Trematodes

General characteristics:-

1- They are commonly referred to as flukes.
2- Life cycle have 3 morphologica form:-
   a- Egg
   b- Multiple larval stage
   c- Adult worm
3- The eggs, which are the primary morphologic form recovered in human specimens vary; some contain operculum and others distinguished by the presence of spine like schistosoma.
4- The larval stage typically occur outside the human host.
5- The adult flukes are thin or nonsegmented resembling leaves both in shape or thickness.
6- Each adult fluke is equipped with 2, muscle or cup-shaped suckers, one oral and the other located ventrally.
7- The life cycle placed into 2, categories
   a- Those take up residence in intestine, bile duct or lung (organ dwelling).
   b- Those take up residence in blood vessels around the intestine or bladder (blood dwelling).
8- Human infection of organ-dwelling flukes occurs following the ingestion of water plants, fish, crab, or crayfish contaminated with encysted metacercaria.
9- Human infection with blood-dwelling flukes occurs following the penetration of cercaria into skin.
Phylum  \( \rightarrow \)  Platy helminthes

Class  \( \rightarrow \)  Trematoda include :-

1- Intestinal spp.
   Example  \( \rightarrow \)  *Fasciolopsis buski*

2- Liver spp.
   Example  \( \rightarrow \)  *Fasciola hepatica*

3- Lung spp.
   Example  \( \rightarrow \)  *Pavagonimus westermani*

4- Blood spp. Include:-

   a- *Schistosoma mansoni*

   b- *Sch. japonicum*

   c- *Sch. haematobium*
Liver Flukes

**Fasciola hepatica (Fascioliasis)**

*Fasciola hepatica*

Fasiola infections are common in domestic ruminants and wildlife throughout the world and cause massive economic loss in the livestock industry. Humans usually become infected by eating aquatic plants grown in water contaminated with faeces from animals harbouring fasciola.

**Aetiology and life cycle**

F. hepatica (the sheep liver fluke) and F. gigantica (mainly of cattle) cause fascioliasis in humans. The parasites vary in adult and egg size and shape and species of the snail host of the family Lymnaeidae. F. hepatica common in temperate and subtropical areas. Especially in sheep – raising areas. The adult worm lives in the bile duct of the final host and eggs are excreted in the faeces of the host. The eggs undergo further development upon reaching a water body; miracidium then hatch and penetrate a suitable snail host. After multiplication as sporocysts and rediae, free – swimming mature cercariae exit the snail, attach to aquatic vegetation and become metacercarial cysts. These cysts establish infection upon ingestion by man and other mammals. They excyst in the duodenum, then migrate through the intestinal wall into the body cavity through Glisson’s capsule across the liver parenchyma and into the bile duct, where they may live for many years egg are excreted 3-4 months after ingestion. Generally the life cycle is
maintained by domestic animals, particularly by sheep for *F. hepatica* and cattle/buffalo for *F. gigantic*; it is completed in 4-6 months.

**Pathogenesis**

These parasites cause considerable mortality in sheep and cattle, and human morbidity which is dependent on the number of worms and stage of infection. The acute phase occurs during migration of the immature flukes through the liver. Severe pathology results from parasite ingestion and destruction of parenchymal tissue, haemorrhage, parasite death inflammatory responses largely mediated by eosinophils. Repair mechanisms can lead to extensive fibrosis. Increased pressure atrophy of the liver and periportal fibrosis. The chronic phase, during which parasites are present in the bile ducts tends to be less severe tissue change, including, bile duct proliferation, dilatation and fibrosis, is largely caused by mechanical obstruction of the ducts inflammatory responses and the activity of proline, which the fluke excretes in large quantity, proline may facilitate movement of the parasite through the narrow ducts. Anaemia may result from blood loss through bile duct lesions. Death is uncommon, but is usually caused by haemorrhaging in the bile duct and case reports suggest it occurs more frequently in children.

Flukes that migrate out of the intestine but do not locate in the liver can form ectopic lesions in many tissues. These nodules, granulomas or migration tracts are often misdiagnosed as malignant tumours or gastric ulcers.
Clinical Features

Where cases are symptomatic, diarrhea, upper abdominal pain or pain in the right costal margin, urticaria, malaise, weight loss, coughing, fever and night sweats may begin approximately 2 months following ingestion of metacercaria and 1-2 months prior to the onset of egg excretion. The signs of this acute phase of infection are hepatomegaly, splenomegaly, anaemia, weakness and marked peripheral eosinophilia, up to 80%.

