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Popular Article

Bovine Fasciolosis: A Liver Fluke Disease

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Abstract

Fasciolosis is a disease caused by parasite known as *Fasciola.* The disease mainly seen in ruminants like cattle, buffaloes, sheep and goat and also elephants. Depending upon the animals there are various species of the parasite. The trematode fluke mainly affects the liver of the animal and produces various pathological conditions and as a result the animal become emaciated. In cattle, the chronic liver fluke disease is more common than the acute stage. The disease should be diagnosed and the treatment should be provided with proper anthelmintic. Otherwise, it will lead to serious economic loss to the farmers. Faecal sedimentation technique is the most commonly used diagnostic method for diagnosis of fasciolosis in field condition. Prevention of the disease is done by regular deworming of the animals and the control of snail intermediate hosts.

Introduction

Fasciolosis is basically a parasitic disease caused by a trematode of the family Fasciolidae. There are different species of Fasciola found in different animals which causes various pathological conditions particularly affecting the liver of infected animal. Fasciola have indirect life cycle which means they require second intermediate host for their development. The adult forms the parasite leave the intermediate host and encyst on plants to become the infective stage. The animal gets the infection by ingestion of contaminated grass. The infected animal will show clinical signs and symptoms depending upon which it is possible to diagnose the disease. On the other hand, there are some other diagnostic procedures also available which can be used to diagnose the disease. Treatment is done by using anthelmintic drugs specific for trematodes.

Etiology

The primary etiological agent of Fasciolosis in cattle is a trematode parasite named as *Fasciola sp.* There are two species of family Fasciolidae namely *Fasciola hepatica* and *Fasciola gigantica.* They are mainly flatworms having a dorsoventrally flattened body. They are also known as Diastomes. They possess anteriorly situated oral sucker or anterior sucker and posteriorly situated ventral sucker or acetabulum. The mouth is situated at the center of the oral sucker which leads to the pharynx and then to the esophagus. Esophagus ends in two blind ceca which are highly branched. These parasites do not have anus and they excrete their waste materials by regurgitation through mouth. The outer covering is known as tegument which possess spines. The excretory organ is known as Flame cells. *Fasciola hepatica* have broader shoulder than *Fasciola gigantca.* The ventral sucker is present at the level of shoulder and as large as oral sucker. Trematodes are hermaphrodites which means they have both the sexes in single individual. The fine follicular structures fill the lateral fields of the body of the parasite known as vitelline glands. The uterus lies anterior to the testes.

Life Cycle

Fasciola have indirect life cycle. They require intermediate host to become mature fluke. The eggs of the parasite are excreted along with the faeces of the host. The eggs hatch when the temperature is about 26°C. The time required for hatching of egg is about 10-12 days which produces the first larval stage called miracidium. Miracidium is broad anteriorly with a small papilliform protrusion. The tegument of miracidium is ciliated and the organism possess a pair of eye spot. After that the miracidium is ingested by the intermediate host. In case of *Fasciola,* the intermediate host is a snail of genus *Lymnaea.* There are various species of *Lymnaea* which acts as intermediate host of *Fasciola* in different geographical locations. In Europe, Asia, North America and Africa the species is *Lymnaea truncatula* and in Australia it is *Lymnaea tomentosa.* The intermediate host for *Fasciola gigantica* is *Lymnaea auricularia var.rufescens.*The further development of the parasite occurs inside the snail intermediate host. The miracidium further develops into sporocyst. Each sporocyst give rise to five to eight numbers of redia. The redia contains circular thickening behind the level of the pharynx and at the beginning of posterior quarter there is a pair of blunt process. The redia will finally become cercariae. They possess a tail of length twice than that of the body and there is no eye spot. In an around two hours, the cercaria settle on the blades of the grass and the tail casts off from the body of the cercaria. The secretions from the cystogenous glands of the cercaria will form the

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cyst of diameter 0.2mm. These cercaria now become infective which are ingested by the definitive host like cattle.

Following ingestion by definitive host, the excystation of the cercaria occurs in the duodenum of the definitive host. After excystation gradually the cercaria migrate into the abdominal cavity and then to the liver. Migration of the flukes in the liver parenchyma occurs in an around five to six weeks of infection and in seven weeks they begin to enter in the main bile duct. Gradually the cercaria reaches sexual maturity and starts producing eggs. In eight weeks of infection eggs can be detected in bile followed by in faeces. The favourable temperature for the development of the egg is 10-26℃. Below 10℃ there is no development of the eggs. For the development of snail intermediate host, the requirement is clear stagnant or slow-moving water with high oxygen content and abundant aquatic vegetation.

Pathogenesis

The pathological conditions that are produced in fasciolosis are related to the liver. The pathogenesis can be divided into acute or subacute and chronic type. The types of pathogenesis will depend upon the numbers of metacercaria ingested. The clinical signs and symptoms are different in both the cases.

Pathogenesis of Acute and Sub-Acute Fasciolosis

Acute fasciolosis is less common than that of chronic type. The main pathological condition that can be seen in acute fasciolosis is traumatic hepatitis occurs due to the migration of large numbers of immature flukes in the liver parenchyma which makes haemorrhagic tracts in the liver. If the numbers of immature flukes are more then it may cause rupture of liver capsule and bleeding occurs into the peritoneal cavity.

