General pathology of

BILHARZIASIS

Dr Ahmed Roshdi

Prof of Pathology,

Faculty of Medicine, Sohag University

March 2024

Introduction

Parasitic infections

- **Means:**

- Tissue invasion by parasitic organisms.

- **Routs of entrance:**

Through penetration of surface epithelium

- Skin
- Gastrointestinal mucosa
- Respiratory mucosa
- Urogenital mucosa

Introduction

Parasitic infections

- **Routs of spread:**
  - Direct extension: the commonest
  - Through lymphatics
  - Through blood vessels
- **Release from the body:**
  - Urine
  - Fecal matter
  - Vectors (insects)
  - Coughing or sneezing (less common)

Introduction

Parasitic infections

- **Common examples**
  - Schistosomiasis (bilharziasis)
  - Leishmaniasis
  - Filiarasis
  - Hydatid disease
  - Toxoplasmosis
  - Anylostoma
  - Malaria

Introduction

Parasitic infections

- **Common examples**
  - **Schistosomiasis (bilharziasis)**
  - Leishmaniasis
  - Filiarasis
  - Hydatid disease
  - Toxoplasmosis
  - Anylostoma
  - Malaria

Most important

Schistosomiasis (Bilharziasis)

Schistosomiasis

❑ Definition:

Chronic granulomatous disease caused by schistsoma infection

❑ Epidemiology:

- A worldwide distributed disease
- Involves >200 millions worldwide
- Half of the cases are reported in Africa

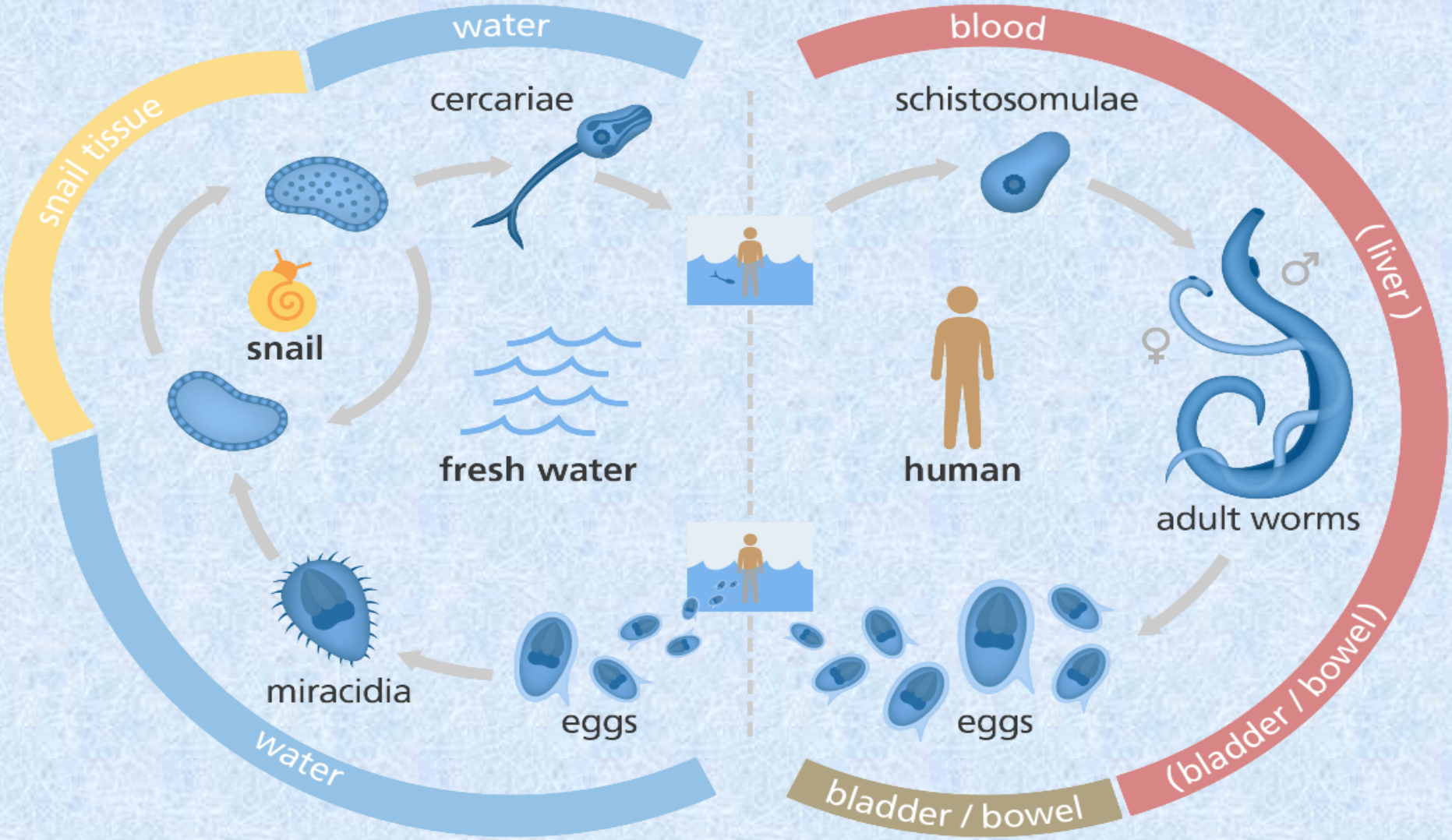
Schistosomiasis

- **Schistosoma organism:**

S. Hematobium	Africa and middle east
S. Mansoni	Africa, middle east, south America and Caribian
S. Japonicum	Far east (Japan)
S. Intercalatum	Central west Africa

Schistosomiasis

- **Schistosoma organism:**



Schistosomiasis

- **In Egypt:**
  - Reported by the German anatomist **Theodore Bilharz in 1851**
  - **Theodore Bilharz research center in Cairo**
  - Both S hematobium and Mansoni are endemic

1825-1862

Schistosomiasis

- **General pathological features:**
  - **Effects by cercaria**
    - Grossly/clinically: acute dermatitis in the form of skin rash or papules at the site of skin penetration.
    - MP: infiltration by neutrophils + eosinophils due to type I HSR
    - The lesions disappear within 2-7 days.

