

Fasciolosis in livestock

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abstract

across the globe livestock industry is heavily affected by fasciolosis and it has adverse effect country's growth. It is present in almost every continent of the earth. Production of animal gets reduced by the infection of present fluke and there are chances of transmission of infection to human also. Current article provides us brief information about some crucial aspect of *Fasciola* spp such as morphology, life cycle, pathogenesis, control and treatment aspect.

Introduction

In 2019 India had about 535.8 million numbers of total livestock, which consist of 302.3, 74.3 and 148.9 million of bovine (cattle and buffalo), sheep and goat respectively (<https://www.nddb.coop/information/stats/pop>). These animals are used for both milk and meat purpose and due to great diversity in their habitat suffer from wide variety of parasitic disease, out of which Fasciolosis plays a crucial role. It is one of the serious helminthic diseases which occur in hilly as well as plain region. This disease can reduce the productive potential of animal and can cause huge loss to country economy and farmer income. Infection this parasite can also infect human. In this popular article we have discussed general aspect of parasite including its morphology, life cycle, pathogenesis, control and treatment aspect.

General structure of *Fasciola* species

Fasciola hepatica mainly occur in bile duct of small and large ruminants, dogs, horse, man, cat etc. It is leaf like in shape and is greyish brown in colour. anterior portion is broad than posterior region. Cone shape projection is present in anterior region followed by well develop shoulder. anterior sucker is present on cone and ventral sucker is present at level of shoulder. Body is covered by spiny tegument, intestinal caeca is highly branched, testes and ovary is also branched, vitelline gland is present in lateral region (Soulsby 1982, Bhatia et al 2016). another important species is *Fasciola gigantica* whose general difference and structure

is given in latter section. *F. hepática* is mainly found in Himalayan region and *F. gigantea* is found in Meghalaya, andaman and Nicobar, Jammu and Kashmir, Sikkim, Haryana, Uttar Pradesh, Punjab, Bihar, Maharashtra, Gujarat, Madhya Pradesh etc (Bhatia et al 2016).

appearance of fluke



Fasciola hepática (abdisa 2017)



Fasciola gigantea (abdisa 2017)

Difference between 2 important species of Fasciola

S. no	<i>Fasciola hepática</i>	<i>F. gigantea</i>
1	Found in hilly area (temperate)	Found in plain area (tropical)
2	Size small	Size is large
3	anterior cone is large	anterior cone is smaller
4	Shoulder are broad and prominent	Shoulder are not prominent
5	Body is grayish brown	Body is more transparent
6	Ovaries, testes and intestinal caeca are more branched	Ovaries, testes and intestinal caeca are less branched
7	Egg is small (130-150 by 63-90 um)	Egg is large (156-197 by 90-104 um)
8	Snaíl is amphibious (<i>Lymnaea truncatula</i>)	Snaíl is aquatic (<i>Lymnaea auricularia</i> , <i>L. acuminata</i> [Indian subcontinent])
9	Development in snaíl is shorter duration as compared to <i>F. gigantea</i>	Development in snaíl is longer duration as compared to <i>F. hepática</i>
10	Control is easy because IH is amphibious	Control is difficult because IH is aquatic

Life cycle

adult fluke are present in the DÍ i.e. definitive host (sheep, goat etc). Fertile egg comes out of fluke body and via bile reaches to the duodenum of DÍ. at appropriate humidity and temperature, egg hatch in the environment and produce 1st larval stage i.e. miracidium. With the help of boring action of anterior spine and secretion of apical gland, miracidium penetrate into the body of IH i.e. intermediate host (snail). It removes its ciliated outer covering and form sporocyst (2nd larval stage), after some time sporocyst form 5-8 redia (3rd larval stage), these redia have ring like thick part at pharynx region and 2 blunt process at hind end. Redia then form cercaria (4th larval stage), it has a rounded body with no eye spot and a tail of twice in length. Cercaria then leaves the body of snail and swim in water body with help of tail. It then crawl on local water vegetation and with the help of secretion secreted by cystogenous gland a covering is formed on body, tail is casted off and it turn into metacercariae (5th larval stage). This metacercariae is ready for infection to DH. When DH ingests this vegetation, metacercariae reach to gastro-intestinal tract (GIT). Excystation (removal of outer covering) take place in duodenum. Host factors such as trypsin, pancreatin, and cholesterol help in the removal of covering. Juvenile fluke are released in GIT, within 24hrs via penetration of intestinal wall reaches to abdominal cavity and at last penetrate liver capsule. after penetration they first migrate in parenchyma and reach to bile duct and form adults (Soulsby 1982, Bhatia et al 2016).

