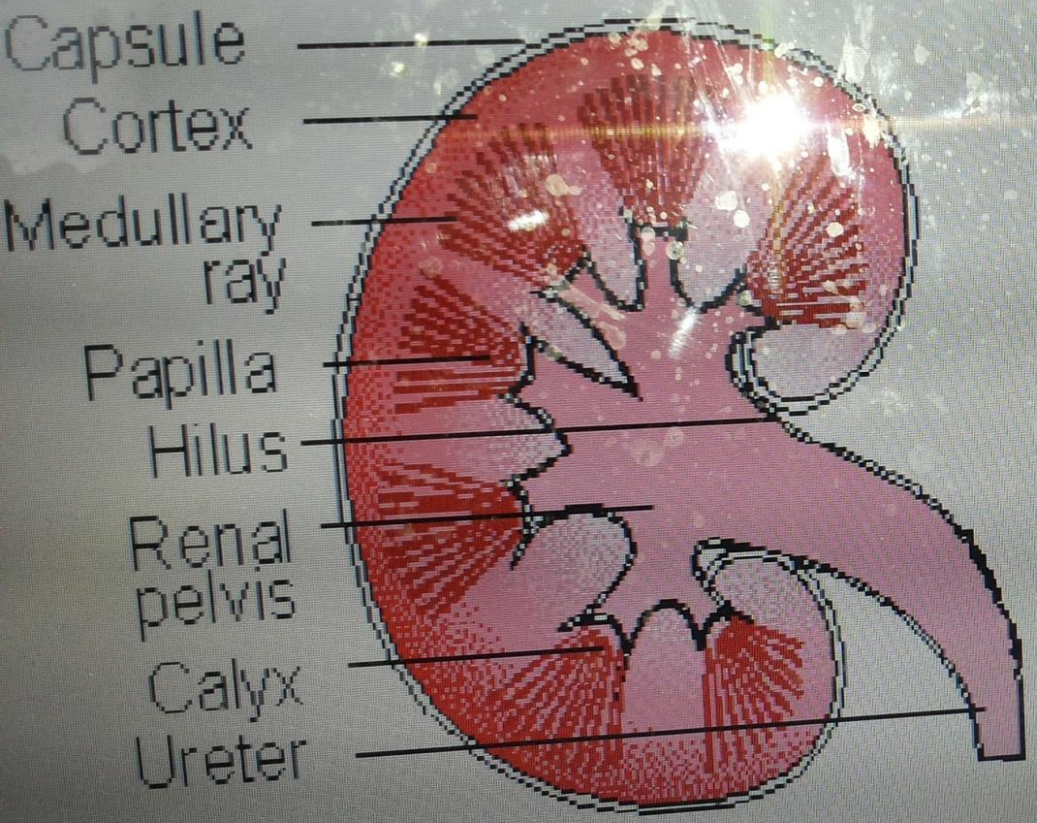
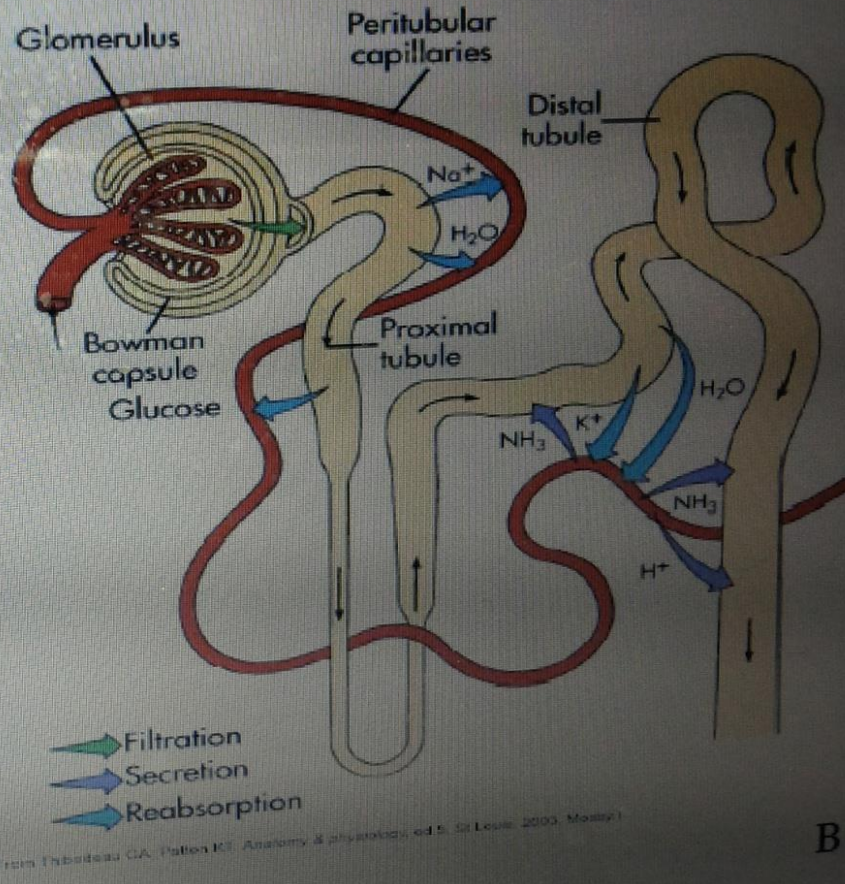
Fig 1: a. Cross section of the kidney b. The nephron (functional unit of the kidney)

