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General Review of Fasciolosis in Domestic Ruminants

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Abstract

Fasciolosis is a most widespread trematode disease of sheep, goats, cattle, and buffaloes and occasionally affects humans, thus considered as a zoonotic infection. *Fasciola hepatica* and *Fasciola gigantica* are the two liver flukes commonly reported to cause fascioliosis in ruminants. The life cycle of these trematodes involves snail as an intermediate host (IH) which belongs to the genus *Lymnea*. In acute infection, sudden death and severe anaemia were noticed without any evidence of fluke eggs in the faeces. Sub-acute and chronic infections are associated with weight loss, severe hypochromic macrocytic anaemia, hypoalbuminemia hyperglobulinemia, bottle jaw and ascites. Fasciolosis causes huge economic losses due to mortality, reduction in meat and milk, morbidity, impaired growth, reduction in carcass weight, wool growth and quality, decreased feed intake, conversion and lowered resistance. Diagnosis of fasciolosis is based on clinical signs, grazing history, seasonal occurrence, examination of faeces by laboratory tests, serology and post-mortem examination. The disease can be controlled by reducing the intermediate host population by drainage, fencing, molluscicides and by using anthelmintics.

Key words: Fasciolosis, *F.hepatica, F.gigantica, Lymnea, Metacercariae,*

Introduction

Fascioliosis stands out as one of the most severe and prevalent helminthic diseases affecting domestic animals in the Indian subcontinent. It inflicts substantial economic losses on the livestock industry through factors such as animal mortality, reduced vitality, liver condemnation, and decreased milk production. These losses are estimated to reach approximately 3.2 billion USD annually on a global scale (Mehmood et al., 2017). The culprits behind fasciolosis are Fasciola hepatica and F. gigantica, commonly known as "liver flukes." Fasciola hepatica is typically found in temperate regions, while F. gigantica has been identified as the cause of fasciolosis in tropical and subtropical areas, with the snail *Lymnaea auricularia* rufescens serving as its intermediate host (Thapar and Tandon, 1952).

Etiology

Fasciolosis is caused by two parasitic organisms known as Fasciola hepatica and F. gigantica. These parasites are classified within the Phylum: Platyhelminthes, Class: Trematoda, Subclass: Digenea, Order: Echinostomida, Superfamily: Fascioloidea, Family: Fasciolidea, Genus: Fasciola, and the Species: F. hepatica and F. gigantica.

Morphology

Fasciola hepatica is characterized by its leaf-shaped body, which has broad shoulders and a noticeable cone-shaped projection at the front. Its outer surface, or tegument, is equipped with sharp spines, and it typically measures around 5 cm in length. In contrast, F. gigantica is larger than F. hepatica, reaching lengths of up to 7.5 cm. Its body shape is more reminiscent of a leaf, featuring a short

conical anterior end without the prominent shoulders seen in F. hepatica.

When it comes to their eggs, Fasciola species produce oval, yellowish-brown eggs that lack any embryonic development and have an indistinct operculum. It's important to distinguish these eggs from those of other flukes, particularly the larger eggs produced by paramphistomum parasites.

Life Cycle

Adult flukes residing in the bile ducts expel eggs into the bile, which subsequently travels into the intestine and is expelled from the body through feces. These eggs, when passed in the feces, are underdeveloped and require at least 10 days to hatch under optimal conditions, typically at temperatures between 22-26°C. Below 10°C, their development is significantly limited.

Once hatched, the liberated miracidium, a small and actively swimming larva, has a brief lifespan of approximately 24 hours. Within this short window, it must quickly locate a suitable snail as its intermediate host. Successful penetration of the snail's tissue occurs within 3 hours. Inside the infected snail, the miracidium progresses through various developmental stages, including sporocysts and rediae, ultimately reaching the final stage, the cercaria. These cercariae are released from the snail, attach themselves to aquatic vegetation, and encyst to form infective metacercariae.

