Fascioliasis (Chentsey)
It is a disease due to infestation by liver flukes, which infests principally sheep, goats and cattle although many other mammalian species are also susceptible.

Etiology

**Family:** Fasciolidae  
**Species:** *F. hepatica, F. gigantica, Fascioloides magna* (mammals and man), *Fasciolopsis buski* (pigs and man)  
**Characteristics:** large, leaf-like, suckers present  
**Hosts:** parasites of liver and bile ducts of herbivorous animals and man  
**Intermediate hosts:** fresh water snails  
**Diseases caused:** Fascioliasis (Acute and Chronic Fluke Disease)

Host range
- Sheep are highly susceptible.  
- Cattle, Goats are susceptible.  
- Buffaloes, horses, pigs less susceptible.  
- Deer acts as reservoir host.  
- Men act as accidental/unnatural host.

Transmission - Ingestion of metacercaria-contaminated grasses

Life Cycle

![The Life Cycle of Fasciola hepatica.](image_url)
Pathogenesis

**Acute fascioliasis**

During the early stages, large number of immature flukes of 6-8 weeks age migrate rather extensively through the tissues of the liver causing considerable mechanical destruction of the liver parenchyma with marked haemorrhage. Depending on the number of invasive flukes, rupture of the liver capsule may occur with haemorrhage into the peritoneal cavity. Animals may die within a few days of the onset of severe clinical signs. Liver becomes enlarged, pale and friable and shows numerous haemorrhagic tracts on the surface. Fibrinous clots on the liver surface and throughout the peritoneal cavity are seen. Small flukes of 0.7-2 mm can be squeezed from the cut surface or obtained from the excess of peritoneal fluid.

**Chronic fascioliasis**

It is the most common form of the infection in sheep, cattle and other animals. This type occurs commonly in light and repeated low dose infections. In long standing chronic hepatitis, there is inflammation of tissues including that of bile ducts which become fibrosed and thickened with proliferation of epithelial cells. In heavy infections, fibrosis and inflammation spread to almost the whole of the liver, the bile ducts show hypertrophy and calcification and the whole organ becomes hardened. In cattle, the walls of the bile ducts are commonly calcified and protrude markedly from the surface and are difficult to cut with a knife. They resemble the stem of a clay pipe, giving the common name of pipe-stem liver to the infection.

The anaemia in fascioliasis is associated primarily with intrabiliary haemorrhage due to the blood-sucking activity of the adult parasites. The calculated blood loss is approximately 0.5 ml/day/fluke.

**Clinical signs**

**Acute form** - seen invariably in sheep

- High mortality
- Haemorrhages and even bleeding from natural orifices.
- Diarrhoea in heavy infection.
- Development of edema, severe anemia.
- Painful at the region of liver in abdomen on palpation.
- Sub acute form - occurs in all spp. except horses, pigs.
- Enteric signs like dysentery
- Anemia, edema
- Fast decrease in production, anorexia, rough hair coats, etc.

**Chronic form** - commonly seen in field and is endemic. The affected animal is inactive and shows:

- distended abdomen
- ascites
- loss of appetite
- black scouring and persistent loose motion.
- oedema, and bottle-jaw due to the presence of watery swellings under the mandible.

The animal becomes gradually weak resulting in production loss

**PM findings**

Enteritis, haemorrhages in peritoneum, pipey liver in chronic cases, gritty sound when liver is cut due to calcification, plenty of flukes in bile ducts, etc.

**Diagnosis**

A tentative diagnosis can be made based on the following:

- History of grazing in snail-infested areas.
- The clinical manifestation of the disease.

Diagnosis can be confirmed by finding the eggs in the faeces. In size, shape and character, the eggs of *Fasciola* resemble those of some Amphistomes. The eggs of *Fasciola* have a yellow-tinge shell with an indistinct operculum and embryonic cells. The amphistomes eggs have a transparent shell and distinct opercula and embryonic cells, and frequently with a small knob at the posterior pole. The amphistome eggs are generally larger than of *Fasciola*. It is best if the faecal samples are taken from the rectum at about mid-day when the eggs are discharged maximum.
Differential diagnosis

- Fascioliasis in sheep needs to be differentially diagnosed from Anthrax.
- Chronic Fascioliasis needs differential diagnosis from Paratuberculosis, haemorrhagic septicaemia, haemonchus, etc.