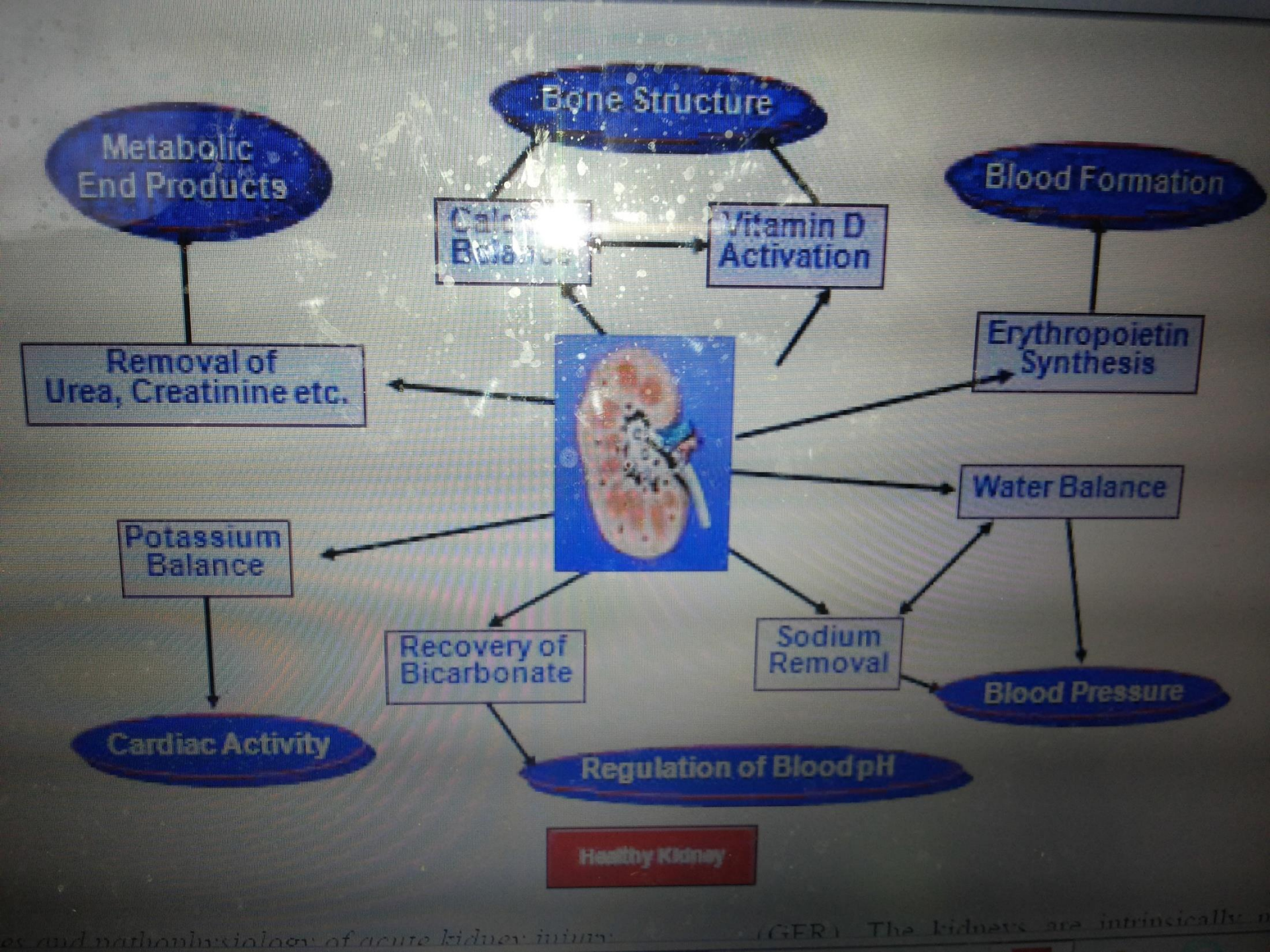


A



B

Fig 2: Summary of the physiologic functions of the skin


Bone Structure
Metabolic End Products
Blood Formation
Vitamin D Activation
Removal of Urea, Creatinine etc.
Erythropoietin Synthesis
Water Balance
Potassium Balance
Recovery of Bicarbonate
Sodium Removal
Blood Pressure
Cardiac Activity
Regulation of Blood pH
Healthy Kidney

AKI

DEFINTION:-It is a clinicolaboratory syndrome occurs due to sudden and significant reduction in renal function manifested by:-

a)Oliguria (UOP<1ml/kg/hr) or anuria.

b)Disturbance in fluid, electrolytes &Acid-base status.

c)Retention of waste products like urea,creatinin, etc

AKI may be of two forms(Oliguric or Nonoliguric).

Early recognition & management is crucial.

AKI

CAUSES:-Major causes are:-

1)PRERENALcause which lead to reduction in the renal perfusion which lead to decrease GFR and this may result from:-

a)Hypovolemia Vomiting&Diarrhea(G.E)

Hemorrhage

Burns

AKI

b)Hypotention

Septicemia.

Hypothermia.

Bleeding.

Heart Failure.

Hypoalbuminemia(Nephrosis)

c)Hypoxia

Extensive pneumonia.

Respiratory depression(central).

AKI

2)Renal causes which include:-

a)Glomerulonephritis;

PSAGN

SLE nephritis

MPGN

RPGN

b)Intravascular coagulation;

Hemolytic uremic syndrome(most common cause Of AKI in toddlers).

Renal vein thrombosis.

AKI

c)Acute Tubular Necrosis(ATN)

Heavy metals(Mercury&Gold).

Drugs(Aminoglycosides&Diuretics).

Hemoglobinuria(Hemolysis).

Myoglobinuria(Crush injuries).

