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Bovine Fasciolosis in Ethiopia-A review

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Abstract

In Ethiopia, fasciolosis is a parasitic disease that affects most population of cattle. Fasciolosis exists in almost all parts of the country. It is caused by commonly known species of liver fluke that are *Fasciola hepatica* and *Fasciola gigantica* which mainly affects domestic ruminants. Fasciolosis is more apparent in young cattle and is usually chronic in nature. Adult flukes in the bile ducts cause inflammation, biliary obstruction, distraction of liver tissue and anemia. Snails of family Lymnaeidae are main intermediate hosts having great role on the transmission of the disease and the infection is acquired through grazing on swampy pasture. The epidemiology of fascioliasis is strictly linked to the geographical and environmental characteristics of the area where transmission occurs. The disease mostly diagnosed by prior knowledge of the epidemiology of the disease in a given environment; observation of clinical signs, information on grazing history, seasonal occurrence and standard examination of feces in the laboratory. The affected cattle should be effectively treated with oral administration of a narrow spectrum anthelmintic such as Triclabendazole in addition to reducing the population of the intermediate host to control the disease. Now a days, fasciolosis is recognized as emerging human disease over the world even if only few case reports of human fascioliasis are available in Ethiopia, as the disease mostly affects animals in the country. The disease causes a significant economic loss in cattle production by inflecting direct and indirect loss at different parts of Ethiopia. In cattle industry, the loss mainly occurs through mortality, liver condemnation reduces production of milk, meat and expenditure for anthelmintics. To control and prevent the disease, the strategic destruction of snail population should be implemented throughout the country to break down the life cycle of liver fluke.

Keywords: Bovine; *Fasciola Gigantica; Fasciola Hepatica;* Fasciolosis; Liver Fluke

Introduction

Bovine fasciolosis is one of the most important parasitic diseases of cattle causing mortality and Production losses in various parts of Ethiopia. It is the priority disease in the highland as well as in lowland areas of the country [1]. This important helminthic disease is caused by two trematodes such as, *Fasciola hepatica (F. hepatica)* (the common liver fluke) and *Fasciola gigantica (F. gigantica)*. The disease is a plant-borne trematode zoonosis, and is classified as a neglected tropical disease (NTD). It affects humans, but its main host is ruminants such as cattle and sheep. In Europe, Americans and Oceania, only *F. hepatica* is a concern, but the distributions of both species overlap in many areas of Africa and Asia [2].

F. hepatica and F. gigantica are the most common species of liver flukes that cause hepatobiliary system infection mainly in cattle and sheep that they have an impact on public health. Human fascioliasis is caused by *F. hepatica* as it is recently been recognized as an emerging and re-emerging zoonotic disease in several countries [3]. World health organization (WHO) now recognized fasciolosis as an emerging human disease and 2.4 million people are infected with Fasciolosis, and further 180 million are at risk of infection [4,5]. *F. hepatica* was shown to be the most important fluke species in Ethiopian livestock with distribution over three quarter of the nation except in the arid northeast and east of the country. It is the most important trematode that causes liver fluke disease of domestic ruminants in temperate areas [6].

The life cycle of *F. hepatica* goes through the intermediate host and several environmental larval stages [7]. Intermediate hosts of *F. hepatica* are air-breathing fresh water snails from the family Lymnaeidae. Although several Lymnaeid species susceptible to *F. hepatica* have been described, the parasite develops only in one or two major species on each continent. Galba truncatula is the main snail host in Europe, partly in Asia, Africa, and South America. *Lymnaea viator, Lymnaea neotropica, Pseudosuccinea columella,* and *Lymnaea cubensis* are most common intermediate hosts in Central and South America [8,9].

A diagnosis may be made by finding yellow-brown eggs in the stool. In addition, an enzyme-linked immunosorbent assay (ELISA) test is the diagnostic test of choice [10,11]. *F. gigantica* is similar in shape to *F. hepatica* but is larger (75mm), with less clear defined shoulders. It is found warm climates (Asia, Africa) in cattle and buffalo, in chronic fasciolosis. The life cycle is

similar to that of *F. hepatica* except for species of snail intermediate hosts. The pathology of infection, diagnostic procedures, and control measures are similar to those for *F. hepatica* [12].