Treatment

1. Triclabendazole is the drug of choice for fascioliasis, effective against all stages of the parasite, when given @ 12 mg/kg BW in sheep and 10 mg/kg BW in cattle.
2. Oxyclozanide @10-15 mg/kg orally in cattle and at 15-20 mg/kg in sheep is highly effective against mature flukes. The dose may be increased to three times (45 mg/kg BW) in treatment against immature flukes (acute fascioliasis).
3. Rafoxanide @ 7.5 mg/kg is 99% effective against mature flukes, 98% effective against six-week old flukes and 50-90% effective against 4-5 weeks old flukes.
4. Closantel @ 10 mg/kg orally is also used in sheep and cattle
5. Albendazole @ 7.5 mg /kg in sheep and at 15 mg/kg in cattle has greater than 90% efficacy against adult flukes.

Control

The control of the parasite is dependent upon three factors:
1. destruction of the snail which serve as the intermediate hosts,
2. treatment of the infected livestock , and
3. Control of wild mammals that serve as reservoir hosts.

The number of snails can be reduced by proper drainage and by removal of vegetation on which the snails feed. The snails can be destroyed by 0.5 % copper sulphate spraying over the region (approx. 9 kg CuSO₄ /acre required) and other molluscicides like sodium pentachlorophenate and rearing of ducks. Avoid the animals from grazing in the marshy areas/stagnant water sources.

All the animals that are infected with Fasciola should be treated twice a year in a systemic way. A few untreated infected animals, may spread the infection quickly to other animals provided the intermediate hosts are available. The control measures against the snails and treatment of the infected animals should be carried out simultaneously in a given locality.

A number of wild animals serve as reservoir hosts for Fasciola, and as long as these carry the infection, it is difficult to control fasciolosis effectively.

Vaccination - irradiated young fluke vaccines orally being studied as a prophylactic measure against fascioliasis.
Amphistomiasis

Stomach flukes are parasites of ruminants which particularly affect cattle and sheep. The flukes are often found in small numbers but only seriously affect livestock under certain conditions, in heavy infections, and in certain growth stages. The disease is characterized by severe enteritis. Young cattle are the usual subjects of the disease. Heavy infection with immature flukes in the upper intestine can cause serious ill-health and death.

**Etiology**

**Family:** Paramphistomatidae  
**Genus:** Paramphistomum, Cotylophoron (rumen flukes)  
**Species:** Paramphistomum cervi, Cotylophoron cotylophoron, Calicophoron calicophoron  
**Characteristics:** The adult parasites are pear-shaped, pink or red, up to 15 mm long, and attach to the lining of the rumen. Immature forms are found in the duodenum and are 1-3 mm long.  
**Disease:** Paramphistomiasis or amphistomiasis

**Life cycle**

Eggs are passed in the feces, and miracidiae hatch in the water and infect planorbid or bulinid snails. Development in the snail is similar to that in the life cycle of *Fascioloides hepatica*, with the snail shedding cercariae that encyst on the herbage. In the ruminant host, the young flukes excyst and remain in the small intestine for 3-5 wk before migrating forward through the reticulum to the rumen. Eggs are produced 7-14 wk after infection.

**Pathogenesis**

Adult flukes do not cause overt disease, and large numbers may be encountered. The immature flukes attach to the duodenal and, at times, the ileal mucosa by means of a large posterior sucker and cause severe enteritis, possibly necrosis, and haemorrhage. Affected animals exhibit anorexia, polydipsia, unthriftness, and severe diarrhoea. Extensive mortality may occur, especially in young cattle and sheep. Older animals can develop resistance to reinfection but may continue to harbor numerous adult flukes. The large, clear, operculated eggs are readily recognized, but in acute paramphistomiasis there may be no eggs in the feces. Known occurrence in the area and examination of the fluid feces may reveal immature flukes, many of which are passed in these cases.

**Clinical signs**

- Characteristic and persistent fetid diarrhoea accompanied by weakness, depression, dehydration and anorexia.  
- Submaxillary oedema  
- Visible paleness of the mucosa.  
- Death usually occurs 15-20 days after the first signs appear.