Shock.

AKI

d)Acute Interstitial Nephritis(AIN);
Infections(Viral or Bacterial)
Drugs(antibiotics like penicilin,cephalosporin and Nonsteroidal antiinflamatory agents like Indomethacin or ibobrufein).

e)Tumours(Renal parenchymal infiltration, tumour lysis syndrom,uric acid nephropathy.

AKI

3)Postrenal causes:- These include;

a)Structural obstruction,like

- pelviureteric junction(PUJ)obstruction.
- Posterior uretheral valve(PUV).
- Tumours.

b)Acquired obstruction; Stones&Blood clots.

c)Functional obstruction(Vesicoureteric reflux).

AKI

Acute Tubular Necrosis(ATN) is the most common cause of AKI in children which result from renal under perfusion.

Sever vascular compromise may lead to arterial or venous thrombosis with Acute Cortical Necrosis(ACN)

ATN may be reversible.

AKI

CLINICAL FEATURES:-

a)Patients may present with pallor (anemia) oliguria,odema(salt&water retention)vomiting lethergy,high B.P&uremic encephalopathy.

b)signs&symptomsof the underlying disease will be present.

AKI

- Vomiting&diarrhea in a 6 months old infant who is dehydrated&oliguric may indicate prerenal renal failure.

- A 6Yrs old child presented with hematuria periorbital odema&high B.P and sometimes fluid oveload may indicate AGN as a cause of AKI

AKI

- A newborn baby with prenatal history of hydronephrosis on ultrasound scanning may indicate an obstruction.

- A child with rash and arthritis may suggest diagnosis of SLE or H.S PURPURA.

- A palpable renal mass may indicate RVT or obstructive uropathy.

