Malaria, Leshmaniasis & Schistosomiasis in Children

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OBJECTIVES

- By the end of this lecture each student should be able to:
  - Build a plan for taking history for a child presented with fever (acute or prolonged).
  - Differentiate between the different clinical presentation of malaria.
  - Revise the differential diagnosis of huge splenomegally.
  - List clinical presentation of leshmaniasis.
  - Diagnose and treat visceral leshmaniasis.
  - Compare between different types of schistosomiasis.
  - List complications of schistosomiasis and name the most important drug used for treatment.
MALARIA -----EPIDEMIOLOGY

- Acute and chronic problem

**Mode of transmission**

- Female anopheles mosquitoes
- Blood transfusion
- Contaminated needles
- Transplacental
- Organ transplantation
How does infection develop?

Malaria
(Plasmodium spp.)

Mosquito Stages
1. Oocyst
2. Ruptured oocyst
3. Release of sporozoites

Sporogonic Cycle
4. Oocyst
5. Ookinetes
6. Macrogametocyte
7. Microgamete entering macrogamete
8. Exflagellated microgametocyte

Exo-erythrocytic Cycle
9. Mosquito takes a blood meal (injects sporozoites)

Human Liver Stages
10. Liver cell
11. Infected liver cell

Ruptured schizont
12. Schizont

Human Blood Stages
13. Mosquito takes a blood meal (ngests gametocytes)
14. Gametocytes

Erythrocytic Cycle
15. Immature trophozoite (ring stage)
16. Mature trophozoite
17. Ruptured schizont
18. Schizont
19. Gametocytes

Legend:
\( \text{= Infective Stage} \)
\( \text{= Diagnostic Stage} \)
Severity of disease and host factors

several host factors determine the outcome of exposure to malaria:

• Naturally-acquired immunity.

• Red cell and haemoglobin variants.

• Foetal haemoglobin (HbF)

• Duffy blood group
This results from the:

1. Destruction of the RBC. Anemia
2. The liberation of parasites and RBC materials into the circulation, Fever
3. Host reaction to these events (Immunopathologic events)
Malaria---Clinical Presentation

- Asymptomatic during IP
- Classical presentation—simple malaria
- Severe Malaria
- Cerebral malaria
- Long term relapse ----- *P. vivax* and *P. ovale*
- Hyper reactive malaria splenomegally.(chronic malaria)
Clinical Course Of P. falciparum

A. Asymptomatic parasitaemia
   - natural immunity / high malaria endemicity
B. Simple, uncomplicated malaria / some degree of immunity to malaria / no life-threatening disease.

Fever is the most constant symptom of malaria.
Clinical features--Simple malaria

- Nausea, vomiting, diarrhea
- Abdominal pain
- Back pain
- Myalgia
- Paroxysm of fever (rigors, sweating)
- Headache
CLINICAL FEATURES – SEVERE MALARIA

- Impaired level of consciousness
- Respiratory distress
- Multiple convulsions
- Severe anemia
- Bleeding
- Circulatory collapse
- Pulmonary edema
- Haemoglobinuria
- Jaundice
- Hypoglycemia
- Acute renal failure
- Acidosis
Severe malaria --investigations

- Hb
- PCV
- Glucose
- Blood Gases
- LP if indicated
- blood Group & cross matching
- Blood culture
- Urea & Electrolytes
Severe Malaria - Complications

BLACK WATER FEVER

- Algid malaria
- Renal failure
- Splenic rupture
- Hypoglycemia
D:CEREBRAL MALARIA

- Caused by *P. falciparum*.
  - MOST COMMON AMONG CHILDREN
  - Rapidly developing encephalopathy (un arousable coma)
  - Develop over several days or suddenly
  - 20-40% fatality rate if not treated appropriately.
  - > in parasitemia >5%
CEREBRAL MALARIA

CLINICAL PRESENTATION

- Always decreased level of consciousness
- Drowsiness
- Headache
- Confusion
- Delirium
- Hallucinations
- Deep coma
- Seizures
- Muscle twitching
- Contracted or unequal pupils
- Retinal hemorrhage
- Hemiplegia
- Absent tendon reflexes
- Positive Babinski sign
- Opisthotonus (mention 2 differential diagnosis)
Malaria----Diagnosis

1. A good history
   - Residence or a recent visit (in the preceding 3 months) to a malaria endemic area
   - The diagnosis of malaria should be considered in any unwell person who has been in a malaria area recently

2. Physical examination
   - Identify signs consistent with malaria: fever, pallor, jaundice, splenomegaly

3. LAB diagnosis
MALARIA—LAB DIAGNOSIS

- Microscopy: Thick film for parasite identification & Thin blood film: used for species identification and...
Other methods of diagnosis of malaria

These are not routinely used in clinical practice. They include:

a) Antigen capture kits.
b) PCR
c) Fluorescent techniques.
d) Serologic tests.
P. Falciparum Malaria—Diagnosis

- Criteria that suggest falciparum malaria
  - Symptoms < 1 month after return from endemic area
  - Parasitemia > 2%
  - Rings form with double chromatin dots
  - Erythrocytes infected with more than one parasite
Appearance of *P. falciparum* in thin blood films

Ring forms or trophozoites; many red cells infected – some with more than one parasite
TREATMENT — UNCOMPLICATED MALARIA