Adult flukes in the bile ducts may be associated with cholangitis and calculous or acalculous cholecystitis. Through their large size and the inflammatory and fibrotic response, the infection may cause obstruction leading to cholestaitc jaundice, nausea, pruritus, abdominal pain, hepatomegaly and fatty food intolerance. In severe cases, ascites with blood and severe anaemia may ensue. Since these moderate signs and symptoms do not differ from cholangitis and cholecystitis of other causes, the infection often goes unnoticed until worms are observed at surgery or histopathology. Eosinophilia and a history of eating water plants should be considered in the differential diagnosis.

Diagnosis and investigations

1- Fascioliasis has been diagnosed by observation of eggs during faecal examination,
2- by parasite-specific antibody detection in a variety of immunodiagnostic assays,
3- by radiological methods laparotomy.
4- Dietary history is also helpful, particularly in investigating outbreaks.

Examination of faces for eggs is of limited use since eggs are not excreted during the invasive stage of infection, when many patients present with severe symptoms. Often eggs are un-detectable during the chronic phase, but whether the techniques used are insensitive for very low egg outputs in light infections (< 100 eggs per gram) or eggs are not being produced is unclear.

A further problem with faecal examination is that eggs may be detected after ingestion of liver from infected animals. This does not indicate infection; thus positive cases should be reconfirmed if liver has been eaten recently.

Immunodiagnostic tests using every available technique have been reported in the literature, from skin tests to antibody and antigen detection assays. but cross-reactivity with other trematode infections is problem in areas where they coexist. Fasciola – specific ELISAs using partially purified fluke antigens are available.

The advantage of immunodiagnosis over parasitological techniques is that they can detect early, prepatent infections as well as chronic ones with little or no egg output. In contrast to other infections, levels of antibody in ELIAs appear to drop rapidly after successful treatment, so the assays tend to detect only active infection.
Intestinal flukes

Fasciolopsiasis

Fasciolopsiasis is caused by the giant intestinal fluke, *Fasciolopsis buski* it is in the same family as *Fasciola*, and its life cycle and morphology are similar. However Fasciolopsis infection is largely confined to Asian countries.

Life cycle

In contrast to *fasciola*, the final host range of *F. buski* is limited and many mammals are refractory. Humans and pigs become infected through the consumption of viable metacercaria attached to the seed pods of plants. Metacercaria are not present on the edible seed of these plants, ingestion occurs during removal of the pods with the teeth and lips. Metacercaria are also found free on the surface of ponds, and infection may occur from drinking water.

*F. buski* excysts in the duodenum and the escaping larvae attach to the duodenal and jejunal wall. The larvae become mature adults in 3 months and produce large numbers (an estimated 10000 – 25000 per day per worm) of large, yellow, operculated eggs if these eggs reach water sources, further development and embryonation occurs over 3-7 weeks, then miracidia hatch and enter snail intermediate host (family planorbidae). After multiplication as sporocysts and redia, free- swimming cercaria attach and encyst on seed pods.
Pathogenesis and pathology

Eosinophils accumulate at the site of parasite attachment on the jejunal or duodenal wall where mechanical injury and inflammation bleed due to formation. These ulcers sometimes bleed due to capillary damage or become abscesses. Mild infection in healthy people is associated with lower haematocrit and serum levels of vitamin B_{12} but no apparent change in other nutrients. This may result from parasite sequestering of vitamin B_{12} or its impaired absorption from the damaged intestinal mucosa. Although a few parasites cause little damage, the presence of many (hundreds to thousands) is associated with severe pathology and sometimes acute intestinal obstruction. Extensive intestinal ulceration may interfere with digestion, and cause malabsorption, leading to severe malnutrition and wasting. Oedema also occurs in severe cases it may result from toxic parasite metabolites. Allergic reaction or from hypoalbuminaemia secondary to electrolyte and protein imbalance from chronic malabsorption.