The taxonomic classification of the organisms that cause fasciolosis is presented as **Phylum**:-Platy helminthes, **Class:**-Trematoda, **Sub-class:**-Digenea, **Super Family**:-Fasciolidea, **Genus**:-Fasciola, **Species**:-*Fasciola hepatica* and *Fasciola gigantic* [13,14]. Among the above species of animal, bovine fasciolosis is a chronic wasting disease caused in the liver and bile ducts respectively by immature and adult trematodes of the genus Fasciola. Economically important infections are seen in cattle and sheep in three forms: chronic, which is rarely fatal in cattle but often fatal in sheep; subacute or acute, which is fatal primarily in sheep [15]. Recently, worldwide losses in animal productivity due to fasciolosis were conservatively estimated at over US$ 3.2 billion per annum [16]. In general, Fasciolosis is one of the major setbacks to livestock productivity, incurring huge direct and indirect losses in the country. Therefore, the objective of this paper is to review currently available literatures on bovine Fasciolosis in Ethiopia.

Morphology of *Fasciola spps*

Fasciola hepatica is one of the largest flukes of the world, reaching a length of 30mm and a width of 1mm. The adult parasite has a flat leaf-like body, typical of flukes, and measures 20 to 30mm long by 8 to 15mm wide [13,17]. These flatworms form seven different developmental stages: eggs, miracidia, sporocysts, rediae, cercariae, metacercariae, and adult flukes. They are dorsoventrally flattened, the tegument is covered with scaly spines, and they have two suckers (distome arrangement with the oral sucker and acetabulum close together). They have a bifurcate blind gut and each worm is hermaphroditic, possessing both male and female reproductive organs. The pair of testes, also highly branched, is located in the posterior half of the body. The relative compact ovary is located just above the testes and is linked to a short convoluted uterus opening to a genital pore above the ventral sucker. The vitellaria are highly diffuse and branched in the lateral and posterior region of the body. It has an anterior elongation (a cephalic cone) on which the oral and ventral suckers, which are approximately of equal size. The intestine of the adult parasite is highly branched, with numerous diverticulae extending from the anterior to the posterior of the body [18]. *F. gigantica* is the largest of the human liver and lung flukes. It is a parasite very similar to *F. hepatica,* with length that may vary 25 to 75 mm long by 15 mm wide. It has a shorter cephalic cone, a larger ventral sucker and a more anterior position of the testes. The egg of *F. hepatica* measures 150µm by 90µm in size and very similar in shape to that of *F. gigantica* [14]. The egg of the latter is larger in size (200 µm x 100 µm) [13]. *Fasciola* eggs should be distinguished from the eggs of other flukes, especially from the large eggs of *Paramphistome. Fasciola* eggs has a yellowish brown shell with an indistinct operculum and embryonic cells whereas *Paramphistome* egg has transparent shell, distinct operculum with embryonic clear cells, and possess a small knob at their posterior end [14, 19].

Etiology of *Fasciola spps*

Fasciolosis is a parasitic worm infection caused by the common liver fluke *Fasciola hepatica* as well as by *Fasciola gigantica. Fasciola hepatica* is the most common and important liver fluke and has a cosmopolitan distribution in cooler climates. *Fasciola hepatica* may infest all domestic animals, including equine and many wild life species, but chronically infected sheep are the most important source of pasture contamination. Human cases are usually associated with the ingestion of marsh plants such as water cress. A similar but larger fluke, *F. gigantica* is restricted to warmer regions including parts of Africa and Asia [9,20]. Economically important infection of fasciolosis is seen in cattle. Eggs are passed in the feces, and miracidia develop within as little as 9-10 days (at 22-26 °C [71.6-78.8 °F]; little development occurs below 10 °C [50 °F]). Hatching only occurs in water, and miracidia are short-lived (~3 hr). Miracidia infect Lymnaeid snails, in which asexual development and multiplication occur through the stages of sporocysts, rediae, daughter rediae, and cercariae. After 6-7 wk (or longer if temperatures are low), cercariae emerge from snails, encyst on aquatic vegetation, and become metacercariae. Snails may extend the developmental period by hibernating during the winter. Metacercariae may remain viable for many months unless they become desiccated. After ingestion by the host, usually with herbage, young fluke's excyst in the duodenum, penetrate the intestinal wall, and enter the peritoneal cavity, where they migrate to the liver. The time required for this transit can vary and results in delayed development rates, which affects the efficacy of some treatments because many are effective against flukes only later in their development. The young flukes penetrate the liver capsule and tunnel through the parenchyma for 6-8 wk, growing and destroying tissue. They then enter small bile ducts and migrate to the larger ducts and, occasionally, the gallbladder, where they mature and begin to produce eggs. The prepatent period is usually 2-3 months, depending on the fluke burden. The minimal period for the completion of one entire life cycle is ~17 wk. Adult flukes may live in the bile ducts of sheep for years; most are shed from cattle within 5-6 months [21].

