

Fascioliasis in Animals

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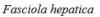
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Fascioliasis in animals are mainly caused by the digenetic trematode helminths *Fasciola hepatica* and or *F.gigentica*. the word 'gigentica' coin from its larger size (than *F. hepatica*) that measures 25-75 mm in length and upto 12 mm in breadth. In the tropical country like india, the fascioliasis in animals is predominanatly caused by *Fasciola gigentica* and its termed as "tropical fascioliasis". The condition is also called as "distomosis", "Liver rot" etc. in Hindi and Gujarati it is called as "Yakrut krumi", "Yakrut kida" and so on.







Fasciola gigantica

Figure.1. Image of two species Fasciola hepatica and Fasciola gigantica of genus fasciola.

The animals affected: The affected animals are mainly- bovines, bubalines, ovines and caprines. However, other species of animal's viz. canines, feline etc. may also affect. The disease is characterized by hepatitis, fibrosis of liver, loss of appetite, anaemia and poor production. The disease occurs in two forms, acute and chronic. In cattle, it is chronic in nature whereas in sheep, both the forms may occur. The immature fluke is more detrimental and cause more harm to the host by damaging the liver parenchyma due to migration which leads to the haemorrhage and necrosis of

liver parenchyma. The situation may more complicated by the invasion of secondary anaerobic bacteria- *Clostridium novyi*, which is also known as "black disease". Finally mature flukes settle in bile ducts of definitive host. The blood sucking behavior of the fluke further develops anaemia in host.















Figure. 2. Image showing different species of animals affect with *fasciola* infection.

The disease is also characterized by progressive anaemia and hypoalbuminaemia which result in pale mucus membrane, submandibular odema (Bottle jaw) and weakness in movements. Very often the affected animals remain behind the flock or isolated while grazing due to weakness.

Intermediate host: the snails act as intermediate hosts. In india the snails responsible are mainly Lymnea species, and they are L. accuminata, L.auricularia, L.rufescens, L.truncatula. in snail host, several asexual multiplicative stages are forms- first sporocyst stage developed in the tissue near the site of penetration (foot, antenna, gill). Thereafter radiae stage is migrated to glandular tissues (hepatopancrease and gonads) and culminating in the release of tailed carcariae.







nnea truncatula Lymnea auricularia

Figure.3. Intermediate host for F.hepatica and F.gigentica

How the disease transmits: F.gigentica has indirect life-cycle, involving mammalian definitive hosts molluscan intermediate hosts. Transmission between the two hosts occurs within water, via the formation of motile and encysted larval stages. Adults' flukes produce numberous eggs (up to 300 per days) which are shad in host faeces. The eggs emryonated in water ina few days to form miracidia, which hatch out in 9-10 days in warm weather (longer in cool weather). Miracidia actively seek snail hosts by chemotaxis and penetrate snail tissues wihin a few hours or die after 24 hours. The snails live in freshwater and/ or wet soils and survive dry periods by burrowing and aestivating. Once the miracidia penetrate a snail, they form mother sporocyst that lack digestive organs but feed by absorption. Sporocysts produce multiple daughter radiae by asexual reproduction. Radiae feed on snail tissues, eventually maturing to single tailed caercariae which borre their way out of the snail.carcariae being emerging 5-7 weeks after infectionand several hundred (sometimes thoussands) of cercariae may be produced. Parasites can also survive for months in aestivating snails buring in the soil during dry periods. Emergent cercariae swim to suitable substances (grass, leaves of aaquatics plants etc.) and form encysted metacercariae by shedding their tails and producing thick cyst walls. Metacercariae are quiescent infective stages which can survive on aquatic vegetation or in water for several weeks. Animals become infected when they ingest metacercariae with feed or water. Metacercariae excyst in the small intestines releasing juvenile worms which penetrate the gut wall and migrate around the body cavity for several days. They move to liver and burrow through the capsule into the parenchyma where they wander for 5-6 weeks before setting in the bile ducts. Worms become sexually mature and being producing eggs 8-13 weeks after infection. Adult's flukes can live for up to 10 years but mostly infections in domestic animals exhibit marks seasonal variation.

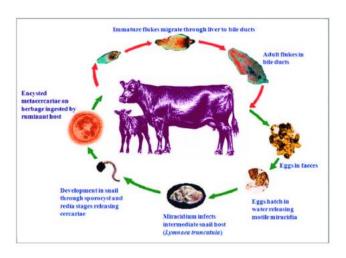


Figure.4. Life cycle of fascciola in animals

Pathogenesis: The several factors are responsible for the pathogenesis in fasciolosis. (a). The number of metacercaria ingested by animals. (b). The species of animals- The parasites is more pathogenic in sheep than another ruminant. (c). The age of animals-The young animals are less effective than old animals. Fasciolosis may be acute, subacute and chronic.

Acute fascioliosis: It is mainly found in sheep after 4-6 weeks of ingestion. This is caused by the immature flukes which migrate extensively in the liver parenchyma. As a result, there is extensive



A focal necrotic lesion in the liver.

damage of the liver parenchyma with profuse haemorrhages. This damages if also due to the feeding of hepatic cells by flukes. Death of the animals occurs due to internal haemorrhages, in heavy infection. Severe rupture of liver capsule, haemorrhagic tract on liver surface and fibrinous blood clot are found on liver and in peritoneal cavity. Complication of acute fasciolosis in sheep: Several species of the anaerobic bacillus like *Clostridium perfringens* or *C.novyi* may be present in the normal liver without causing any harm but they start to multiply rapidly in the infected liver with fasciola, finally causing the 'black disease', in sheep.







