Acute Renal Failure & Malaria

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Definitions

• Inability of kidney to maintain homeostasis leading to a buildup of nitrogenous wastes

• Different to renal insufficiency where kidney function is deranged but can still support life

Definitions

- Occurs over hours/days
- Lab definition
 - Increase in baseline creatinine of more than 50%
 - Decrease in creatinine clearance of more than 50%
 - Deterioration in renal function requiring dialysis

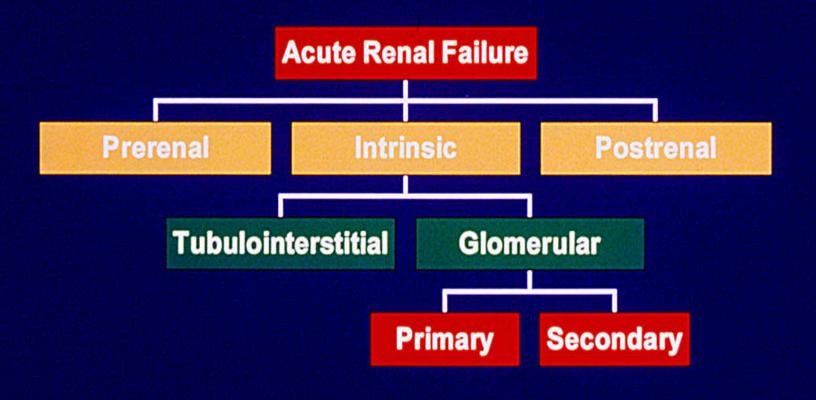
Classification of ARF

• Pre renal (functional)

• Renal-intrinsic (structural)

• Post renal (obstruction)

Work-up of Acute Renal Failure



Causes of ARF

• Pre-renal:

Inadequate perfusion

- check volume status
- Renal:

ARF despite perfusion & excretion

- check urinalysis, FBC & autoimmune screen
- Post-renal:

Blocked outflow

— check bladder, catheter & ultrasound

Causes of ARF

Pre-renal	Renal	Post-renal
Absolute	Glomerular	Pelvi-calyceal
hypovolaemia	(RPGN)	
Relative	Tubular	Ureteric
hypovolaemia	(ATN)	
Reduced	Interstitial	VUJ-bladder
cardiac output	(AIN)	
Reno-vascular	Vascular	Bladder neck-
occlusion	(atheroemboli)	urethra

Clinical Presentation

- Anuria: No UOP
- Oliguria: UOP<400-500 mL/d
- Azotemia: Incr Cr, BUN
 - May be prerenal, renal, postrenal
 - Does not require any clinical findings

Clinical Signs & Symptoms

- Hyperkalemia
- Nausea/Vomiting
- HTN
- Pulmonary edema
- Ascites
- Asterixis
- Encephalopathy

Laboratory Diagnosis

- UEC
- Acid-Base balance
- Urine analysis
- BUN
- LFT
- FBC & blood slide
- Others USS (?obstruction).

Malaria & ARF

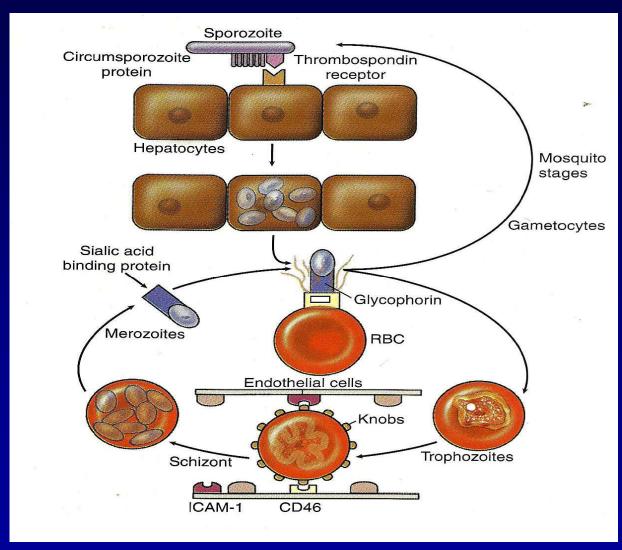
- Malaria is one of top 10 killer diseases in world
- ARF occurs in <1% of pf malaria, but mortality up to 45%
- Common in adults than children, recent trends- high incidence
- Diagnosed when sr. creat.>3mg/dl or urine output <400ml/24 hrs
- Renal involvement varies from mild proteinuria to severe azotemia
- Malarial ARF is associated with cerebral malaria, Jaundice, Anaemia, ARDS/Pulm. edema & Hypoglycaemia

- *In mild cases* not much change in renal parenchyma- may be minimal tubular degeneration, mild renal parenchymal change & presence of vacuoles
- *In severe cases* Tubular degeneration with distal tubular necrosis, Proximal tubules are often loaded with malarial pigments, Hb granules may be seen in the tubular cells

- Most patients have little or no proteinuria & urinary sediment contains occasional granular and hyaline cast but no RBC.
- Absence of hypertension, Rapid resolution without residual impairment & predominant in adults rather than children with urinary findings suggests- ARF results from ATN & not glomerulonephritis

- ARF- mediated thro' several mechanisms
- 1.Effect of pRBC on microcirculation- knob like processes formation on surface of RBC which helps in anchoring to the endothelium
- Cytoadherence- due to thrombospondin formation from vascular endothelium- specific to pf (not in pv/pm) so ARF only in pf.
- Loss of deformability of pRBC according to need of microcirculation- slugish circulation- renal ischemia

Mechanism of cytoadherence



Ref: Robins Pathological Basis of Diseases, 6th Ed.

2. Hypovolumia may occur due to Fever (hyperpyrexia), sweating, decreased intake of fluid, vomiting etc.

3.DIC

- 4.Increased plasma viscosity due to infection
- 5.Release of chemical mediators- TNF, cachectin, cytokines, interleukines etc causes- vasoconstriction, increased vascular permiability, catecholamine release(SIADH) hemoconcentrarion, shock & tubular necrosis
- 6. Hyperbilirubinaemia due to hemolysis, Black water fever in G6 PD deficiency patients is also associated with ARF

Laboratory Diagnosis & Monitoring

- Blood slide
- As in ARF (BUN, etc..)
- Bilirubin
- Urine output monitoring

End

Main Reference: Robins Pathological Basis of Diseases, 6th Ed. Chapter on renal failure and infectious diseases.

Others (www): Deb Goldstein teaching slides, 2005.

Cherelle Fitzclarence teaching slides, 2010

Dr Saroj K Mishra & Dr Kishore C Mahanta, teaching slides, India.

Download seminar notes on:

www.pathologyatsmhs.wordpress.com

File in PDF and PPT format

Feedback

What was presented well and you understood concepts?

What was not presented well and not understood well?

How can seminar be improved?

Go to www.pathologyatsmhs.wordpress.com and leave feedback comments