- First-line treatment: chloroquine/ARTUSENATE
  For *P. vivax* and *P. ovale* infection plus primaquine.
- 2. Second-line treatment: Fansidar
- 3. Third-line treatment:
  - Mefloquine
  - Quinine
Cerebral malaria and severe Malaria

---Treatment

1) **Supportive treatment**
2) **Treatment of complications**
3) **Antimalarial treatment**
   - Quinine infusion
   - Artemether injections
SCHISTOSOMIASIS

"bilharziasis" after Theodor Bilharz who identified the parasite first in 1852.
World Wide Distribution of Schistosomiasis
Pathophysiology

The adult worms migrate against portal blood flow

- The mesenteric venules of the small intestine — *S. japonicum*
- The mesenteric venules of the colon — *S. mansoni* and *S. intercalatum*
- The vesical venous plexus — *S. haematobium*
Clinical Features

- **Acute infection** — Acute symptoms may present as swimmer's itch or Katayama fever.
- **Intestinal schistosomiasis**
- **Hepatic schistosomiasis**
- **Urinary schistosomiasis** — terminal hematuria
- **Neurologic complications** — transverse Myelitis
Hepatic Schistosomiasis

1. Inflammatory hepatic schistosomiasis causes hepatomegaly and splenomegaly in children.

2. Chronic hepatic schistosomiasis:
   - splenomegaly, portal hypertension, ascites and hematemesis from esophageal varices.
Diagnosis

- Demonstration of parasite eggs in stool or urine /multiple samples.
- Serology
- Abdominal U/S for periportal fibrosis, measure portal vein pressure.
- Upper GI endoscopy in cases of suspected esophageal varices
Treatment of schistosomiasis

Praziquantel
Leishmaniasis

- Group of diseases caused by infection with one of the protozoan parasites of the genus *Leishmania*
- Designated one of the five most important diseases worldwide by the World Health Organization
- 20 million people infected worldwide
It is known in the Kingdom back to 1950. Ministry of Health has established the leishmaniasis unit in the 1980 Under The precautionary medicine to follow-up the disease in the Saudi cities
(1) **Cutaneous Leishmaniasis**: caused by *L. tropica*, *L. major*, *L. ethiopica*, *L. mexicana*, *L. braziliensis*

(2) **Mucocutaneous Leishmaniasis**: caused by *L. etheopica* and *L. braziliensis*

(3) **Visceral Leishmaniasis**: caused by *L. donovani*.
Sand fly takes a blood meal (injects promastigote stage into the tissue)

Promastigotes are phagocytized by macrophages

Promastigotes transfer into amastigotes inside macrophages

Amastigotes multiply in cells (including macrophages) of various tissues

Ingestion of parasitized cell

Amastigotes transform into promastigote stage in midgut

Divide in midgut and migrate to proboscis
Cutaneous leshmaniasis — clinical presentation

- Exposed skin (face & extremities)
- One or more lesion
- Papular, nodular, plaque like or ulcerative lesion
- Non tender ulcer surrounded by a sharp indurated erythematous margin
- No drainage, unless infected by bacteria
- Ended with residual scar
Diagnosis

- is mainly clinical in and the golden role is: any boil >1 month duration is leishmaniasis until prove otherwise
- Direct smear
- Culture in NNN media.
- Leishmanin skin test
- Immunological tests
- Polymerase chain reaction
Treatment

- LOCAL:-
  - Chemical treatment mainly Na stibogluconate (pentostam)
  - Physical treatment: as Cryotherapy, infra red therapy, Excision or cautery
Visceral leishmaniasis

- There are geographical variations.
- The diseases is called kala-azar
- *Leishmania infantum* mainly affect children
- *Leishmania donovani* mainly affects adults
VIScERAL LESHMANIASIS—CLINICAL PRESENTATION

- During the 1st few weeks to months:
  - Intermittent fever
  - Loss of energy
  - Active kalazar within 2-8 months
  - Increase in spleen size
- Classical features develop approximately 6 month after the onset of the illness
- High fever

- Marked splenomegally
- Hepatomegally
- Severe cachexia

At the terminal stages, massive hepatosplenomegally
- wasting, pancytopenia.
- jaundice, edema and ascites, bleeding episodes, in late stages secondary bacterial infections
- Rapid clinical course has been noted in up to 20% of cases
What are the differential diagnoses of huge splenomegally?
Visceral leishmaniasis--diagnosis

1. CBC: anemia, thrombocytopenia, leukopenia
2. High ESR
3. Elevated hepatic transaminasis
4. Hyperglobulinemia— IgG

(1) Parasitological diagnosis:

- Bone marrow aspirate
- Lymph node
- Liver biopsy
- Splenic aspirate

1. Microscopy
LD bodies in bone marrow

Figure-2: Smear of bone marrow aspirate showing Leishmania Donovani bodies.
(2) Immunological Diagnosis:

- Specific Direct Agglutination Test (DAT)
- Skin test (leishmanin test). Non specific
- Non specific detection (formal-gel) test or by electrophoresis.
Treatment:

- Pentavalent antimony- sodium stibogluconate (Pentostam)
- Amphotericin B

Treatment of complications:

- Anemia
- Bleeding
- Infections
Any questions?