Intermediate hosts

The snail intermediate hosts for *Fasciola spp*. are in the family Lymnaeidae, particularly species in the genera Lymnaea, Galba, Fossaria, and Pseudosuccinea. At least 20 snail species have been identified as intermediate hosts for one or more Fasciola spp. Snail species may differ with respect to their suitability to serve as intermediate hosts for *F. hepatica* versus *F. gigantica* [12]. The epidemiology of Fasciolosis is dependent on the ecology of the snail intermediate hosts. *Lymnaea species*, most common in Europe, Asia, Africa and North America; *L. bulimoides* in North America; and *L. tomentosa* in Australia. Other species, which have been incriminated in the transmission of *F. hepatica,* include *L. viator* and *L. diaphena* (South America), *L. columnella* (USA, Australia, Central America and New Zealand) and *L. humilis* (North America) [13,14].

L. truncatula is the most common intermediate host for *F. hepatica* in different part of the world [22] and in Ethiopia [23]. It is an amphibious or mud-dwelling snail which prefers moist temperature conditions (15-22 °C) though it appears that variants found in the tropics have adaptation to higher temperature mostly in the lowlands areas and can breed and survive at 26 °C with sufficient moister. The most important intermediate hosts of F. gigantica are *L. natalensis* and *L. auricularia* [13,14,24]. *L. natalensis* is the recognized intermediates host for *F. gigantica* (Yilma and Malone, 1998). Other species serving as secondarily hosts to this species are *L. Rufescens* and *L. acuminate* (Indo-Pakistan) and *L. rubiginosa* (Malaysia) [24].

Life Cycle of *Fasciola spps*

Definitive hosts of the fluke are cattle, sheep, and buffaloes. Wild ruminants and other mammals, including humans, can act as definitive hosts as well [8]. The life cycle of a typical trematode begins with an egg. Some trematode eggs hatch directly in the environment (water), while others are eaten and hatched within a host, typically a mollusk. The hatchling is called a *miracidium*, a free-swimming, ciliated larva. Miracidia will then grow and develop within the intermediate host into a sac-like structure known as a sporocyst or into rediae, either of which may give rise to free-swimming, motile cercariae larvae. The cercariae then could either infect a vertebrate host or a second intermediate host. Adult metacercariae or mesocercariae, depending on the individual trematode's life cycle, will then infect the vertebrate host or be rejected and excreted through the rejected host's feaces or urine [25].

The life cycle of *Fasciola spp*. is a typical of digenetic treamatodes. Eggs laid by the adult parasite in the bile ducts of their hosts pass into the duodenum with the bile [5,13,14]. Therefore, immature eggs are discharged in the biliary ducts and passed in the stool. Eggs become embryonated in freshwater over ~2 weeks; embryonated eggs release miracidia, which invade a suitable snail intermediate host. In the snail, the parasites undergo several developmental stages (sporocysts, rediae, and cercariae). The cercariae are released from the snail and encyst as metacercariae on aquatic vegetation or other substrates. Humans and other mammals become infected by ingesting metacercariae-contaminated vegetation (e.g., watercress). After ingestion, the metacercariae excyst in the duodenum and penetrate through the intestinal wall into the peritoneal cavity. The immature flukes then migrate through the liver parenchyma into biliary ducts, where they mature into adult flukes and produce eggs. In humans, maturation from metacercariae into adult flukes usually takes about 3-4 months; development of *F. gigantica* may take somewhat longer than *F. hepatica* [26].

The cercaria of *Fasciola spp*. have a rounded body measuring between 0.25 and 0.35 mm long, with a long thin unbranched tail measuring approximately 0.5 mm long. The mobile cercaria snail generally leaves the snail 4-7 weeks after infection by migrating through the tissues of snails. This is during moist conditions when a critical temperature of 10 °C is exceeded. On emerging from the snail the cercaria attaches to submerged blades of grass or other vegetation like watercress; the tail falls away and the cercarial body secretes a four-layered cyst covering from cystogenous glands located on the lateral regions of the body. The formation of the cyst wall may take up to two days. The metacercaria (encysted, resistant cercariae) is the infective form to the definitive host. Generally, metacercaria are infective to ruminants such as cattle and sheep, but also to other mammals including human beings. One miracidium hatching from a fluke egg can produce up to 4,000 infective cysts (metacercariae) due to the vegetative multiplication at the sporocyst and redai stages. The metacercarial cyst is only moderately resistant, not being able to survive dry conditions. If however, they are maintained in conditions of high humidity and cool temperatures, they may survive for up to a year [5, 13,14].