**Lesions (necropsy findings)**

- Subcutaneous oedema and accumulation of fluid in the body cavities.  
- Gelatinous fats depot.  
- Mucosa in the upper part of the duodenum thickened, covered with blood-stained mucus,  
- Patches of haemorrhage under the serosa.  
- Small, flesh coloured flukes are present.  
- Microscopically, immature flukes in the mucosal surface and deeper layers.

**Diagnosis**

- Clinical signs, in environmental conditions suitable for the propagation of flukes and where host snails can be found should arouse suspicion of intestinal amphistomiasis.  
- The large, clear, operculated eggs are easily recognized, but in acute paramphistomiasis there may be no eggs in the faeces.  
- Confirmation depends on demonstration of immature flukes in faeces or at necropsy.

**Differential Diagnosis**

- Nutritional deficiency of copper
- Intestinal roundworm infestation
- Johne’s disease in adult animals
- Weeds poisoning
- Arsenic and lead poisoning

**Treatment**

Although treatment for adult fluke has no direct benefit to the animal, it may reduce the source of infection for the snail intermediate host. This then reduces the size of the next generation of infective fluke larvae on pasture. Treatment with an appropriate drench should be timed for autumn and spring.

Effective treatment of immature fluke infection requires the combination of following measures:

1. Removal of stock from the source of infection, usually swampland.
2. Treatment with a drench which is effective against immature fluke:
   a. Oxyclosanide @ 18.7 mg/kg two days apart (two doses) give consistent result against immature amphistomes in cattle
   b. Hexachlorophene @ 20 mg/kg SD. But may show toxicity at this rate.
   c. Niclosamide @ 160 mg/kg SD or two doses at 3 days apart is effective in cattle.
   d. Niclsoamide @ 100 mg/kg, effective against immature amphistomes in sheep
3. Others- Resorantel, combination of bithional and levamisole
4. Supportive therapy to treat dehydration and any secondary bacterial infection may be needed.

**Control**

1. Remove animals from infected pasture during outbreak
2. Metacercariae may persist on pasture for 2 or 3 months after flood water has dried out and susceptible animals should be kept away during the period of risk.
3. Treatment between the seasonal peaks.
4. Destruction of host snails by the use of molluscicides
5. Drainage and fencing of affected areas
6. Provide alternative source of water
7. Treat stock with drenches effective against stomach fluke

**Distribution of Rumen and Gastro-intestinal flukes in developing countries**

<table>
<thead>
<tr>
<th>Distribution of Rumen and Gastro-intestinal flukes in developing countries</th>
<th></th>
<th></th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td></td>
<td></td>
</tr>
</tbody>
</table>
Schistosomiasis

Schistosomes are thin, elongated flukes, up to 30 mm long that live in blood vessels of the final host. The female lies in a longitudinal groove of the male. Various water snails act as intermediate hosts. The schitosomes, or blood flukes, affect both humans and animals, causing serious diseases worldwide. The adults of most schistosomiasomes live in portal and mesenteric veins, others reside in pelvic veins. Schistosoma nasalis reside in veins of the nasal cavity.

Etiology

Family: Schistosomatidae

Species:
Important to animals: Schistosoma bovis (cattle and sheep), S.nasalis (cattle), S.indicum (horses, cattle, goats);
Important to human are: S. mansoni, S.haematobium, S japonicum.

Identification:
• Sexes are separate,
• Adult found in veins of digestive and urinary tract of mammals and birds.
• Eggs lack operculum and contain a fully developed miracidium when discharged in faeces or urine.
• Infection acquired through skin penetration.