Profuse haemorrhages

Liver capsule

Fibrosis of liver

Sub-acute fascioliosis: This is found 8-10 week after infection. When young flukes reach the bile produce cholangitis. where pathognomic leason like haemorrhagic tract, subscapular haemorrhagic, hepatomegaly etc.

Chronic fascioliosis: It is the most common form of the infection in sheep, cattle and other animals. It occurs after 10-12 weeks after infection when adult fluke is developed. They cause cholangitis, obstruction of bile duct and fibrosis. The main pathogenic effects are anaemia due to loss of blood 0.5ml/fluke/day approximately hypoalbuminaemia due to utilization and retention of nitrogen. Hypoalbunaemia is also evident due to leakage of protien through the hyperplasic biliary mucosa and create a condition is known as "bottle jaw condition". A hyperplastic cholangitis is caused by the presence of the adult flukes in the bile ducts. In heavy infections, fibrosis and inflammation spread almost to 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. In cattle, sometimes the parasites are found in other organs especially lungs, they occur in hazel nut sized cyst containing brownish gelatinous material







Pipe stem liver condition

Bottle jaw condition

Hazel nut condition of lung

Figure. 6. Infected animal showing lesions during chronic fasciolosis.

in which dead/ calcified parasites can be seen is called hazel nut condition of lung (Rai, 2024).

Clinical Signs & Symptoms

✓ In acute condition: Infected animal becomes inactive, Weakness, Lack of

- appetite, Shows distended abdomen. Ascites, Death. In acute cases in sheep, the animal dies suddenly, blood-stained froth appears at the nostrils and blood is discharged from anus as in the case of anthrax.
- ✓ In sub-acute condition: Pale mucous membrane. Submendibular oedema. Enlargement of liver and Ascitis etc.
- ✓ Chronic condition: Stiff gait, Loss of appetite, Black scouring (Black color diarrhoea), 'Bottle jaw' due to the presence of watery swellings under the mandibles due to hypoalbunaemia., Anaemia, Pale mucus membrane, in cattle, digestive disturbances are more marked. Constipation is marked, Faecal are passed with difficulty, Diarrhea in extreme cases.

Epidemiology: The disease has been reported from different parts of the country with varying degrees of infection. Brahmbhat et al., 2021 recorded highest incidence of Fasciola infection was noticed in Jaffrabadi buffaloes (2.65%) than Gir cattle (1.72%) Junagadh, Gujarat. Yadav et al., 2007 recorded fascioliasis in 11.7% in cattle and 18.68% in buffaloes in tari region. In plain of northern India, Singh et al., 2009 reported fascioliasis in 19.3% cattle and 30.2% buffaloes in U.P and Gupta et al., 2011 found 2.20% in cattle 1.395 in buffaloes in M.P.

However, the snail Lymnea auricularia act as an intermediate host in indian condition. This infection occurs in animals grazing on areas with ponds, lakes dams or channels, source of fodder, nature of pasture, breed susceptibility and other factors also play an important role for the degree of infection. In heavy rainfall areas this disease is most prevalent. Highest prevalence of adult flukes occurs in wet season like June to October. Very low temperature >15C is not good for breeding snail as well as for the development of parasites. They usually prevalent in low lying plain area of tropical and subtropical regions (Sharma et al., 2011).

How to diagnosis: In chronic case (mature fluke) examination of faecal sample for presence of egg is useful. But for the diagnosis of immature fluke the same may be not effective. Copro-PCR may be useful for the purpose. The fasciola egg is having yellowish shell and indistinct operculum and embryonic cells. The fasciola egg can be easily distinguished from amphistome egg where later is



having distinguished operculum and embryonic cells.

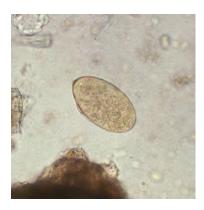


Figure. 5. Egg of Fasciola.

On post parterm examination, haemorrhagic lesions and burrowing/ migratory tracts may be seen on cut-section of liver. The adult fluke can be found lodging in bite ducts.

How to treat: A range of anthelmintics shows variable activity, including carbon tetrachloride (0.5 ml/Kg.b.wt.), triclabendazole (10-15 mg/kg b.wt), rafoxanide (7.5 mg/kg.b.wt), nitroxynil (10-15 mg/ kg. b.wt), niclofolan, closantal (1mg/66 kg.b.wt) and oxyclosanide (10-15 mg/kg.b.w), but their use may be contra- indicated under certain condition in certain animals. Therefor it is always advised to take treatment under the supervision of qualified veterinarian.

How to control the disease:

- 1. **Reduction of snail population** using copper sulphate or draining swampy field. Snail control is often difficult particularly in high rainfall areas where even temporary pools may harbor large snail populations (the aestivate in the ground during dry condition).
- 2. **Restriction of livestock** to aquatic vegetations and avoiding aquatic plants.
- 3. Fodder processing: If fodder come from water stagnant field, then the portion of the fodder which is well above the water level can be used as fresh fodder to the animals especially stall-fed animals. The fodder stalks which immersed in the water can also be fed to the animals in form of hay after exposing them to sunlight for one week (since the metacercariae cannot survive above 28° C). Making of silage of fodder from marshy land is another good option as heat generated in silo pit kills the metacercariae. Fasciola eggs can survive in dung pit for several months. So, compost the

- animal's dung at high temperature before using it as a natural fertilizer in the low land cultivation pasture field.
- 4. Chemical control: Used various chemical like copper sulphate 10-35mg/hect, Sodium pentachlorophenate 5-10 ppm in water, Ntritylomorpholine 45 kg/680 lt/hect, Ammonium sulphate or quick lime, Neem leaf and Eucalyptes leaf powder also mollacicidal effect
- 5. **Mechanical control:** Collecting of snail in mass and then destroying them with hard object **References**
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