The metacercaria cysts, when ingested along with the contaminated vegetation by the definitive host enter into the small intestine, releasing the young parasite, which penetrates the gut wall, entering the peritoneal cavity. From there, it migrates directly to the liver over a period of approximately seven days, directly to the liver. The juvenile fluke (also referred to as adeloscaria) then penetrates the liver tissues, through which it migrates, feeding mainly on blood, for about six weeks. After this period, the fluke enters the bile ducts, maturing in to a fully adult parasite after about 3 months from initial infection. Egg production then commences and completing the life cycle. Adult flukes can survive for many years in the livers of infected hosts and lay between 20,000 and 50,000 eggs/day. The rate of egg production is responsible for the degree of pasture contamination and thus greatly influences the epidemiology of the disease. The epidemiology of the disease is also influenced by the grazing habits of the animals. Animals grazing in wet marshy areas, favored by the intermediate host, are more likely to become infected. Typically, long and wet seasons are associated with a higher rate of infection. However, sheep are more likely to ingest large numbers of cysts during dry periods following a wet season. This is due to a reduction in available pasture, forcing the animals to graze in swampy areas or in areas where the water has receded, thus exposing them to vegetation heavily infected with metacercariae [27]. Some research shows metacercariae (infective Cysts) on herbage can remain infective for several months (up to 9-10 months) if conditions are cool and damp [28]. Their survival depends on moisture and moderate temperatures. Metacercariae will not survive for more than six weeks at 25 degrees, but can survive for eight weeks at temperatures of -2 degrees [29].

Epidemiology

The epidemiological pattern of fascioliasis is quite varied: the infection usually has a hypo-endemic pattern, with low and stable levels of prevalence among a defined population. Sporadic outbreaks may occur among such populations: these are usually related to sudden changes in climatic conditions that boost the life-cycle of either the parasite or the snail, or both. Scientists have also

found that the epidemiology of fascioliasis is strictly linked to the geographical and environmental characteristics of the area where transmission occurs, and different patterns can be distinguished. This suggests that fascioliasis may adapt to different ecological niches [4] as cited by Wokem and don [30].

The epidemiology of fasciolosis depends on the grazing habitat preference of the animal. Njau and Scholtens [31] reported that metacercaria can survive up to 3 months after harvesting in hay from endemic highland areas that are consumed by the ruminants in arid and lowland areas, particularly during the dry season when suitable grazing pastures are scarce; local crowding of animals along the banks of streams and ponds during the dry season. When nutritional conditions are generally compromised also provides an important dynamics for infection transmission. Irrigation would have major effects on transmission [32].

Fasciolosis is considered an important limiting factor for bovine and ovine production. In general, infection of domestic ruminants with *F. hepatica* and *F. gigantica* causes significant economic loss estimated at over US$ 200 million per annum to the agricultural sector worldwide, with over 600 million animals infected. In developed countries, the incidence of *F. hepatica* ranges up to 77% [16,33]. Evidence suggests that sheep and cattle may be considered the main reservoir host species, pigs and donkeys being secondary [34]. In tropical regions, fasciolosis is considered the single most important helminth infection of cattle with prevalence rates of 30-90% in Africa, 25-100% in India and 25-90% in Indonesia [16].

F. hepatica is a temperate species and it is found in Southern America, Northern America, Europe, Australia, and Africa, but found in the highlands of Ethiopia and Kenya. It is the major cause of liver fluke disease in Ethiopia. Its tropical counterpart, *F. gigantica,* on the other hand is widely distributed in tropical countries, in Africa and Asia, parasitizing domestic ruminants and other herbivores in almost every continent. In Ethiopia, *F. gigantica* is found at altitudes below 1800 m.a.s.l. while *F. hepatica* is found at altitude between 1200-2560 m.a.s.l. [32]. Mixed infections by the two species can be encountered at 1200-1800 m.a.s.l. The annual loss due to endo-parasite in Ethiopia is estimated at 700 million Ethiopian birr/ annum [35].