Schistosomiasis

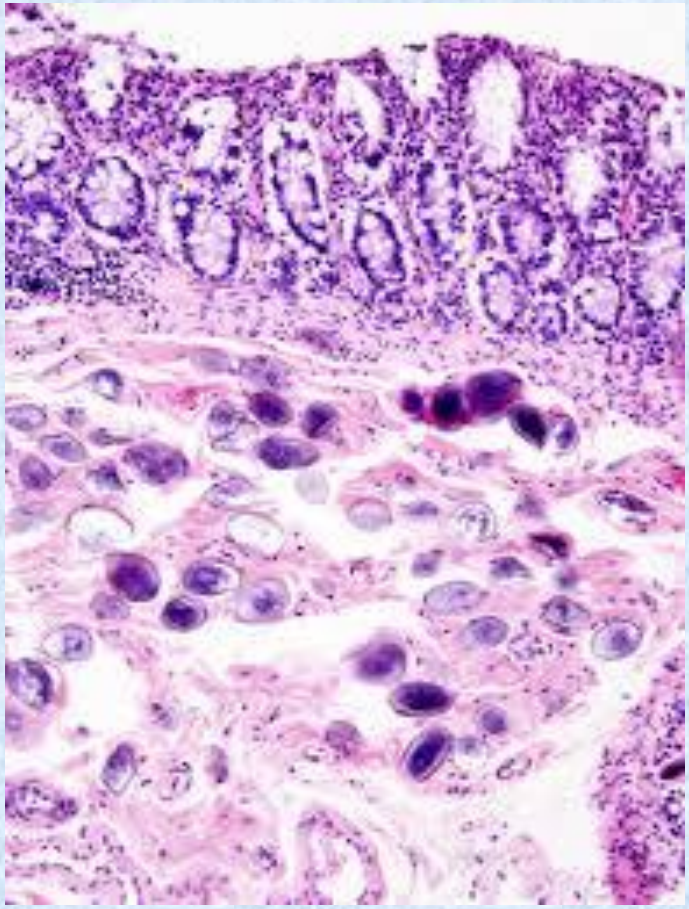
- **General pathological features:**
  - **Effects by the worms**
    - Living worms: minimal effects in the form of brown pigments at certain sites
    - Dead worms: May induce localized tissue necrosis due to endotoxins released from dead worms. ***MP***: dense infiltrate of eosinophils, macrophages and neutrophils.

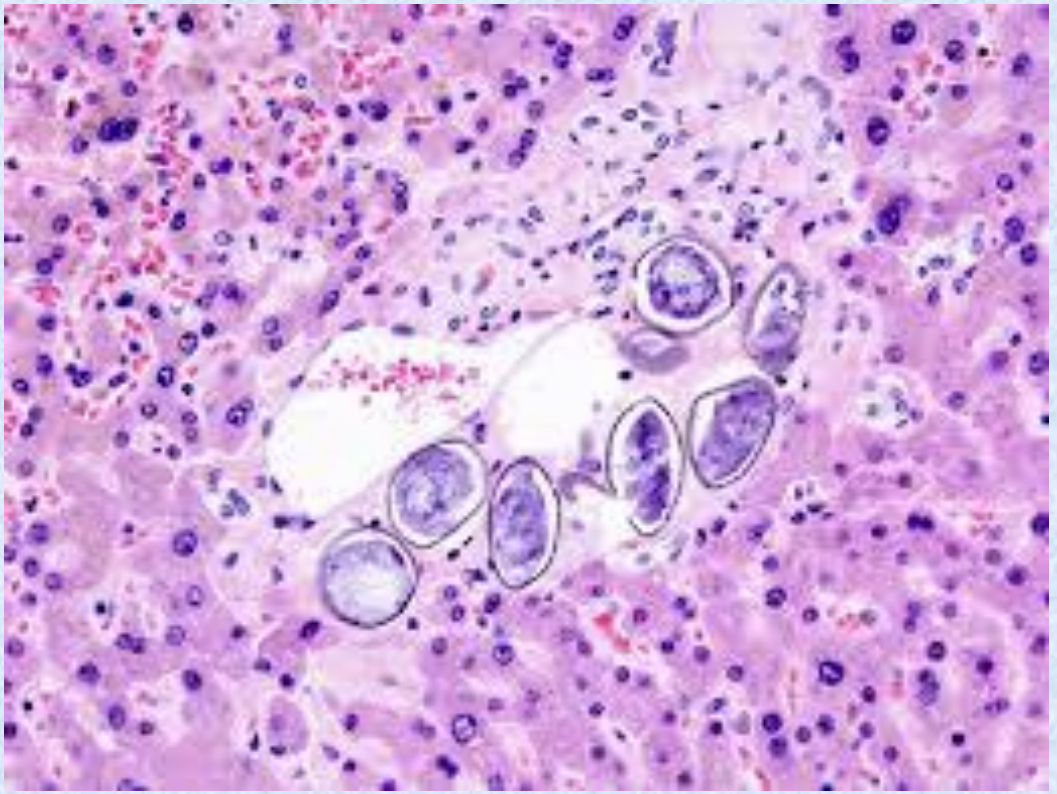
Schistosomiasis

- **General pathological features:**
  - **Effects by the ova**
    - The most important damaging effect.
    - Egg antigens induce granulomatous inflammation (Type IV hypersensitivity reaction): macrophages (epithelioid cells), lymphocytes, plasma cells, eosinophils and giant cells.
    - Healing occurs by fibrosis and the ova may show calcification.
    - The extent of damage depends on number of deposited ova and surrounding fibrosis.

Schistosomiasis

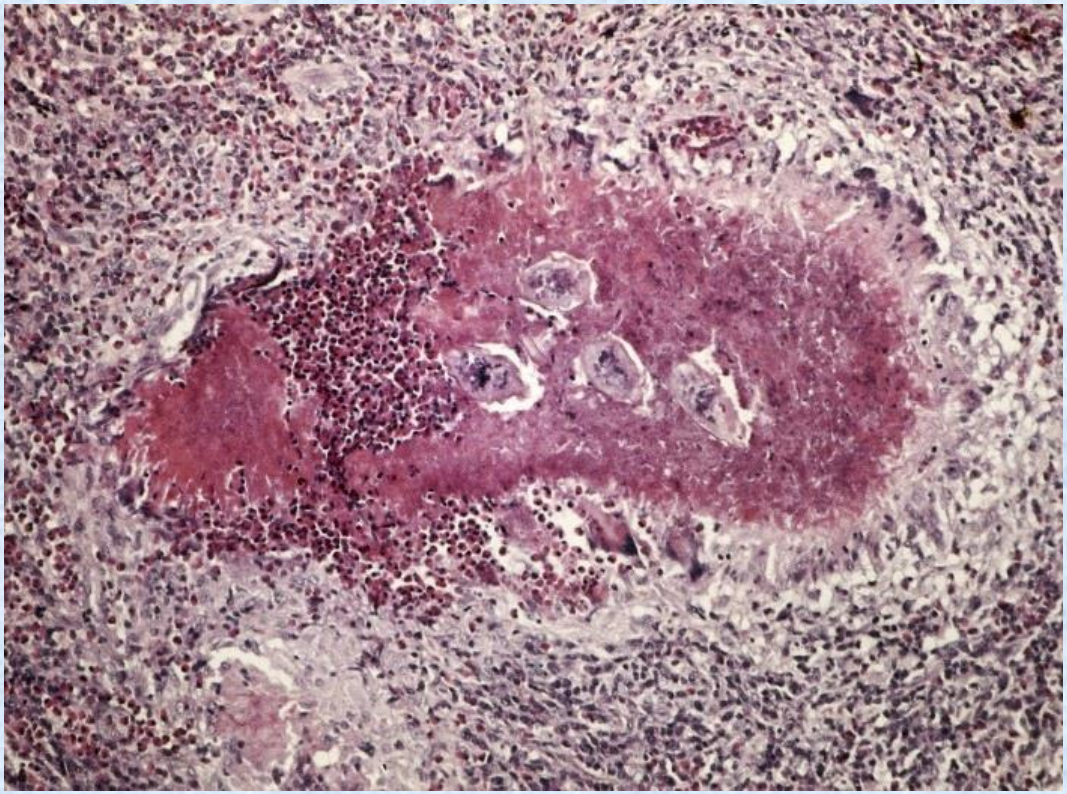
- **General pathological features:**
  - **Effects by the ova**

