Parasitic Infections Causing Urinary Tract Disorders

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The Predominant Parasitic Diseases

1. Malaria
2. Filariasis
3. Schistosomiasis
4. Echinococcus
Malaria

- Only *P. falciparum* and *P. malariae* are clearly associated with renal disorders
- Occurs only in small percentage of patients
Kidney Disorders in Malaria

1. Glomerululopathy
2. Nephrotic syndrome
3. Acute Renal Failure
4. Black Water Fever
Kidney Disorders in Malaria

Glomerulopathy

- As a result of endothelial damage dan immune complex deposition → circulating immune complex can be detected, deposition of Ig around glomeruli can be seen histologically

- Endothelial damage as a result of renal microvascular disturbance (due to intravascular coagulation) and local inflammatory response
Kidney Disorders in Malaria

Nephrotic syndrome

- Aetiology: mostly due to chronic infection of *P. malariae* (quartan malaria)
- Histopathology: membranoproliferative glomerulonephritis (focal/segmental/global glomerulosclerosis)
- Immunology: granular deposits of IgM, IgG and C3 in mesangial and subendothelial
Kidney Disorders in Malaria

Acute Renal Failure
- One of severe manifestations in falciparum malaria
- Histopathology: tubulointerstitial damage (tubular necrosis, interstitial edema, cellular cast & hemoglobin in tubuli)
Acute Renal Failure

Pathogenesis:
- Infected RBC $\rightarrow$ increased rigidity and adhesiveness $\rightarrow$ intravascular coagulation $\rightarrow$ impaired blood flow in the microcirculation
- Hemolysis
- Hypovolemia
Black Water Fever
- Fever + dark urine (hemoglobinuria)
- Parasites is normally absent or hardly found
- History of taking inadequate dosage of aminoquinoline drugs (quinine, mefloquine)
Schistosomiasis

- Aetiology: trematodes from the genus Schistosoma.
- 4 main species infect humans:
  - *S. mansoni*
  - *S. japonicum* (Intestinal schistosomiasis)
  - *S. mekongi*
  - *S. haematobium* causes urinary schistosomiasis
Sporocysts in snail (successive generations)

1. Eggs hatch releasing miracidia

2. Miracidia penetrate snail tissue

3. Scolex of S. mansonii

4. S. mansonii

5. Cercariae released by snail into water and free-swimming

6. Penetrate skin

7. Cercariae lose tails during penetration and become schistosomulae

8. Circulation

9. Migrate to portal blood in liver and mature into adults

10. Paired adult worms migrate to:
   - Mesenteric venules of bowel/rectum (laying eggs that circulate to the liver and shed in stools)
   - Venous plexus of bladder

S. japonicum

S. haematothobium

http://www.dpd.cdc.gov/dpdx
Urinary Schistosomiasis

- Has been infecting humans for at least 4000 years (specific hieroglyph in ancient Egyptian). So prevalent in Egypt that boys were expected to go through a “male menarche”—sometime during adolescence, it was normal for them to urinate blood.
- *S. haematobium* infections continue to be a significant public health problem in Africa & the Middle East, second to malaria among parasitic diseases.

- Incidence men : women = 9:1 in regions where men are primarily freshwater fishermen or farmers using irrigation
Urinary Schistosomiasis

- Higher incidence in young boys and women where women fetch water for household use & young boys often play in or near water → rate of transmission depends on cultural practices.

- Age: usually occurs in individuals < 30 years
Urinary Schistosomiasis

- typically affects the bladder, ureteral involvement ± 30-65% of patients
- the commonest cause of haematuria worldwide
- May happen in children (an 8 year old boy with macroscopic, end stream haematuria)
- Often chronic
Urinary Schistosomiasis

**Pathogenesis**

1. Fluke migrates to perivesical venous plexus
2. Attach to the walls of the venous plexus
3. The females deposit eggs
4. Penetrate into the lumen
5. Encapsulated in vesical tissue
   - Inflammatory granulomatous rx
   - Fibrosis
   - Calcification of the dead ova
6. Calcification spreads around the bladder wall
Urinary Schistosomiasis

Pathogenesis

- The earliest calcification occurs 50-120 days after deposition. The female fluke can produce up to 3500 eggs/day.
- The degree of calcification correlates to the number of eggs deposited.
- A calcified bladder has ± 0.5 - 1 million eggs/cm. The extent of calcification correlates to the number of eggs in the bladder lumen.
- The bladder wall becomes fibrotic but still distensible and maintains a normal capacity.
1. Ureteral strictures can be found
2. Beading of the lower ureteral segment as disease progresses.
3. Subsequent ureteral fibrosis leads to calcifications of the distal ureter → X ray: characteristic pattern of linear or parallel calcifications
4. 80% of the strictures occur in the bladder wall near the junction with the ureters.
Pathology in Ureter