Cattle have a natural resistance and, under normal conditions, clinical disease is only likely in young cattle. However, this resistance, which allows chronically infected cattle to spontaneously recover, and previously infected animals to partially resist reinfection, is due to a more intensive tissue reaction than in sheep. This response results in a fibrous mechanical barrier (fibrosis) against re-infection, by impeding the usually preferred migration of young flukes into the ventral lobe of the liver. The subsequent enlargement (hypertrophy) of the right lobe helps the host survive by leaving sufficient undamaged liver tissue. In chronic cases, mineralization (calcification) of, and fibrosis around bile ducts also causes the elimination of liver flukes. Calcification tends to be minimal or absent in sheep [21,36]. Despite cattle being more resistant to liver fluke disease (fasciolosis) than sheep, it is only possible because of chronic fibrotic changes in the liver, as already mentioned. Infection can be picked up at any time and animals can be repeatedly infected. However, even with a small number of fluke present, there may be production losses if not overt disease. Grazing behavior is another important factor when considering fluke disease. Cattle willingly graze wet areas including fluke habitats at any time, while sheep and goats tend to graze them only when other feed is scarce [21].

Clinical signs

Cattle develop a greater fibrotic reaction in the liver compared to sheep, therefore parasite survival is reduced. Acute fasciolosis is uncommon in cattle, unlike in sheep where it is a highly pathogenic. There is limited development of immunity to fluke, which does not prevent infection. Additionally, the immunity may wane at housing; therefore all age groups of cattle are potentially at risk of fluke infection [37] as cited by Farm health online [38]. Acute fasciolosis occurs as disease outbreak following a massive, but relatively short-term, intake of metacercariae [24]. The high fluke intake is often the result of certain seasonal and climatic conditions combined with a lack of appropriate fluke control measures. It typically occurs when stocks are forced to graze in heavily contaminated wet areas because of overstocking and/or drought. Animals suffering from acute fasciolosis especially sheep and goat, may display no clinical signs prior to death; while some may display abdominal pain and discomfort and may develop jaundice [14, 24]. In some cases, the liver capsule may rupture and fluid may lick into the peritoneal cavity causing death due to peritonitis. More commonly, on ingestion of fewer metacercaria, fever and eosinophilia is seen [14]. Death usually results from blood loss due to hemorrhage and tissue destruction caused by the migratory juvenile flukes in the liver resulting in traumatic hepatitis. This is more commonly seen in sheep than in other hosts. Sub-acute fasciolosis is caused by ingestion of a moderate number of metacercaria and is characterized by anemia, jaundice and ill thrift. The migrating fluke causes extensive tissue damage, hemorrhage and in particular liver damage. The result is severe anemia, liver failure and death in 8- 10 weeks [24]. In one dairy herd, subclinical ketosis was attributed to fluke infection of the cows [39].

The clinical signs of chronic fasciolosis are variable and depend upon the number of metacercariae ingested, but often include: Weight loss, Anaemia, Bottle jaw, Diarrhoea, Constipation [40]. Fluke infection may predispose to other conditions due to impaired liver function and can reduce milk yield and fertility [41]. Chronic fasciolosis is the most common clinical syndrome in sheep and cattle. It occurs when the parasite reaches the hepatic bile duct. The principal effects are bile duct obstruction, destruction of liver tissue, hepatic fibrosis and anemia. The onset of clinical signs is slow. Animals become gradually anemic and anorectic, as the adult fluke becomes active within the bile duct and signs may include dependent oedema or swelling under the jaw ('bottle jaw'). Affected animals are reluctant to travel. Death eventually occurs when anemia becomes severe. Additional stress upon anemic animals, such as droving, may lead to collapse and death. Cattle typically present with signs of weight loss, anemia and chronic

diarrhea [19]. In addition to these, a condition known as '*black disease*' is a complication, which usually is fatal. Here, a secondary infection due to the bacterium *Clostridium novyi* Type B, proliferating in necrotic lesions produced by the young larvae migrating in the liver is responsible for the fatal outcome. Chronic fasciolosis provides the right environment in the liver for the germination of the spores of the bacterium [20].

Pathogenesis

The development of infection in definitive host is divided into two phases, the parenchymal or migratory phase and billary [42]. Acute hepatic fasciolosis is caused by the passage of young fasciola hepatica through the liver parenchyma. Clinical signs occur 5-6 weeks after the ingestion of large number of metacercaria. By this time, the migrating flukes are large enough to do substantial mechanical damage to liver. Acute hepatic insufficiency and hemorrhage result. Quiescent spores of clostridium novy may become activated by the anaerobic necrotic conditions created in the liver parenchyma by migrating *F. hepatica*, causing infectious hepatica necrotic hepatitis (black disease) in sheep and cattle. This migration has also been thought to stimulate the development of occasional case of bacillary hemoglobinuria in cattle [43].