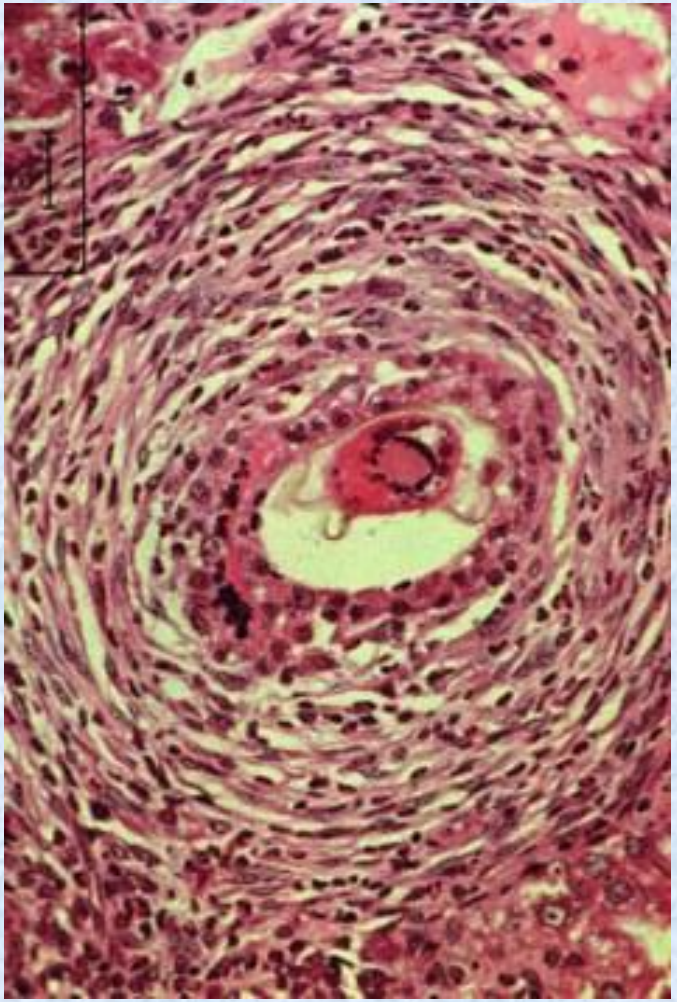




Schistosomiasis

- **General pathological features:**
  - **Effects by the ova**





Schistosomiasis

- **Common organs involved:**
  - **Urinary system**
  - **Male genital tract**
  - **Female genital tract**
  - **lungs**
  - **Large intestine**
  - **Liver**
  - **Spleen**
  - **Skin**

Schistosomiasis

- **Common organs involved: most important**
  - **Urinary system**
  - **Male genital tract**
  - **Female genital tract**
  - **lungs**
  - **Large intestine**
  - **Liver**
  - **Spleen**
  - **Skin**

Urogenital Bilharziasis

Urogenital bilharziasis

- **General landmarks:**
  - Caused mainly by S hematobium
  - Urinary bladder is the main affected site
  - The ova affect the whole wall thickness but **submucosa** is heavily involved
  - The main presentation is terminal hematuria
  - Bilharzial granuloma and dense fibrosis are frequent

Urogenital bilharziasis

- **Pathological features of UB bilharziasis:**
  - **Gross features:**
    - ***In chronic prolonged cases:***

      The following lesions develop:

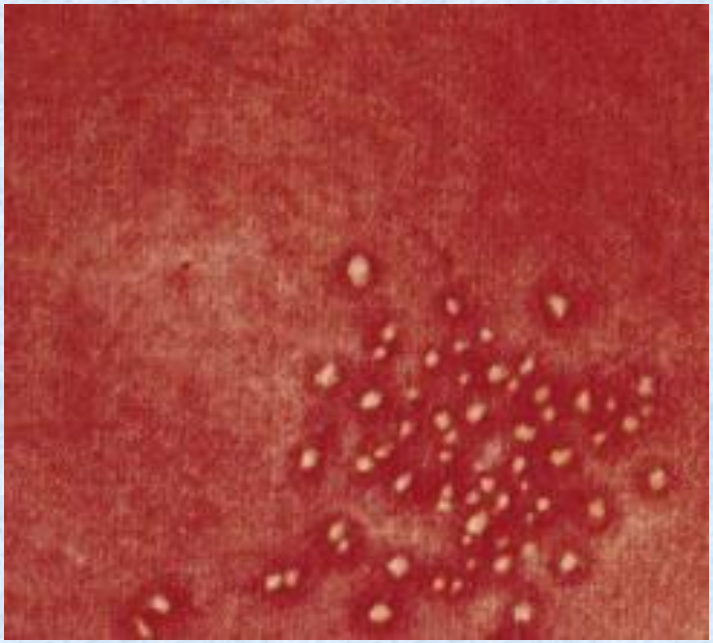
      1. Sandy patches
      2. Bilharzial ulcers
      3. Bilharzial polyps

Urogenital bilharziasis

- **Pathological features of UB bilharziasis:**

Sandy patches

- Very common
- Gross: dirty yellow raised patches
- MP:
  - Atrophic epithelium
  - Fibrotic submucosa
  - Packed calcified ova



Urogenital bilharziasis

- **Pathological features of UB bilharziasis:**

Bilharzial ulcer

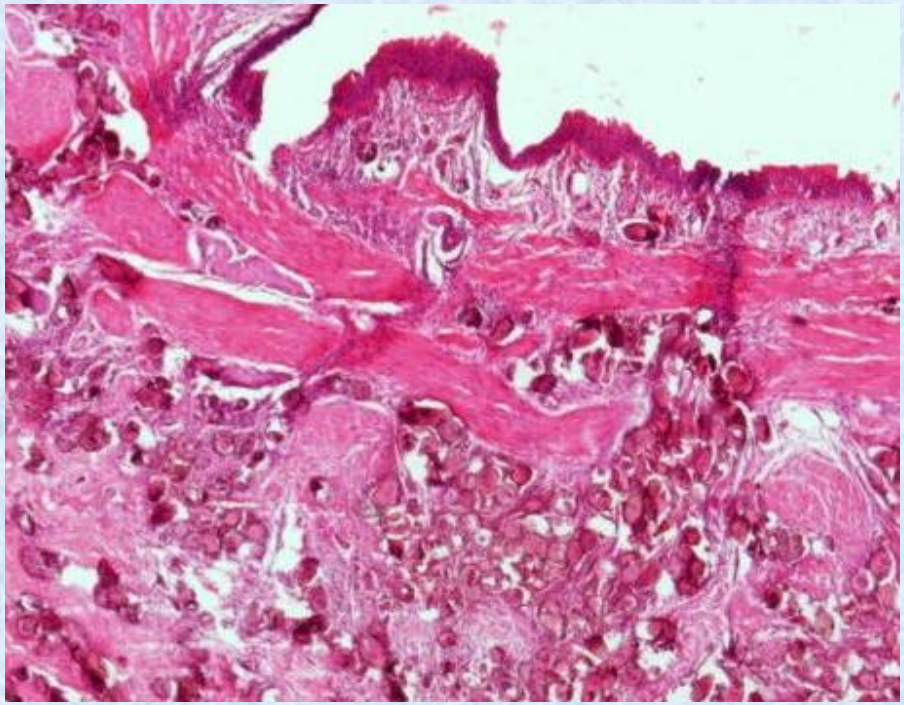
- In 20% of cases.
- Shallow, deep of fissure like.
- Granulation tissue at ulcer floor
- MP: calcified bilharzial ova with fibrosis.