Pathogenesis

(1) *acute fascioliasis* - This condition is caused due to simultaneous migration of large number of immature fluke in liver. It is mainly observed in late summer due to presence of higher cercarial load on vegetation. Migration of juvenile fluke cause marked haemorrhage and massive destruction of parenchyma. They also feed on hepatic cells. In some case liver capsule also get rupture due to higher parasitic load and haemorrhages are also formed in peritoneal cavity. Sudden death of animal may occur; post mortem examination shows enlarged liver with pale haemorrhagic tract on liver surface. Sometime fibrinous clot can also been seen on hepatic surface and peritoneal cavity. Numerous immature flukes can also be observed, acute fascioliasis lead to the formation of necrotic lesion and it potentiate proliferation of Gram-positive, endospore-forming anaerobic bacteria i.e. *Clostridium oedematians novyi* and lead to "black disease".

(2) **Chronic fascioliasis**- It leads to 2 types of condition i.e. hepatic fibrosis and hyperplastic cholangitis

(a) **Hepatic fibrosis**- as discussed above that migration of juvenile fluke lead to haemorrhage, necrosis and destruction of liver parenchyma moreover migration of fluke cause the formation of thrombus in hepatic vein, as a result proper flow of blood get hampered and this result in formation of coagulative necrosis of parenchyma. after some weeks healing and regeneration of lesion begin, collagen is laid down and resulted in the formation of fibrosis. Many time contraction of scar tissue also occurs and it alters normal hepatic architecture. In order to restore normal architecture, band of fibrous tissue are formed and it interconnect migratory tract with normal tissue and thus lead to formation of lobules (Soulsby 1982, Bhatia et al 2016).

(b) **Hyperplastic cholangitis**- adult fluke mainly cause this condition. Hyperplasia of bile duct epithelium occurs along the side of fluke attachment. Suckers and spine of adult fluke denude the bile duct wall and it lead to local inflammation. Hyperplastic mucosa became more permeable to various protein mainly albumin, this along with blood sucking habits of adult fluke (blood loss @ 0.5ml/day/fluke) lead to the formation of hypoalbuminaemia and hypoproteinaemia in animal. Many time calcification of fibrotic lesion, walls of bile duct occur and lead to formation of pipe-stem liver. In rare cases hazel-nut-sized cysts are also formed due to parasite in other organs such as lungs (Soulsby 1982, Bhatia et al 2016).

Clinical sign

1. acute fascioliasis:

animal dies suddenly. Blood stained froth appear at the nostrils like in anthrax.

2. Chronic fascioliasis:

animal is off colour, followed by increasing anaemia. Lack of vigour, when the infected animals are driven for long and remains behind amongst the flock. appetite diminishes, mucous membrane pale, oedema. Skin is dry & doughy to touch. Hypoproteinaemia is seen that leads to bottle jaw condition. Marked constipation is seen in cattle. Diarrhoea in concurrent infections with ostertagiosis(Soulsby 1982, Bhatia et al 2016).

Diagnosis

Clínical sign, faecal examination for golden yellow egg with indistinct operculum, serological test such as ELISA, latex agglutination test. ELISA: Cathepsin L, **Bio K201** Sandwich ELISA kit has been developed for detecting *F. hepatica* (Belgium).

Treatment

Triclabendazole (10-12mg/Kg body wt effective against both form: *DOC for acute fasciolosis*, Oxytoclozanide (15mg/Kg body wt effective against mature): safe in milk (3 day withdrawal period), Rafoxanide (7.5mg/Kg body wt effective against mature), Bithionol (35-40mg/kg body wt against chronic form), Nitroxylin (10-15mg/kg body wt S/C effective against both form), Diamphenethide (100mg/Kg body wt good for acute fasciolosis), albendazole (7-15mg/kg body wt effective against mature form) (Soulsby 1982, Bhatia et al 2016).

Control measure

Destruction of IH by use of insecticide such as copper sulphate, sodium pentachlorophenate etc, rearing of snail eating ducts, use of spores of predacious fungi, segregation and treatment of infected animal (Soulsby 1982, Bhatia et al 2016). Control of snail by three ways:

1. Physical: Net in water channel in farms/ flow of water, destruction of breeding ground.
2. Chemical: Copper sulphate (1:100000) or 10-35kg/hectare, N-tritylmorpholine (0.45kg in 680litres/hectare), Cuprous chloride (5ppm), Niclosamide .
3. Biological: Duck and goose rearing, Fish (Black Carp), Nymphs of dragon fly, Predatory Prawn (*Macrobrachium vollenhoveni*), Water bugs (*Sphaerodema urinator*) and Plant extracts.

References

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