Chronic hepatic fasciolosis develops only after the adult flukes establish in the bile duct. They cause cholangitis, biliary obstruction, fibrosis and a leakage of plasma protein across the epithelium. Although this protein can be reserved in the intestine, there is poor utilization and retention of nitrogen leading to hypoalbuminemia. There is also loss of whole blood due to feeding activity of the flukes. This exacerbates the hypoalbuminemia and eventually gives raised to anemia, which is initially normochromic, became hypochromic. These changes are more severe in sheep on a low plane of nutrition [44]. Chronic infection may limit growth and feed conversion in growing heifers and growth rate in beef cattle. *F. hepatica* infection has been reported to increase the susceptibility of cattle to Salmonella Dublin and predispose to a prolonged infection and fecal excretion [45]. Food intake is reduced and these leads to reduction in efficiency of utilization of metabolizable energy and a reduction in calcium and protein deposition in the carcass. The fibrotic response of the liver to fluke induced damage varies with the host and may partially accounts for different liver to filuke induce damage varies with the host and may partially account for differing species susceptibilities. The severity reaction in cattle, calcification of the bile ducts appears to hinder the establishment and feeding of challenge infection. There by reinforcing immune response goes in there mode of resistance [44].

Pathogenesis of fasciolosis varies according to the parasitic development phases; parenchymal and biliary phases. The parenchymal phase occurs during migration of flukes through the liver Parenchyma and is associated with liver damage and hemorrhage. The biliary phase coincides with parasite residence in the bile ducts and results from the haematophagic activity of the adult flukes and from the damage to the bile duct mucosa by their circular spines [24]. In the bile ducts of some permissive hosts, such as the sheep, rabbit, rat and mouse, the biliary stage of the disease is common. In others, such as cattle and humans, few flukes survive beyond the migratory phase and biliary disease is relatively rare. Light infections due to *Fasciola hepatica* may be asymptomatic. However, they may produce hepatic colic with coughing and vomiting; generalized abdominal rigidity, headache and sweating, irregular fever, diarrhea and anemia [46]. In domestic ruminants, an adverse effect of acute or chronic *fasciolosis* includes decreased weight gain and milk production, decreased female fertility, work power and mortality. Hepatic pathology, even when only limited areas of the liver are damaged, results in significant disturbances in mitochondrial bioenergetics metabolism of carbohydrates, proteins, lipids and steroids, as well as bile flow and bile composition [47]. During the movement of the immature stages of *Fasciola hepatica,* which may continue for months, symptoms may include abdominal pain, an enlarged liver, fever, and diarrhea. The pathology associated with diseases are caused by the inflammation of the bile ducts which causes thickening of the lining and eventually leads to fibrosis that results in reduced flow of the bile and back pressure builds leading to atrophy of the liver parenchyma and cirrhosis. The complexity arises from several sources. Maturation of flukes involves development and growth for over 12-16 weeks during which time the fluke travels between and within organs. Because an individual fluke may pass the same part of the liver twice (or more) during these peregrinations, fresh and resolving lesions caused by the sequential insults may be found in the same section of tissue; as the migratory fluke grows the size of its track through the liver increases as does the damage and the inflammatory response. Calves are susceptible to *fasciolosis* but in excess of 1000 metacercariae are usually required to cause clinical *fasciolosis* [19,46]. The disease is characterized in calves by weight loss, anemia, and hypoproteinemia after infection with 10,000 metacercaria [46]. Resistance develops with age so that adult cattle are quiet resistant to infection. Even though, the rate of development of human fasciolosis is similar to that in sheep, as an unnatural host, only few flukes develop sufficiently to reach the bile duct. Hypoalbuminemia and hyperglobulinemia commonly occur in liver fluke infections in all host species. During the parenchymal stage of the infection, liver damage caused by the migrating flukes compromise liver function, which in sheep and calves is reflected in a decline in plasma albumin concentrations, attributed partly to reduced rate of synthesis and partly to an expansion of the plasma volume [24,46]. Nevertheless, during biliary stage of the infection loss of blood from haematophagia and into the intestines is so extensive, causing severe anemia that synthetic capacity of the liver is insufficient to replace the lost albumin (small molecular size) that oozes through the hyperplastic bile ducts (Cholangitis). Thus, a progressive loss of plasma albumin occurs in all infected host species, starting from around the time the fluke commences blood feeding. This results in disturbance in intravascular and extravascular oncotic pressure leading to the development of edema, often markedly visible at submandibular region of ruminants ('bottle jaw'). Liver trauma is the abrasion caused by cuticular spines and the prehensile action of the suckers and appears to account for the majority of the damage caused in the liver. Death of the host is a consequence of the hemorrhage induced by this damage. The oral sucker is the route by which liver flukes obtain most of their nutrition. It appears