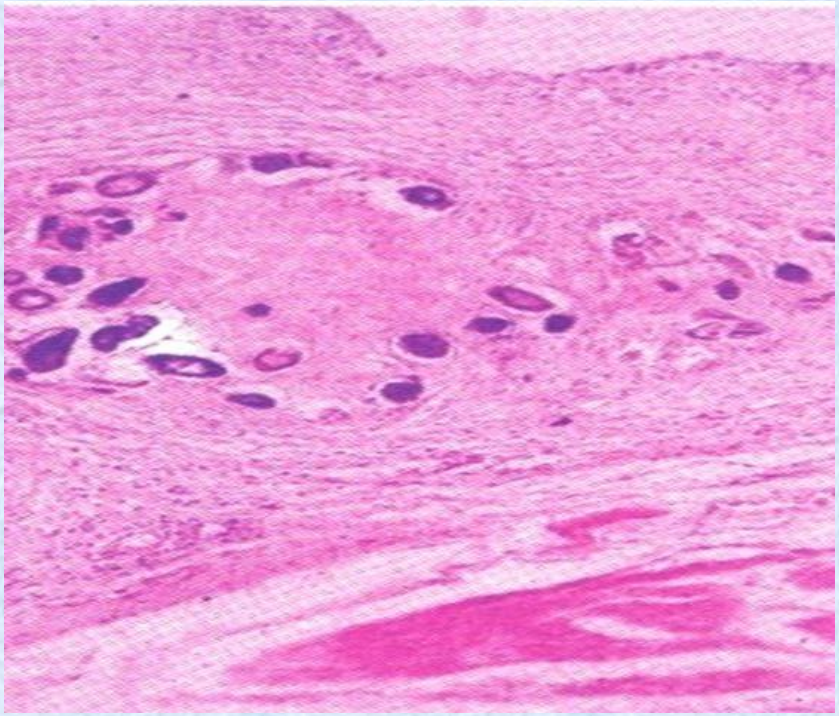
Bilharzial polyp

- Not common
- Single or multiple, but always few in number.
- Small polyps are sessile and larger ones are pedunculated
- MP: polyp with bilharzial granuloma

Urogenital bilharziasis

- **Pathological features of UB bilharziasis:**





Urogenital bilharziasis

- **Pathological features of UB bilharziasis:**

MP of urinary bilharziasis

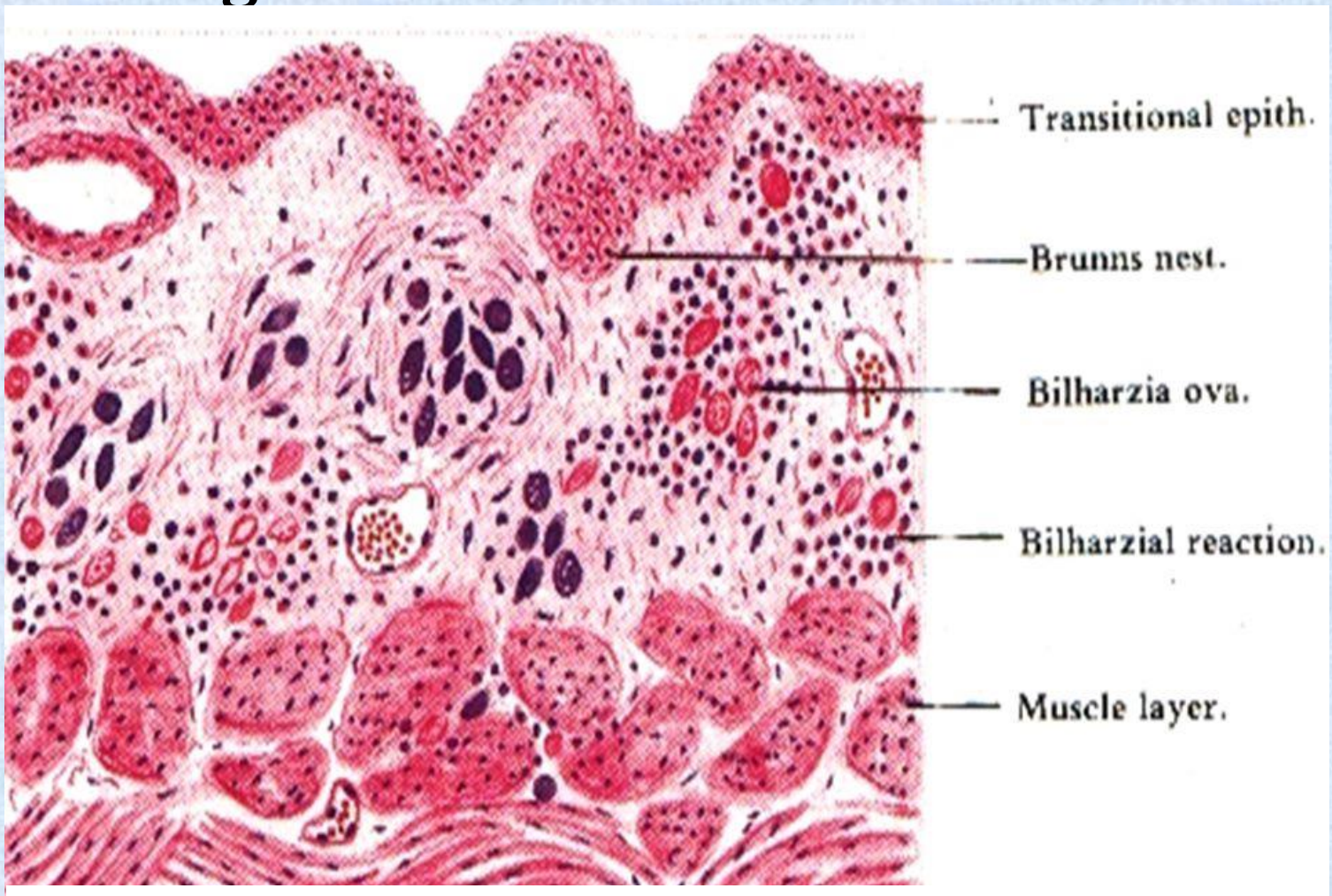
A- Bilharzial granuloma (Describe??)