Infection of the final host occurs when these metacercariae are ingested along with the vegetation. Within the vertebrate host, each metacercaria releases immature flukes that traverse the intestinal wall, migrate through the peritoneal cavity, and penetrate the liver capsule. These young flukes then tunnel through the liver parenchyma over a period of 6-8 weeks, causing extensive damage, before finally entering the bile ducts. The prepatent period, the time from initial infection to when the parasite begins producing eggs, spans 10-12 weeks (Ibrahim, 2017).

Pathogenesis

The pathological consequences of fasciolosis depend primarily on the quantity of metacercariae ingested by the host animal, leading to different forms of the disease:

i. Acute Fasciolosis: Clinical signs typically appear 5-6 weeks after the ingestion of a large number of metacercariae. Acute fasciolosis results from the migration of numerous immature flukes, aged 6-8 weeks, through the liver parenchyma, causing extensive mechanical damage to the liver. This damage leads to acute hepatic insufficiency and hemorrhage. In cases where a substantial number of flukes invade, the liver capsule may rupture, and affected animals can die within days of severe clinical signs emerging. The liver becomes enlarged, pale, and friable, displaying numerous hemorrhagic tracts. Fibrinous clots are observed on the liver's surface and throughout the peritoneal cavity.

ii. Sub-acute Fasciolosis: In sub-acute fasciolosis, the affected liver is covered with migratory tracts, accompanied by evidence of cellular infiltration and early fibrosis.

iii. Chronic Fasciolosis: Chronic fasciolosis typically occurs in cases of light and repeated low-dose infections. Adult flukes establish themselves in the bile ducts, leading to hyperplastic cholangitis, biliary obstruction, fibrosis, and leakage of plasma protein across the epithelium, resulting in hypoalbuminemia. Severe anemia occurs due to the feeding activity of the flukes. In heavy infections, bile ducts may show hypertrophy and calcification, causing the entire organ to harden, a condition commonly referred to as "pipe-stem liver." The dark brown pigment in the hepatic cells is attributed to the deposition of iron-porphyrin in the tissue. In some cases, quiescent spores of Clostridium novyi may become activated due to anaerobic necrotic conditions created in the infected liver parenchyma by migrating Fasciola hepatica, leading to infectious necrotic hepatitis (black disease) in sheep and cattle. Chronic infection can limit the growth rate and feed conversion in growing heifers and the growth rate in beef cattle. Fasciola infection has also been linked to an increased susceptibility of cattle to salmonella Dublin. Chronic fasciolosis is more commonly observed in sheep, especially when they are on a low-nutrition diet.

Clinical findings in fasciolosis include pale mucous membranes, lethargy, a distended abdomen, ascites, anemia, jaundice, a stiff gait, loss of appetite, emaciation, black scouring (dark, tarry feces), and a swollen area under the jaw known as "bottle jaw" due

to edema. In acute cases, severe normochromic anemia and eosinophilia occur due to the migration of young flukes through the liver, and sudden death may result, although no fluke eggs are passed in the feces. In sub-acute and chronic infections, weight loss is associated with severe hypochromic macrocytic anemia, hypoalbuminemia, and hyperglobulinemia. Submandibular edema and ascites occur occasionally in subacute cases but are more frequent in chronic disease. Cattle with chronic fasciolosis may develop diarrhea but tend to lose most of their infections after about six months.

Diagnosis

The diagnosis of fasciolosis relies on several key methods and observations:

i. Clinical Signs:

- Acute Infection: This stage is characterized by sudden death and severe anemia, even though there may be no evidence of fluke eggs in the feces.
- Sub-acute Cases: Signs include rapid loss of condition, severe anemia, a high fluke egg count, and death occurring between 12-30 weeks after infection.
- Chronic Fasciolosis: Gradual wasting, severe anemia with ascites (abdominal fluid buildup), the appearance of "bottle jaw" (swollen area under the jaw), and a very high fluke egg count are typical. Death may occur more than 20 weeks after infection.

ii. Grazing History: Fasciola infection is often associated with flocks or herds that graze in marshy and wetland areas and drink water from locations infested with snails, which serve as intermediate hosts.

