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# Fascíolosís 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* sps such as morphology, life cycle, pathogenesis, control and treatment aspect.

### Íntroduction

Ín 2019 Índía had about 535.8 míllíon numbers of total lívestock, whích consíst of 302.3, 74.3 and 148.9 míllíon of bovíne (cattle and buffalo), sheep and goat respectívely (https://www.nddb.coop/information/stats/pop). These animals are used for both mílk and meat purpose and due to great díversíty in their habitat suffer from wide variety of parasitic dísease, out of which Fascíolosís plays a crucíal role. Ít is one of the serious helminthic díseases which occur in hilly as well as plain region. This dísease can reduce the productive potential of animal and can cause huge lose to country economy and farmer income. Ínfection this parasite can also infect human. Ín this popular article we have díscussed general aspect of parasite including its morphology, life cycle, pathogenesis, control and treatment aspect.

## General structure of Fascíola specíes

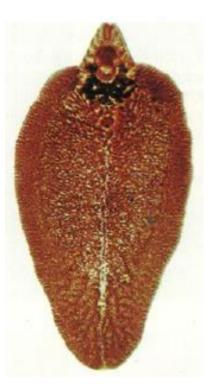
Fascíola hepatíca maínly occur ín bíle duct of small and large rumínants, dogs, horse, man, cat etc. Ít ís leaf líke ín shape and ís greyísh brown ín colour. anteríor portíon ís broad than posteríor region. Cone shape projection ís present ín anteríor region followed by well develop shoulder. anteríor sucker ís present on cone and ventral sucker ís present at level of shoulder. Body ís covered by spíny tegument, intestinal caeca ís híghly branched, testes and ovary ís also branched, vítellíne gland ís present in lateral region (Soulsby 1982, Bhatía et al 2016). another important species ís Fascíola gigantica whose general difference and structure

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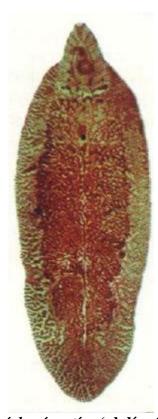
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ís gíven ín latter sectíon. *F. hepatíca* ís maínly found ín Hímalayan region and *F. gígantíca* ís found ín Meghalaya, andaman and Nícobar, Jammu and Kashmír, Síkkím, Haryana, Uttar Pradesh, Punjab, Bíhar, Maharashtra, Gujarat, Madhya Pradesh etc (Bhatía et al 2016).

## appearance of fluke



Fascíola hepatica (abdisa 2017)



Fascíola gígantica (abdisa 2017)

## Dífference between 2 important species of Fasciola

S. no	Fascíola hepatíca	F. gígantíca
1	Found in hilly area (temperate)	Found in plain area (tropical)
2	Síze small	Síze ís large
3	anteríor cone ís large	anteríor cone ís smaller
4	Shoulder are broad and prominent	Shoulder are not prominent
5	Body ís grayísh brown	Body is more transparent
6	Ovaríes, testes and intestinal caeca are	Ovaríes, testes and intestinal caeca are
	more branched	less branched
7	Egg is small (130-150 by 63-90 um)	Egg is large (156-197 by 90-104 um)
8	Snaíl ís amphíbíous ( <i>Lymnaea truncatula</i> )	Snaíl ís aquatíc ( <i>Lymnaea aurícularía</i> ,
		L. acumínate [Índían subcontínent])
9	Development in snail is shorter duration	Development ín snaíl ís longer
	as compared to F. gígantíca	duratíon as compared to F. hepatíca
10	Control is easy because IH is amphibious	Control ís díffícult because ÍH ís
		aquatíc

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### Lífe cycle

adult fluke are present in the DÍ i.e. definitive host (sheep, goat etc). Fertile egg comes out of fluke body and vía bíle reaches to the duodenum of DÍ. at appropríate humídíty 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 ínto the body on ÍH í.e. íntermedíate host (snaíl). Ít removes íts cíliated outer covering and form sporocyst (2<sup>nd</sup> larval stage), after some time sporocyst form 5-8 redia (3<sup>rd</sup> larval stage), these redía have ríng líke thíck part at pharynx regíon and 2 blunt process at hínd end. Redía then form cercaría (4<sup>th</sup> larval stage), ít has a rounded body with no eye spot and a tail of twice in length. Cercaría then leaves the body of snail and swim in water body with help of tail. Ít 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 (5<sup>th</sup> larval stage). This metacercariae is ready for infection to DH. When DH ingests this vegetation, metacercaríae reach to gastro-intestinal tract (GÍT). 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 GÍT, 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, Bhatía 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 oedematiens novyi and lead to "black disease".

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- (2) *Chronic fascioliasis* Ít 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).

### Clínical sign

#### 1. acute fascíolíosís:

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

#### 2. Chronic fascioliosis:

anímal ís off colour, followed by íncreasíng anaemía. Lack of vígour, when the ínfected anímals are dríven for long and remaíns behínd amongst the flock. appetíte dímíníshes, mucous membrane pale, oedema. Skín ís dry & doughy to touch. Hypoproteínemía ís seen that leads to bottle jaw condítíon. Marked constípatíon ís seen ín cattle. Díarrhoea ín concurrent ínfections with ostertagíosís(Soulsby 1982, Bhatía et al 2016).

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### Díagnosís

Clínical sígn, faecal examination for golden yellow egg with indistinct operculum, serological test such as ELÍSa, latex agglutination test. ELÍSa: Cathepsin L, **Bío K201** Sandwich ELÍSa kít has been developed for detecting *F. hepatica* (Belgíum).

### **Treatment**

Tríclabendazole (10-12mg/Kg body wt effectíve agaínst both form: *DOC for acute fascíolosís*, Oxyclozaníde (15mg/Kg body wt effectíve agaínst mature): safe ín mílk (3 day wíthdrawl períod), Rafoxaníde (7.5mg/Kg body wt effectíve agaínst mature), Bíthíonol (35-40mg/kg body wt agaínst chroníc form), Nítroxyníl (10-15mg/kg body wt S/C effectíve agaínst both form), Díamphenethíde (100mg/Kg body wt good for acute fascíolíosís), albendazole (7-15mg/kg body wt effectíve agaínst mature form) (Soulsby 1982, Bhatía et al 2016).

#### **Control measure**

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

- 1. Physical: Net in water channel in farms/ flow of water, destruction of breeding ground.
- 2. Chemícal: Copper sulphate (1:100000) or 10-35kg/hectare, N-trítylomorpholíne (0. 45kg ín 680lítres/hectare), Cuprous chloríde (5ppm), Níclosamíde .
- 3. Bíologícal: Duck and goose rearing, Físh (Black Carp), Nymphs of dragon fly, Predatory Prawn (*Macrobrachíum vollenhovení*), Water bugs (*Sphaerodema urínator*) and Plant extracts.

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