B- Urothelial changes

1. Urothelial atrophy
2. Urothelial Hyperplasia
3. Brunn`s nests
4. Cystitis cystica
5. Cystitis glandularis
6. Urothelial dysplasia
7. Squmaous metaplasia
8. Squamous dysplasia

Urogenital bilharziasis

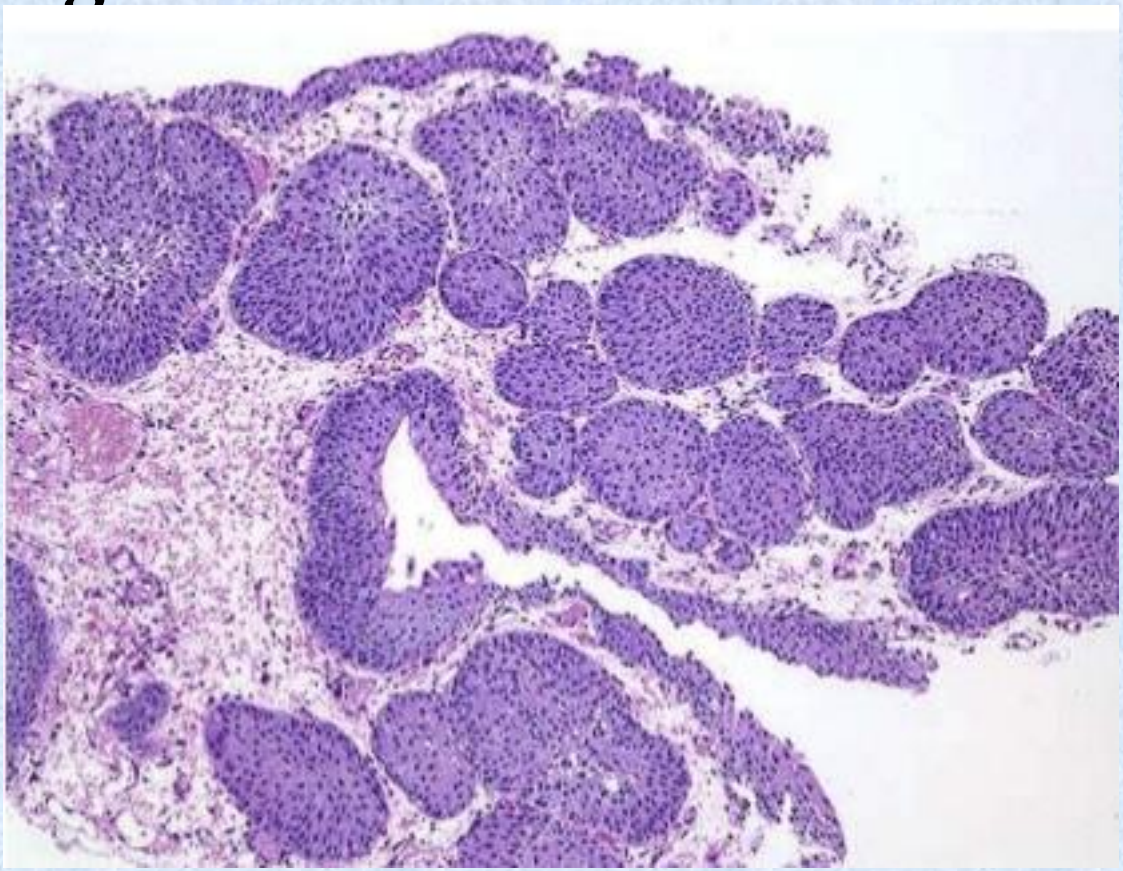
- **Pathological features of UB bilharziasis:**



Bilharzial granuloma

Urogenital bilharziasis

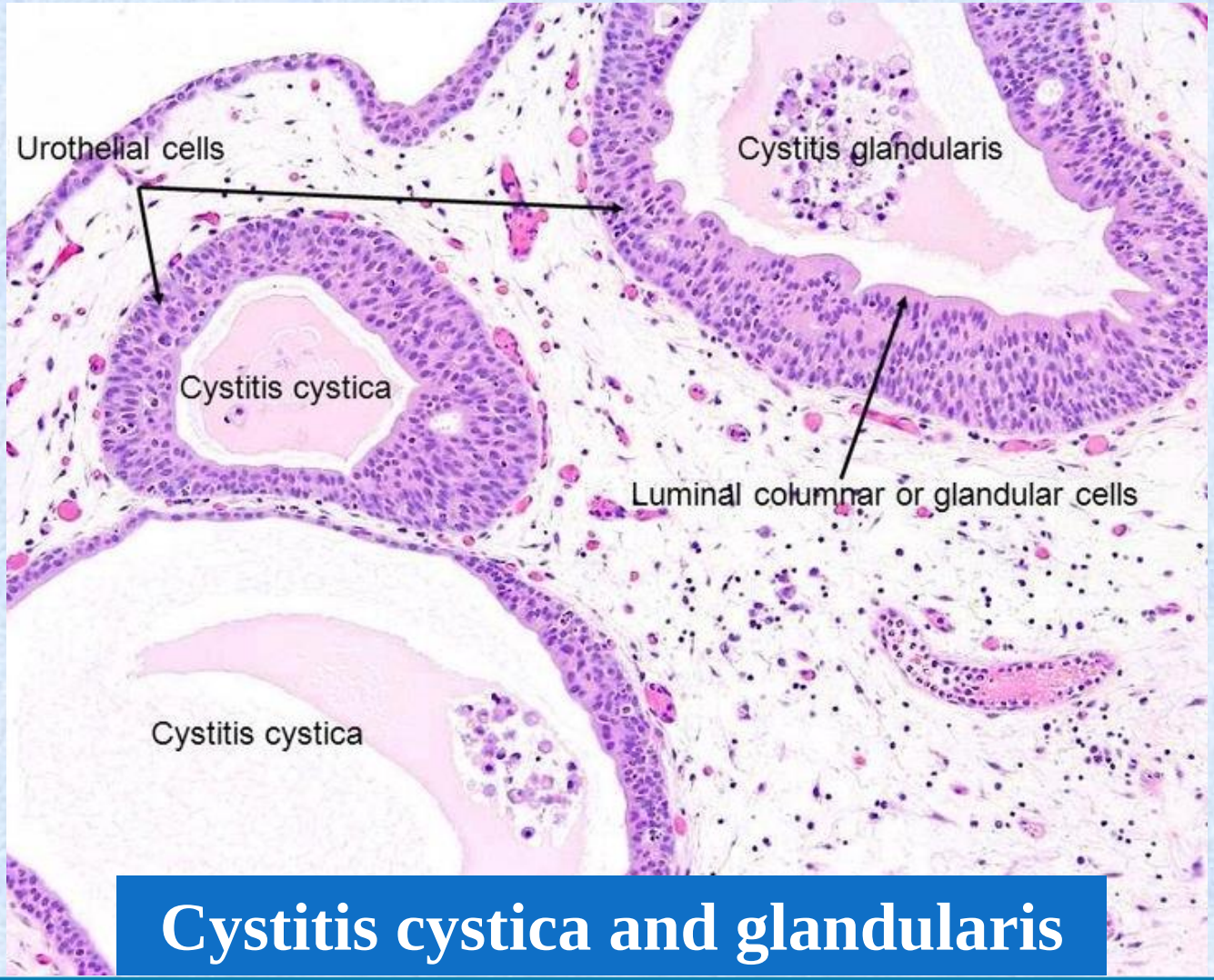
- **Pathological features of UB bilharziasis:**