Clinical Features

Symptoms are generally absent or mild, and may include: diarrhea, hunger pains, flatulence, poor appetite, mild abdominal colic, vomiting, eosinophilia and fever. The abdominal pain may mimic that of peptic or duodenal ulcer. Late, severe cases present with ascites or oedema of the face, abdomen and legs, anaemia, anorexia, weakness and vomiting and patients may pass stools containing large amounts of undigested material. Deaths have been reported.
Diagnosis and investigations

Diagnosis by faecal examination is not difficult, given the large quantity and large size of the eggs. Stoll’s dilution, formalin ether concentration, direct smears and kato techniques have been used successfully. Differentiation from Fasciola eggs is difficult so that a dietary and clinical history should also be considered.

Lung Flukes

Lung Flukes *Paragonimus westermani*

**Paragonimiasis**

These flukes are distributed widely, and infect an estimated ten million people in China alone. Infection can cause severe respiratory or cerebral disease, depending on intensity, duration and site where the parasites become lodged.

**Life cycle**

*Paragonimus* lives in the lungs of some mammals—mainly wild and domestic cats, but also dogs, monkey humans. Parasite eggs are coughed up from the lungs and either expectorated in the sputum, or swallowed and excreted in the faeces. When these eggs reach water, further development occurs until miracidia hatch; they penetrate a snail host (mainly species of Thiaridae and Pleuroceridae). Multiplication and development occur in the snail until cercariae are produced. These may enter the second intermediate host, namely crabs, crayfish and one species of shrimp, while free-swimming or during
consumption of the snail by the crustacean. The cercaria encyst in the gills, liver and muscle tissue of the crab and require about 2 months to become infective metacercaria. When the crabs are eaten by a final mammalian host, the cysts excyst in the small intestine, penetrate the intestinal wall, travel from the peritoneum to the subperitoneal tissue, passing through the diaphragm to the lungs- where they mature in 2 months. Adult worms may live for 20 years. Due to aberrant migration, larvae sometimes become lodged in ectopic sites, e.g. brain, abdomen, groin, skin or heart. In some mammals which serve as paratenic hosts, the larvae exit the small intestine and lodge without further development in muscle tissue. When a final mammalian host consumes the paratenic host, the larvae survive passage through the stomach and escape through the intestinal wall. Migration to the lungs then results in maturation and egg excretion.

**Pathogenesis and pathology**

The site of pathology of paragonimiasis depends upon the migratory route of the larvae and the tissue in which they lodge. Inflammatory responses to the adult worms, immature worms and eggs are similar, regardless of location. Beginning with leucocyte (mainly eosinophil) infiltration and finally resulting in thick cysts or abscesses and ultimately calcification. The parasites live within these fibrotic, grayish – white capsules (1.5-5 cm in diameter) in pairs or triplets, surrounded by thick, blood-streaked fluid and numerous eggs. Later, the capsules may be empty or fluid filled. Eggs trapped in the tissue may also provoke granulomas at the periphery of the necrotic area. The capsules occur most frequently in the upper right quadrant of the lungs and, in the fewer cases of cerebral involvement, the posterior portion of the brain is usually affected. In both human and experimental
infections there appears to be a subsidence of inflammation in later periods. This may be due to modulation of the immune response.

**Clinical features**

Pulmonary infection is accompanied by a chronic productive cough, with brownish purulent sputum containing streaks of blood and parasite eggs in *P. westermain*; the signs mimic bronchiectasis, bronchopneumonia, or tuberculosis not responding to antibiotics.

**Diagnosis and investigations**

The clinical signs are fairly pathognomonic, particularly when diet history and residence in an endemic area are known, but misdiagnosis is commonly due to unfamiliarity with the disease. Tests to rule out tuberculosis, skin tests and sputum smear and culture are helpful in differentiation. Eggs of *paragonimus* may be found in the faeces. Sputum, gastric washings or tissue. Blood in the sputum contains sufficient eggs to be visualized by direct smear. Sputum without blood should be collected over 24 hours, centrifuged and the sediment dissolved in 3% sodium hydroxide for examination for eggs. Eggs may not be present in the sputum of infected children or elderly people, but faecal examination using a concentration technique may reveal eggs. Adult worms might be expectorated after treatment or found in skin lesions. Skin and immunodiagnostic tests using parasite antigens are highly sensitive and useful for surveys and in diagnosing the infection(s). Complement fixation tests and ELISA detect early as well as chronic infection and titres decline rapidly (becoming negative in 1-2 months) after cure. These tests therefore assist in assessing cure following treatment.