When the animal survives the acute fasciolosis then it may develop subacute fasciolosis in which the animal lose weight rapidly and the abdomen distended many folds.

Pathogenesis of Chronic Fasciolosis

Chronic fasciolosis is more common in cattle. When the infected animal consumes less numbers of metacercaria for a longer duration of time it causes chronic fasciolosis. The pathogenesis that may develop in chronic fasciolosis are of two types namely, Hepatic Fibrosis and Hyperplastic cholangitis.

When the immature flukes produce migratory tracts in the liver parenchyma during acute phase of the disease, with the advancement of time thrombus formation occurs in the hepatic veins

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and liver sinusoids and it produces a state of ischaemia followed by coagulative necrosis of liver parenchyma. Healing and regeneration of the affected area occurs via growth of collagen tissue and lead to Hepatic fibrosis. The hepatic architecture is destroyed when the scar tissue starts its contraction. To restore the normal architecture of liver fibrous tissue connects the migratory tracts to the normal tissues which divides the hepatic parenchyma into many lobules. In an around seven days after infection, lymphocytes, eosinophils and macrophages start migrating from hepatic vein into the surrounding tissues.

The other condition which will arise in chronic fasciolosis is known as hyperplastic cholangitis. This condition is caused by the presence of adult flukes in the bile ducts of the definitive host. Hyperplasia of the bile duct epithelium occurs and infiltration of eosinophils and monocytes in the lamina propria is a characteristic feature. The inflammatory reaction occurs in the bile duct epithelium as the suckers and spines of the adult fluke denude the epithelium. The parasite deposits its eggs in the smaller bile ducts and with the passage of time granulomatous reactions will occur to such eggs. Due to hyperplasia of the biliary system, the ducts become permeable to the plasma proteins and there is a leakage of proteins, particularly albumin. This is reason why hypoproteinaemia develops chronic fasciolosis. The other condition which is seen in chronic fasciolosis is the calcification of the bile duct wall. Due to the calcification the duct protrudes markedly from the surface which is difficult to cut with a knife and resemble the stem of a clay pipe. This phenomenon is known as Clay pipe stem liver. This is a characteristic finding of chronic fasciolosis in cattle. Moreover, *Fasciola hepatica* has been reported to increase the susceptibility of cattle to Salmonella Dublin infection.

Clinical Signs of Fasciolosis

Clinical signs are the major indications for the diagnosis of the disease. In cattle the disease may produce three types of clinical signs namely acute and chronic. In acute condition the animal may die suddenly and blood-stained froth may appear at the natural orifices similar to anthrax. The abdomen may be distended which is painful and there is severe damage to the liver. Anaemia is also produced due to blood sucking activity of the parasite.

In cattle the chronic case of fasciolosis is more common than the acute one. The major clinical signs that are developed in chronic fasciolosis are mainly anaemia, unthriftiness, anorexia. The mucous membrane become pale and skin become dull. Due to hypoproteinaemia submandibular oedema develop and this condition is known as **bottle jaw** which is a characteristic sign of chronic

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fasciolosis. General debility and emaciation are also produced due to the liver involvement. Constipation is common in cattle in which the animal passes faeces that are hard and brittle in nature. Diarrhoea is evident in extreme case. In calves' emaciation and weakness may lead to prostration.

Diagnosis of Fasciolosis in Cattle

The diagnosis of the disease is possible with the help of history and clinical signs. Moreover, faecal sedimentation test is done in the field condition to detect the eggs of the parasite in the faeces of infected animal with help of a microscope. The eggs should be differentiated from the eggs of paramphistomes. The *Fasciola* eggs have yellow shell with indistinct operculum and they are smaller than the eggs of paramphistomes. The shell in the eggs of paramphistomes are transparent and distinct operculum and they are large the differential diagnosis should be done with diseases like Haemonchosis, infectious necrotic hepatitis.

Treatment of Fasciolosis in Cattle

For the treatment of Fasciolosis there are some anthelmintic used which is giving excellent results. The drug of choice to treat the disease is Triclabendazole which is active against both mature and immature flukes. Oxyclozanide is an another anthelmintic which is effective against mature flukes. The other anthelmintic which can be used to treat fasciolosis are Rafoxanide, Nitroxynil, Albendazole. Along with the anthelmintic liver supportive should also be provided.

Prevention and Control of Fasciolosis in Cattle

Prevention of fasciolosis is essential as it causes economic loss to the farmers. Fasciolosis can be controlled by regular deworming of cattle. Moreover, the animals should not be allowed to graze in swampy areas as it is a major source of infection. Another method of control of snail intermediate host is the biological control where ducks and frogs are used which can eat the snails and thereby breaks the lifecycle of the parasite. Molluscicides can also be used to control the intermediate host but care should be taken so that it doesn't infect the waterbodies which is dangerous to the aquatic flora and fauna. Moreover, physical destruction of the snail can be done.

Conclusion

Fasciolosis is a common parasitic disease in ruminants especially which may cause serious economic loss to the farmer. So, prevention of the disease is better than treating it after the infection. To prevent veterinarian should be able to create awareness among the farmers regarding the disease and its consequences. The proper deworming schedule should be followed in every livestock farm

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to prevent the disease. Also, care should be taken so that animal can no graze on swampy areas where there is a great chance of getting the infection. The infected cattle should be treated as early as possible.

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