Brunn`s nests

Urogenital bilharziasis

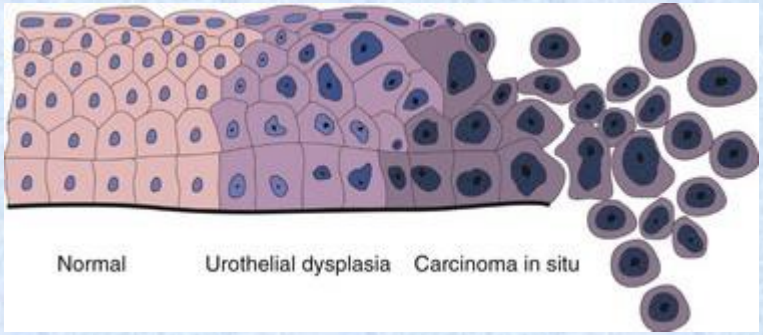
- **Pathological features of UB bilharziasis:**

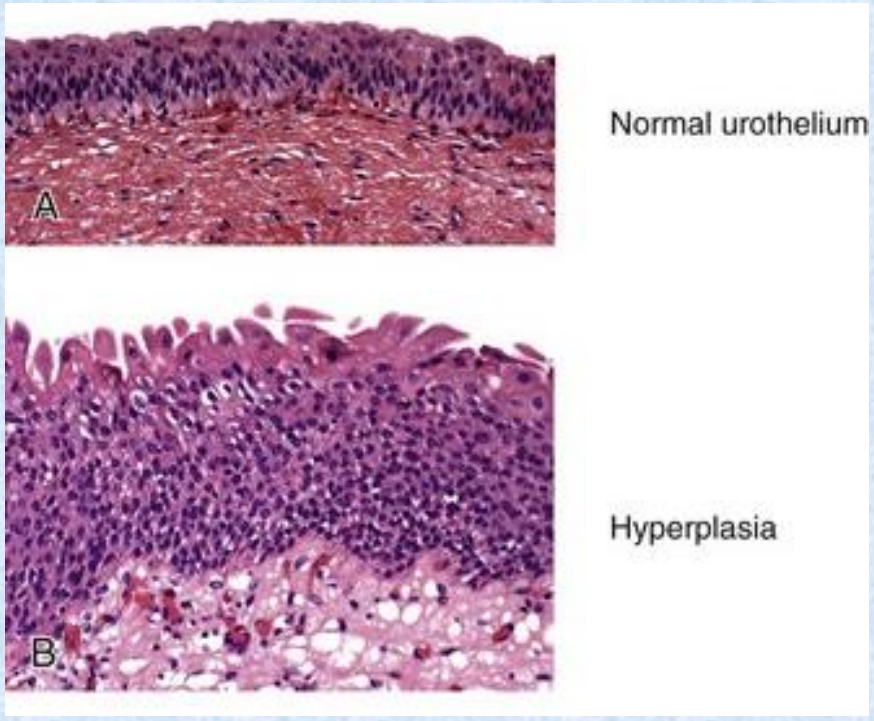


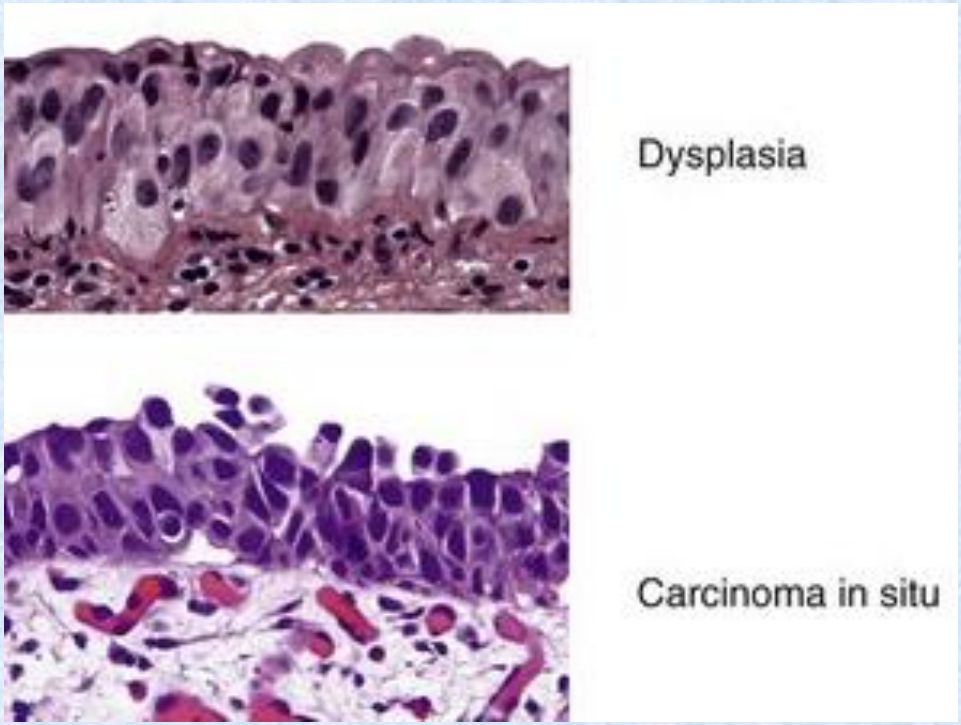
Cystitis cystica and glandularis

Urogenital bilharziasis

- **Pathological features of UB bilharziasis:**







Urogenital bilharziasis

- **Complications of UB bilharziasis:**

1. Hematuria causes anemia (microcytic hypochromic)
2. Secondary bacterial infection of the bladder wall and peri-vesical tissue.
3. Ova and epithelial debris provide nuclei for phosphate stone formation
4. Fibrosis at the bladder neck causes hypertrophy of UB wall
5. Dilatation of bladder wall with bilateral hydroureter and hydronephrsosis.
6. Urinary bladder diverticula formation
7. Renal failure
8. **Predispose to cancer bladder:** Urothelial, squamous or glandular carcinomas

Intestinal Bilharziasis

Intestinal bilharziasis

- **General landmarks:**
  - Caused mainly by S mansoni
  - Colorectal region is the main affected site
  - The ova affect the whole wall thickness but **submucosa** is heavily involved
  - In sever cases, multiple serosal nodules (granulomas) may be seen
  - The main presentation is bilharzial dysentery

Intestinal bilharziasis

- **Pathological features of intestinal bilharziasis:**
  - **Gross features:**
    - ***In chronic prolonged cases:***

      The following lesions develop:

      1. Bilharzial polyp
      2. Bilharzial ulcers
      3. Sandy patches
      4. Intestinal wall fibrosis

Intestinal bilharziasis

❑ Pathological features of intestinal bilharziasis:

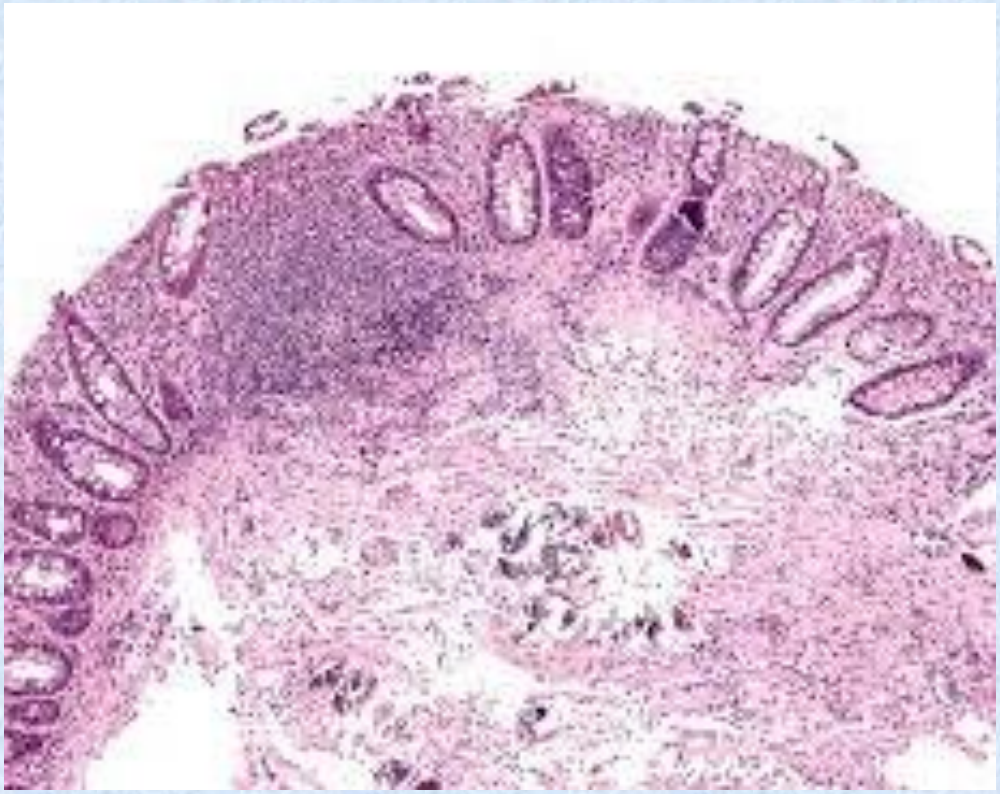
Bilharzial polyp

- Very common
- Pathogenesis: Granuloma formation → mucosal elevation → polyp formation
- Grossly: - Single or multiple
  - Sessile, pedunculated or complex
  - May reach a large size
- MP: core of fibro-connective tissue with bilharzial granuloma (**describe??**)

Intestinal bilharziasis

- **Pathological features of intestinal bilharziasis:**

Bilharzial polyp



Intestinal bilharziasis

❑ **Pathological features of intestinal bilharziasis:**

Bilharzial ulcer

- Less common than polyp
- Develop at site of ova penetration, falling of mucosa or polyp avulsion
- Gross: small or large ulcers with irregular outlines, granular floor and firm base
- MP: Bilharzial granuloma

Sandy patches

- Rare

Intestinal wall fibrosis

- Of varying degree
- Leads to chronic abdominal pain
- May lead to chronic intestinal obstruction

Intestinal bilharziasis

- **Complications of intestinal bilharziasis:**

1. Recurrent intestinal hemorrhage (bilharzial dysentery) leading to microcytic hypochromic anemia.
2. Secondary bacterial infection of the ulcerated mucosa
3. Intestinal obstruction:; due to:
   - Fibrosis of the wall (chronic obstruction)
   - Obstruction by a large polyp (acute obstruction)
   - Secondary to intussusception (acute obstruction).
4. Spread of bilharzial to the liver (bilharzial fibrosi)

Intestinal bilharziasis is NOT precancerous

Hepatic Bilharziasis

Hepatic bilharziasis

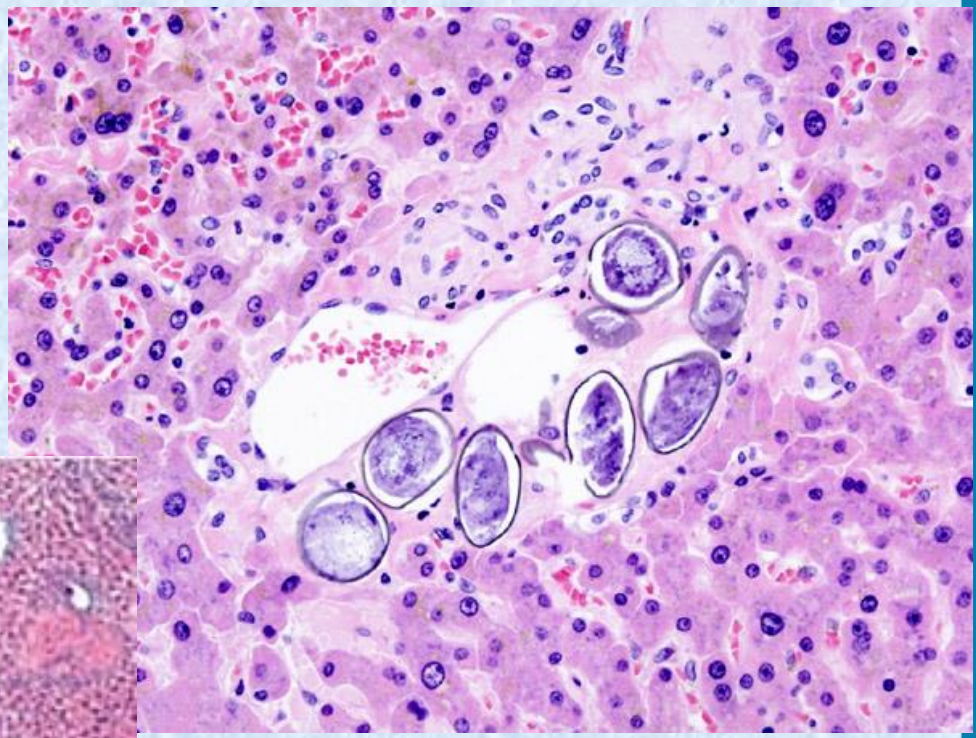
- **Pathogenesis of hepatic bilharziasis lesions:**
  - A common disease in Egypt.
  - Always secondary to intestinal bilharziasis.
  - The main lesion involves **PORTAL TRACTS**
  - Emboli of ova reach the liver by the portal vein.
  - Multiple bilharzial granulomas develop in **portal tracts** with subsequent healing by fibrosis (periportal fibrosis).
  - Dilated anastomosis between hepatic portal venules and hepatic arterioles
  - **Hepatic lobules (architecture) are almost intact**