to cause considerable damage to liver tissue and macerated hepatic cells have been observed inside the sucker and pharynx. The oral sucker extends during migration and feeding from the earliest stages is capable of disrupting cells. The muscular pharynx assists in this process and oral sucker is a major organ involved in tissue disrupting. Although the inflammatory process has an important role in protecting the host against severe consequences of liver damage by the flukes, perhaps by retarding the growth of the parasite and contributing to hepatic healing process, there is accumulated evidence, in rats, that the response also contributes to hepatic dysfunction. There is evidence also that the infected rat liver is under oxidative stress during the parenchymal stage of the infection. The liver plays a central role in the physiology of the body, being responsible for a large proportion of the body's amino acid metabolism, for carbohydrate and lipid balance, urea synthesis, detoxification metabolism, ketogensis, albumin and glutathione synthesis as well as aspects of homeostasis. Therefore, it is to be expecting that many systemic changes will be induced by liver fluke infections that ultimately cause reduced productivity in livestock. Both anorexia (inappetance) and the quality of the diet of infected sheep contribute to hypoalbuminemia during the infection [46].

Diagnosis

Diagnosis of fasciolosis may consist of tentative and confirmatory procedures. A tentative diagnosis of fasciolosis may be established based on prior knowledge of the epidemiology of the disease in a given environment; observations of clinical signs, information on grazing history and seasonal occurrence. Confirmatory diagnosis, however, is based on demonstration of *Fasciola* eggs through standard examination of feces in the laboratory; postmortem examination of infected animals and demonstration of immature and mature flukes in the liver. The latter is helpful in deciding the intensity of infection. There are other laboratory tests (enzymatic and/or serological procedures used to qualify the infection mainly for research purposes. Serological assays are often used to detect infections due to immature forms where fecal egg output is often nil. Such tests allow the detection of substance like cathepsin *L. proteases*, excretory secretory products, detection of Ag in milk, and ELISA detection of antibodies against the flukes plasma concentration of Gamma-glutamyltransferase (GGT), which are increased with in the bile duct damage [14,24,48]. The good example, Oxidative stress would be one of the consequences of the activity of inflammatory cells such as neutrophils, macrophages and eosinophils in producing oxygen-derived free radicals, nitric oxide and their products. A useful indicator of oxidative stress is the concentration of reduced glutathione in cells. For chronic fasciolosis, confirmatory diagnosis could easily carried out by coproscopic examination employing sedimentation technique. *Fasciola* eggs have high specific gravity and sedimentation is preferred to floatation. When the latter is employed, floating medium such as $ZnSo_4$ should be used. As Fasciola eggs may be confused with Paramphistomum eggs, addition of methylene blue in the fecal suspension will facilitate ease identification by providing a blue and contrasting microscopic field [49]. The PCR method is proper for most epidemiological surveys. PCR with DSJF/DSJ3 primers were used to identify *F. hepatica* eggs from fecal samples of naturally infected various domestic ruminants and accordingly, PCR was found to be more powerful diagnostic tool to detect Fasciola infection. PCR-RFLP tests showed that *F. hepatica* was dominant species in animals and no evidence of *F. gigantica* was observed [50,51].

Current status of Bovine Fasciolosis in Ethiopia

Currently in Ethiopia, *Fasciola hepatica* is wide spread in areas with altitude of 1200-2500 m.a.s.l. Both Fasciola species co-exist in area with latitude raning between 1200-1800 m.a.s.l. [32]. The snail intermediate hosts of Fasciola species in Ethiopia are principally two, which are *Lymnaea truncatula* and *Lymnaea natalensis*. The former one is the intermediate hosts of *Fasciola hepatica* and the later for *Fasciola gigantica*. *Lymnaea natalensis* are widely distributed in low land (kola), in irrigated canals, and pockets of water with vegetation, *Lymnaea truncatula* are usually encountered in medium altitude (Weyna dega) and a high land (dega), also the prevalence of the disease is mostly encountered in these areas [52]. The pathogenic significance of Fasciola spps depends on the favorability of environment they live [53]. The prevalence of bovine fasciolosis due to the *F. hepatica* and *F. gigantica* in Ethiopia has been known by different workers. The prevalence of bovine fasciolosis based on coproscopic result varies from 11.5% [54] up to 87% in Debrebirhan [55]. The abattoir studies have also indicated from 8.1% in Buno province [54] up to 88.57% in Debrebirhan [56]. In different regions of the country, there are different reports that indicate the prevalence of fasciolosis as shown in the table below (Table 1).