Table 1. Important schistosomes of humans and animals

<table>
<thead>
<tr>
<th>Species</th>
<th>Host</th>
<th>Location</th>
<th>Remarks</th>
</tr>
</thead>
<tbody>
<tr>
<td>Schistosoma bovis</td>
<td>Cattle, sheep, goat, horse, mule, antelope</td>
<td>Portal and mesenteric veins</td>
<td>Africa, India</td>
</tr>
<tr>
<td>S. haematobium</td>
<td>Humans, monkey</td>
<td>Pelvic veins</td>
<td>Africa, Western Asia, Europe, Australia</td>
</tr>
<tr>
<td>S. incognitum</td>
<td>Dog, pig</td>
<td>Mesenteric and portal veins</td>
<td>India</td>
</tr>
<tr>
<td>S. indicum</td>
<td>Cattle, sheep, goat, horse, camel</td>
<td>Mesentric, portal and pelvic veins</td>
<td>India</td>
</tr>
<tr>
<td>S. nasalis</td>
<td>Cattle, goat, horse</td>
<td>Nasal veins</td>
<td>India</td>
</tr>
<tr>
<td>S. spindale</td>
<td>Cattle, sheep, goat</td>
<td>Mesenteric and portal veins</td>
<td>India</td>
</tr>
</tbody>
</table>
Life cycle

Most species of pathogenic schistosomes are found in the hepatic portal system, and the principal clinical signs are associated with passage of the spined eggs through the tissues to the gut lumen. One species, *S nasale*, is found in the veins of the nasal mucosa of ruminants and horses, where it may cause coryza and dyspnea. Eggs passed in the feces must be deposited in water if they are to hatch and release the miracidia, which invade suitable water snails and develop through primary and secondary sporocysts to become cercariae. When fully mature, the cercariae leave the snail and swim freely in the water, where they remain viable for several hours. The cercariae invade the final host through the skin and mucous membranes; during penetration, cercariae develop into schistosomula, which are transported via the lymph and blood to their predilection sites. The prepatent period is ~6-9 wk.

In Asia, *S spindale*, *S nasale*, *S indicum*, *S incognitum*, and *S japonicum* are widespread in livestock. The latter species is of particular importance because livestock form a reservoir for the disease in humans. Four species of the related genus Orientobilharzia also infect livestock in Asia—*O turkestanicum*, *O harinasutai*, *O dattai*, and *O bomfordi*.

Fig. 298-1. Life cycle of schistosomiasis.
Pathogenesis
Damage to the host can result from three factors:
1. From the invading cercariae
   - Penetration of the skin by the cercariae → inflammation in man and animals within few hours.
   - This is known as “cercarial dermatitis” / “swimmer’s itch” / “swamp itch”
2. From the presence of adults in the veins
   - Adult blood flukes in the veins → phlebitis & sometimes venous thrombosis
   - Severe vascular lesions when adult worms die or are trapped
3. From the presence of ova in veins or tissues
   - Ova - most important factors → they have terminal spines.
   - Ova in the venules reach venous capillaries. They adhere to the endothelium and become embedded within it.
   - Ova also rupture the capillaries.
   - Migration of ova from blood vessel → lumen of intestine / urinary bladder → haemorrhagic ulcers → haemorrhage, anaemia & hypoproteinaemia.
   - Many eggs get embedded in tissues and stimulate an extreme immunological response

Clinical findings
Hemorrhagic enteritis, anemia, and emaciation, which develop after the onset of egg excretion, are the major clinical signs associated with the intestinal and hepatic forms of schistosomiasis in ruminants. Severely affected animals deteriorate rapidly and usually die within a few months of infection, while those less heavily infected develop chronic disease with growth retardation. Many older cattle in endemic areas of Africa have an effective level of immunity against reinfection. Nasal schistosomiasis is a chronic disease of cattle, horses, and occasionally buffalo. In severe cases, there is a copious mucopurulent discharge, snoring, and dyspnea; milder cases frequently are asymptomatic.

Lesions
In the intestinal and hepatic forms, adult flukes are found in the portal, mesenteric, and intestinal veins. However, the main pathologic effects are associated with the eggs. In the intestinal form, passage of eggs through the gut wall causes the lesions, while in the hepatic form, granulomas form around eggs trapped in the tissues. Other hepatic changes include hypertrophy and hyperplasia of the portal veins, development of lymphoid nodules and follicles throughout the organ, and periportal fibrosis in more chronic cases. Extensive granuloma formation also is seen in the intestine. In severe cases, numerous areas of petechiation and diffuse hemorrhage are seen in the mucosa, and large quantities of discolored blood may be found in the intestinal lumen. Frequently, the parasitized blood vessels are dilated and tortuous. Vascular lesions also may be found in the lungs, pancreas, and bladder of heavily infected animals.