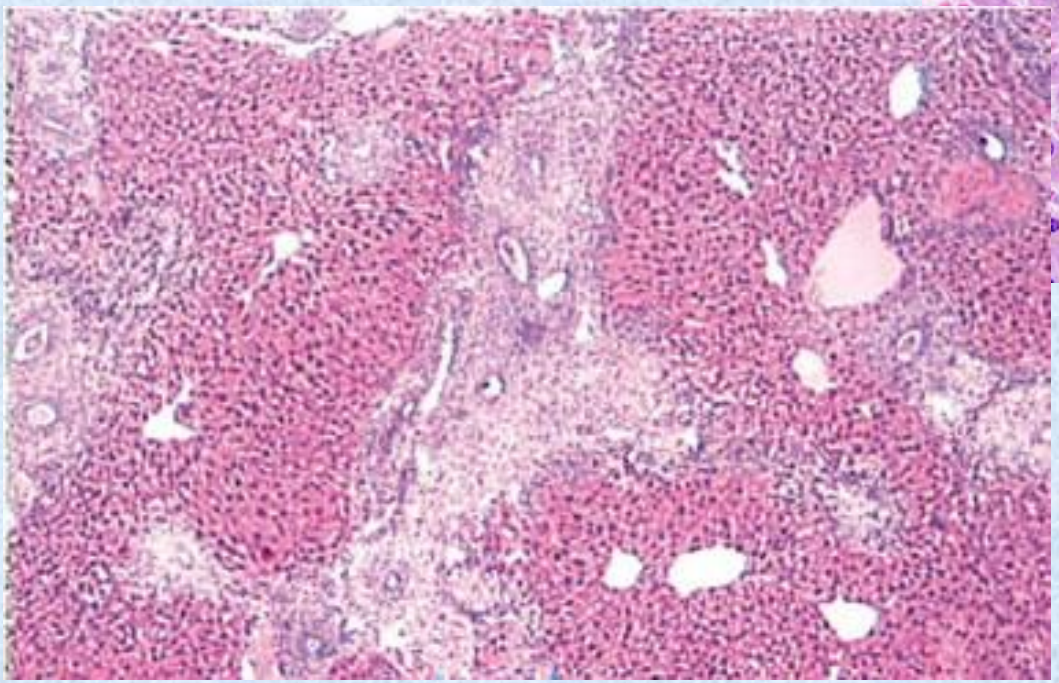
Hepatic bilharziasis

- **MP:**
  - Bilharzial granuloma involving portal tracts (**Describe??**)
  - Hepatic lobules and architecture are intact
  - Hepatocytes may show cloudy swelling or fatty changes
  - Hyperplasia of kupffer cells

Hepatic bilharziasis

- **MP:**





Hepatic bilharziasis

- **Complications of hepatic bilharziasis:**

A. Portal hypertension

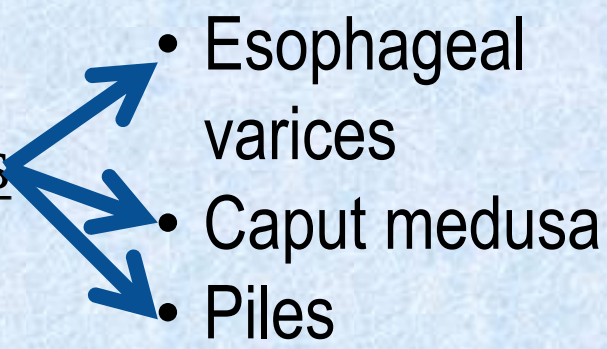
Due to:

-Compression of hepatic portal venules by fibrosis

-Anastomosis of portal venules with hepatic arterioles

Effects:

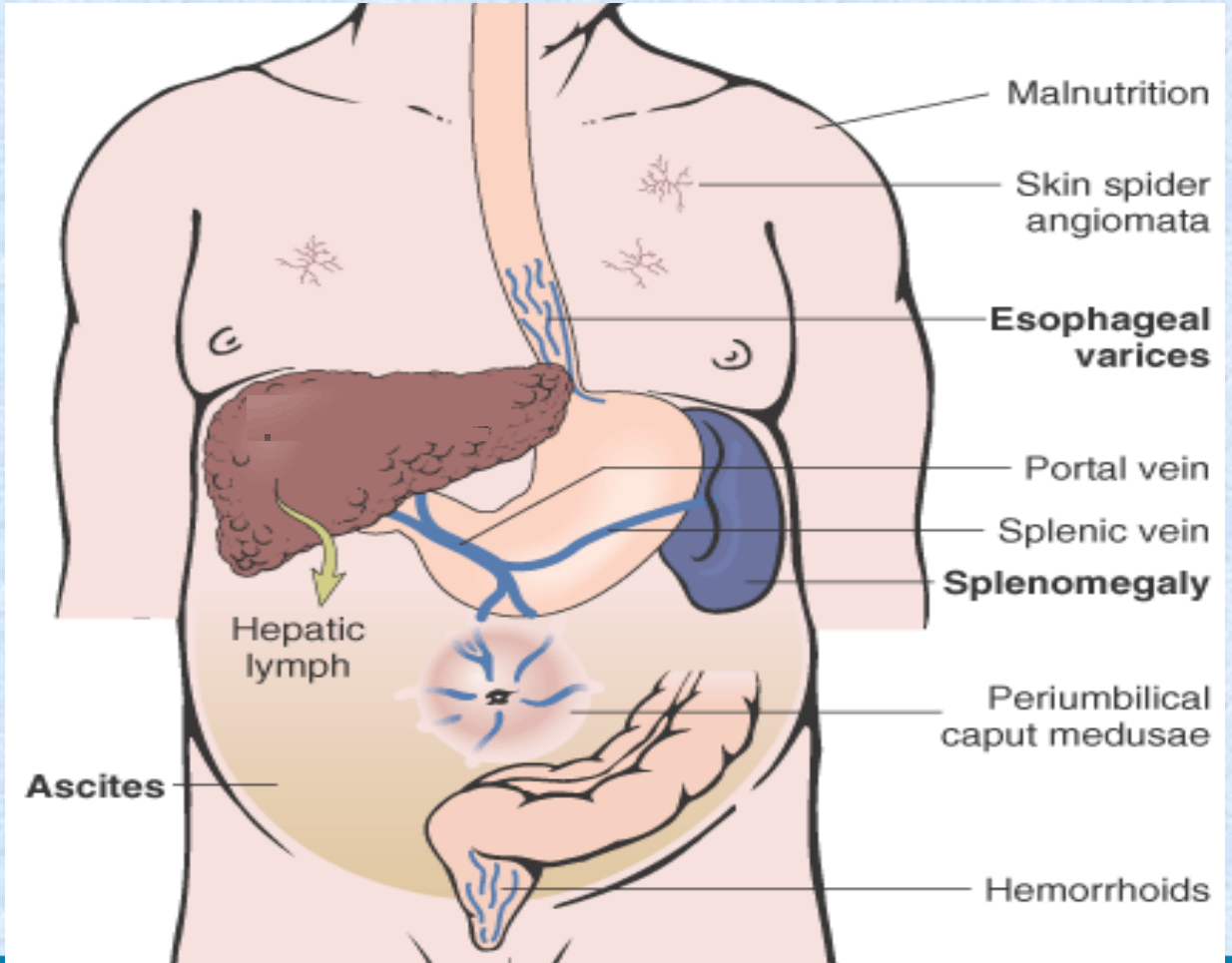
1. Congestive splenemegally
2. Opening of portsystemic collaterals
3. Ascites



Hepatic bilharziasis

- **Complications of hepatic bilharziasis:**

A. Portal hypertension



Hepatic bilharziasis

- **Complications of hepatic bilharziasis:**

B. Thrombosis of portal and splenic veins: due to stasis.

C. Disturbances in liver functions: **rare** and less severe than in cirrhosis, as liver architecture and function are usually not affected in Bilharzial fibrosis

The main cause of death is hematemesis due to esophageal varices.

Thank you