5. Ureter dilatation due to:
   - vesicoureteric reflux
   - stenosis of the ureter
   - edematous ureteral wall → causes deficient peristalsis
   - ureters commonly have persistent filling in the lower segment
Mortality & Morbidity
WHO 2004

- 180 million people live in endemic areas
- 90 million are infected; most of these live in Sub-Saharan Africa.
- 70 million persons suffer from schistosomal hematuria
- 18 million from associated bladder wall pathology
- 10 million from hydronephrosis
- 150,000 people die each year from renal failure + bladder cancers → Overall mortality rate ± 2/1,000 infected patients/year
Complications:

- increased incidence of squamous cell carcinoma of the bladder
- Urolithiasis
- Ascending UTI
- Urethral & ureteral stricture → hydronephrosis, renal failure.
Clinical Manifestations

- a chronic low-grade infection with flu like symptoms.
- Fatigue, a lack of energy
- Headache, a stiff neck
- neurologic symptoms due to central nervous system (CNS) complications.
- microscopic or gross hematuria
- dysuria, urinary frequency, urinary urgency
Lymphatic Filariasis

• Organ affected: lymphatic system
• 3 species cause: *Wuchereria bancrofti*, *Brugia malayi*, *Brugia timori*
• Transmitted by mosquito
• Function of lymphatic system:
  – Keeping in balance of the body’s fluid
  – Fighting bacteria that cause infections
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Lymphatic Filariasis

- Endemic in 80 countries
  - Estimated 1.1 billion people at risk.
  - Approximately 120 million people infected.
    - 90% *W. bancrofti*
    - 10% *Brugia malayi* + *Brugia timori*
Distribution in Indonesia

- *Wuchereria bancrofti*
- *Brugia malayi*  
  - Most widely distributed in Indonesia
- *Brugia timori*  
  - Only eastern parts of Indonesia
• Adult worms
  - Live in the lumen of lymphatic vessels
  - Life span 8 – 10 years
• Microfilariae
  - Present in the peripheral blood at certain time (periodicity)
  - Life span 1 year
• Larva
  - L3 larvae is the infective stage
  - Enter human host via mosquito’s biting
  - Larval development 10 – 14 days
Kidney Disorders in Filaria

The Microfilaraemic stage

- Clinically: most are asymptomatic
- ± 40% microfilaraemic individuals have haematuria and/ proteinuria → low grade renal damage
- Haematuria is associated with the presence of microfilariae → can be reversal
Kidney Disorders in Filaria

Chronic manifestations:

- Hydrocele
- Lymph scrotum: \{ only in *W. bancrofti* \}
- Chyluria
- Elephantiasis
- Lymphedema
Clinical Signs and Symptoms

- Hydrocele: accumulation of fluid in the tunica vaginalis
- Chyluria: rupture of dilated lymphatic vessels into urinary tract → milky urine
- Lymph scrotum: rupture of superficial dilated lymph vessels of the scrotal skin → intermittent discharge of lymph fluid
Chyluria

- Very rare cases
- Patient’s complain: passing milky urine
- Mostly associated also with haematuria
- Cause: rupture of lymphatic vessels mostly at pelvis level of the kidney
- May have spontaneous regression of the symptoms
- Should be differentiated from pyuria and phosphaturia
- Urin examination: emulsion, lymphocytes +
Echinococcus granulosus

Morphology

- Length 3-6 mm
- Scolex: 4 suckers, rostellum with hooks
- Neck
- Segments:
  - 1 immature
  - 1 mature
  - 1 gravid
Echinococcus granulosus
life cycle:

• Definitive host
  - Dogs and other carnivores
  - Adult worm in small intestines.

• Intermediate host
  - Sheep, goat, pigs, camel, man etc.
  - Disease: hydatidosis
**Echinococcus granulosus**

- **Hydatid cyst**
  - Liver, brain, kidney, lung, spleen
  - 10-20 years → coconut

- **Transmission**
  - To man → accidentally ingesting eggs
  - To dog → eating cysts in contaminated meat
Echinococcus granulosus

- Disease in man (hydatidosis)
- Pressure of cyst
- Cyst fluid $\rightarrow$ allergic reaction
- Cyst rupture $\rightarrow$ anaphylactic shock, fluid in bile ducts cause colicky pain and jaundice

Diagnosis:
- Scolex from cyst fluid
- Casoni intradermal test
Echinococcus granulosus

- Treatment: surgical if possible
- Don’t spill cyst fluid or hydatid sand
- Epidemiology: man, dog and sheep
- New zealand, australia, argentina, chili, china etc.
Conclusions

Urinary Tract Disorders in Parasitic Infections could be the results of:

1. The parasite
2. Host immune responses
3. Combination of those two factors