In nasal schistosomiasis, adult flukes are found in the blood vessels of the nasal mucosa, but again, the main pathogenic effects are associated with the eggs, which cause abscesses in the mucosa. The abscesses rupture and release eggs and pus into the nasal cavity, which eventually leads to extensive fibrosis. In addition, large granulomatous growths are common on the nasal mucosa and occlude the nasal passages and cause dyspnea.

Diagnosis
Clinical history and signs are insufficient; the characteristic, terminal-spined eggs must be identified in the feces, rectal scrapings, or nasal mucus for confirmation. Eggs of S bovis (202 × 58 µm) are spindle-shaped; those of S spindale (382 × 70 µm) are more elongated and flattened on one side, and those of S nasale (456 × 66 µm) are boomerang-shaped. The oval eggs of S japonicum are relatively small (81 × 63 µm), with a rudimentary spine. In chronic cases, it may not be possible to find eggs in the feces or nasal mucus, and the diagnosis must be confirmed at necropsy by finding adult flukes in the blood vessels.
**Treatment and control**

Praziquantel (25 mg/kg) is highly effective, although 2 treatments 3-5 wk apart may be required. Transmission of the infection can be reduced by large-scale chemotherapy campaigns, by control of the intermediate snail host using molluscicides (e.g., niclosamide) or habitat modifications, or by fencing off contaminated bodies of water and providing clean drinking water. These measures not only help reduce the transmission of schistosomiasis but also help control other parasitic trematodes such as Fasciola gigantica and Paramphistomum spp, which similarly have water snails as intermediate hosts and frequently occur in the same localities as schistosomes. Antischistosome vaccines are under development.

**Nasal granuloma, snoring disease**

This is caused by *S. nasalis*. The parasites are found in the nasale veins of cattle, buffalo, sheep and goat. Cattle suffer from more severe lesions. In buffaloes the disease is subclinical and there is no granuloma formation.

Incubation period: 3 months

Intermediate host: Indoplanorbis species of snails. Example,

Eggs of fluke enter mucous gland of the nasal cavity evoking a cellular reaction with formation of abscesses.

Rupture of abscess liberates --------- fibrosis and cauliflower-like growth.

Affected animals show rhinitis, mucopurulent discharge. There is dyspnoea and snoring.

Snoring disease in cattle adversely affect the production, body weight gain, and draught capacity of the bullocks.

**Treatment**

1. 2 % Sodium antimony tartarate @ 2 mg/kg in dextrose saline for 4 days
2. 6 % (w/v) Lithium antimony tartarate (Anthiomaline®) @ 20 ml deep intramuscular repeated every 3 months in endemic area.
3. Praziquantel @ 20 mg/kg s/c x 3 days, or @ 60 mg/kg as single dose.
Dicrocoeliasis

**Etiology**

**Order:** Dicrocoeliidae  
**Characteristics:** translucent body  
Parasites located in gall bladder, bile and pancreatic ducts of mammals, birds and reptiles  
**Species:** Dicrocoelium dendriticum  
**Hosts:** sheep, cattle, pigs, deer and other mammals. Most important in sheep  
**Intermediate host:** Terrestrial snail (*Cionella lubrica*) and ant *Formica fusca* (metacercaria)  
Found in bile ducts of sheep, goat, cattle, horses, pig, dogs, and man.  
Found in dry and hilly region.

**Life cycle**

Involves two intermediate hosts.  
Eggs ingested by a land snail.  
It develops to sporocyst I and II and then to cercaria which comes out of the snail. Cercaria is ingested by ants of genus *Formica* where it develops to metacercaria.  
Ingestion of ants by definite hosts results infection.

**Symptoms and lesions**

Liver and bile ducts are distended with dark worms inside.  
Cirrhosis of liver  
Bile ducts become cystic

**Diagnosis**

Confirmative diagnosis by demonstration of eggs in the faeces

**Treatment**

Praziquantel  
Benzimidazole

**Control**

Control of land snails  
Control of ants- ploughing of land reduces ant load in pasture