AKI

LABORATORY FINDINGS:-

1)Blood investigations:

a)CBC;Hb may be low(dilutional/hemolytic anemia).

TLC&platelets may be low(SLE).

Eosinophlia may be seen(interstitial nephritis)

2)Urea,creatinin&uric acid are high.

3)Sodium&calcium are low;potassium and phosphate are high.

4)Blood gases show metabolic acidosis.

AKI

5)Specific investigations according to the cause.

- Complement(C3),low in SLE,PSAGN
- Raised ASO titre as in PSAGN.
- ANA&Anti DNA Abs in SLE.
- Coagulation profile in RVT.

AKI

2)Urine analysis may show hematuria and/or proteinuria and the presence of RBC and granular casts suggest intrinsic renal failure.

wbc&wbc casts suggest tubulointerstitial disease
Eosinophiluria may be seen in interstitial nephritis

Early diagnosis by estimating certain biomarkers in urine(NGAL&CYSTATIN-C).

AKI

	PRERENAL	RENAL
Sp.gravity	raised(>1.010)	<1.010
Osmolality	>750mosm/kg	<350mosm/kg
Urine Na	<20meq/L	>20meq/L
Fe Na	<1%	>2%
urea/creat.ratio	>20	<20

AKI

3)Radiological investigations :-

Chest x-ray may show cardiomegaly and pulmonary congesion(fluid overload).

Abdominal ultrasound may show increased echogenesity(Nephritis)or hydronephrosis as in obstructive uropathy.

4)Renal biopsy in selected cases.

AKI

TREATMENT:-Admit the patient to ICU and observe for vital signs,urine output&register his weight.

A) Treatment of hypovolemia(prerenal cause) which is concerned with volume replacement to avoid progression to acute tubular necrosis.

Normal saline as 10-20 ml/kg given over 30-60 minutes
Followed by this patient should void within two hours.
If patient still dehydrated give more fluids.

If patient is adequately hydrated start diuretic therapy,where furosemide(loop Diuretic)to be given as 2mg/kg I.V&slowly.

If still no urine,give another(higher)doseof diuretic,provided the patient is well hydrated.

If still

AKI

If patient still not passed urine,it indicate that he is in intrinsic renal failure.

At this stage fluid should be restricted to insensible loss+UOP+other losses.

B)food,fluid&medication containing potasium should be restricted until the renal function is reestablished and serum potassium(K+)return to normal or dialysis was initiated.

AKI

The major risk of hyperkalemia is cardiac arrythmias,so an ECG record is needed to Identify these changes.

Treatment of hyperkalemia include;
I.V Sodium bicarbonate,I.V calcium gluconat glucose infusion with insulin&dialysis.

AKI

C)Treatment of metabolic acidosis which may be mild,moderate or sever depending on Blood gas result.

Treatment with Na HCO3 solution which should be given with caution(Risk of fluid overload,hypertension&convulsion).

AKI

D)Treatment of hypocalcemia&hyperphosphatemia

1)Dietary phosphorus restriction.
2)Phosphate binders like Calcium acetate or calcium carbonate is given with food.

Symptomatic hypocalcemia is treated with calcium gluconate (0.5-1cc/kg I.V slowly).

AKI

E)Treatment of hypertension:-

1)Fluid restriction&Diuretics(Furosemide).

2)Vasodialators(Hydralazine)&Ca+ channel blocker agents (nifidipen)may be used.

3)Labetalol may be used in hypertensive encephalopathy.

4)Dialysis is indicated in resistant cases.

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F)Treatment of infection, if present with appropriate and nonnephrotoxic antibiotics.

AKI

G)Dialysis is indicated in the following :-

1)Fluid overload not responding to diuretic and fluid restriction(CCF&pulmonary odema)
2)Sever(resistant)hyprkalemia(s.k+>7meq/L)
3)Sever(resistant)metabolc acidosis(ph<7.1)
4)Signs of uremic encephalopathy.
5)GIT Bleeding

AKI

Types of dialysis(renal replacement therapy) include hemodialysis(HD),peritoneal dialysis (PD)&Continuous renal replacment therapy modalities(hemdialysis,hemofiltration and hemodiafiltration) by the use of multifiltrate machine.

AKI

PROGNOSIS,Recovery is depending on the cause and availability of specific treatment for the cause.

Sepsis is the major proplem in patients with acute kidney injurey