iii. Faecal Examination: The presence of chronic hepatic fasciolosis can be confirmed by identifying a large number of characteristic, indistinctly operculated (lacking a clear lid), thin-walled, and yellowish-brown fluke eggs in the feces, typically using the sedimentation technique. However, it's crucial to distinguish these eggs from those of other flukes, especially the large operculated eggs of Paramphistomum. The specific gravity of these eggs is high, so they do not float in all flotation solutions; Zinc sulfate solution is recommended for this purpose.

iv. Serological Diagnosis: ELISA (enzyme-linked immunosorbent assay) can be used to detect antibodies in serum or milk, which can be particularly useful for diagnosing infection.

v. Necropsy Findings: During necropsy (post-mortem examination), the following findings may be observed:

- Acute Hepatic Fasciolosis: The liver capsule may have small perforations and sub-scapular (beneath the surface of the liver) hemorrhages. The liver itself is enlarged, pale, and friable, with numerous hemorrhagic tracts. Fibrinous clots can be seen on the liver's surface and within the peritoneal cavity (abdominal cavity). The peritoneal cavity may contain excess blood-stained serum. Immature flukes can be demonstrated by thinly slicing a piece of liver and shaking it in water, causing the flukes to settle at the bottom.
- Chronic Fasciolosis: Grossly visible leaf-like flukes are present on the enlarged and thickened bile ducts, particularly in the ventral lobe of the liver. The bile ducts may protrude above the liver's surface, and cysts may form due to blockages caused by flukes and desquamating (shedding) epithelial cells. Calcification of the bile duct walls is a common finding in cattle but not in sheep. The hepatic parenchyma (liver tissue) is extensively fibrosed (contains excessive fibrous tissue), and the hepatic lymph nodes appear dark brown in color.

Treatment

Triclabendazole, administered orally at a dosage of 10-12 mg/kg body weight, is widely recognized as the most effective drug against all stages of fluke infections. Other drugs like benzimidazole (triclabendazole), salicylanilides (closantel, oxyclozanide at 10-15 mg/kg), substituted phenol (nitroxynil at 10 mg/kg, subcutaneous), and rafoxanide (at 7.5 mg/kg body weight) also show effectiveness against flukes, albeit with varying degrees of efficacy against mature and immature flukes. Diamphenethide, at a dosage of 100-150

mg/kg, is a preferred treatment for acute fasciolosis in sheep. Additionally, chemicals such as carbon tetrachloride, hexachlorethane, hexachlorophene, and bithionol can be employed at appropriate dosages.

Control and prevention of fasciolosis are best achieved through an integrated strategic approach, which is more cost-effective and less likely to induce anthelmintic resistance than routine dosing. This approach necessitates a thorough understanding of the epidemiological cycle of the disease. Chemical interventions can be applied in the spring to target snail populations before breeding begins or later in the season when snails are abundant but before cercariae start emerging. Chemicals like niclosamide, copper sulfate, sodium pentachlorophenate, etc., can be utilized for this purpose. Reducing snail populations can also be achieved by limiting their habitat size, which can be done by applying molluscicides, fencing off areas, draining marshy zones, and maintaining ditches, water troughs, and land drains properly.

Prophylactic anthelmintic use aims to reduce pasture contamination with fluke eggs, especially during the period most conducive to fluke development (April to August), and to manage fluke populations during times of heavy burden or nutritional stress in animals. On heavily contaminated pastures, livestock can be protected by treating them between the ingestion of metacercariae and the onset of disease. During this period, animals are treated against immature flukes to prevent their migration and further liver damage. After some weeks, treatment against adult flukes is needed to prevent possible losses from chronic fasciolosis.

In conclusion, fasciolosis has a significant impact on the livestock industry's economy. Economic losses stem from the costs of anthelmintics, drenches, labor, liver condemnation during meat inspection, as well as production losses due to mortality, reduced meat and milk production, slower growth rates, decreased fertility, and lower resistance to other diseases. Reducing pasture contamination with metacercariae can mitigate future risks through strategies like preventing snails from becoming infected with Fasciola or reducing snail populations. A combination of control measures, including strategic anthelmintic treatment with appropriate flukicides, drainage, fencing, and molluscicide use, should be employed to effectively control and prevent the disease and associated losses.

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