Region	Faecal (%)	Abattoir (%)	Reference
Wulnchit	-	34.23	Asefa & Tegegne [55]
Wolkite	41.8	41.8	Tesfaye and Tigist [56]
Nekemte	17.14, 32	22.7, 29.8, 21.9	Abebe [57], Wassie [58], Alula, *et al.* [59] respectively
Jimma	-	58,46.15	Moges [60], Tadele and Worku [61] respectively
Kombolcha	51.13	53.5	Mulugeta [35]
Kalu	15.17	-	Girmay [62]
BahirDar	60.2, -, 36.72	61.97,84.7, 39.9	Fekadu [63], Yohannes [64] Fikirtemariam, *et al.* [65] respectively
Gondar	33.42	33.4,50	Roman [66], and Mesfin [67] respectively

Region	Faecal (%)	Abattoir (%)	Reference
Debrebirhan	87	88, 57	Dagne [53], and Tsegay [54] respectively
Eastern Gojjam	50.50	-	Bayazn [68]
Soddo	-, 15.9	47, 20.3	Abdul [69], and Negesse and Mohammed , [70] respectively
Hawassa	-	30.43	Hailu [71]
Arsi	53.72	41.3	Wondwossen [72]
Assela	20.21, 32.9, 45.25	-, -, 34.97	Yosef [73], Dinka [74], and Shiferaw, *et al.* [75] respectively
Wolliso	34	-	Rahmato [76]
Chilalo	26.2	-	Zerfu [77]
Bale	34.6	49	Abdul-jabar [78]
Zeway	32.7	-	Adem [79]
Western shoa	82.5	-	Yadeta [50]
Western hararge	42.9	12.1	Haymanot [80]
Tigray	-	12.1	Takele [81]
Buno province	11.5	26	Seyoum [52]
Hawzie	-	21.39	Girmay, *et al.* [82]

Table 1: Summary on prevalence of bovine fasciolosis in different region of Ethiopia determined based on coproscopic and abattoir examination

Economic significance of bovine Fasciolosis in Ethiopia

Fasciolosis is an important cause of both production and economic losses in the dairy and meat industries. Over the years, the prevalence has increased and it is likely to continue increasing in the future [83-85]. Bovine Fasciolosis render the liver unsuitable for human consumption. It also cause loss through reduction in meat and milk inhibited reproduction in wool growth and quality, decreased feed intake, conversion and lowered resistance [86]. Fasciolosis cause major economic losses in cattle sheep, goat and buffaloes [87]. In Ethiopia, a rough estimation of economic loss due to decreased production in animal because of fasciolosis was found to be 300 million Birr per year [86].

Plane of abattoir	Annual economic loss (In ETB)	Reference
Assela	698,700.6	Shiferaw, *et al.* [75]
Hawzie	885,500	Girmay, *et al.* [82]
Kombolcha	266, 741.37	Mulugeta [35]
Wolkite	182582.4	Tesfaye and Tigist [56]
Wolaita Soddo	142, 128; 3,711,246	Abdul [69], and Negesse and Mohammed [70] respectively
Wolliso	78, 311.60	Rahmato [76]
Jimma	480,789.0; 54,063.34; 244; 500.84	Zewdu [87], Tadele and Worku [61] and Shimels [88] respectively
Gonder	4497,752.36	Roman [66]
Diredawa	215,000	Daniel [89]
Hawassa	122, 775.54; 296, 370	Hailu [71], Tsehaynesh and Biruk [90] respectively
Bahirdar	200,000	Yohannes [91]
Robe	109; 601.24	Abdul- Jebbar [78]
Mekelle	224; 539.20	Yohannes [91]
Wulnchit	4, 522; 550,000	Asefa & Tegegne [55]
Nekemte	63072	Alula, *et al.* [58]

Table 2: Annual Economic loss summary due to bovine Fasciolosis in different abattoirs of Ethiopia

Control and Prevention

Several control methods against ruminant fasciolosis are available and can either be used independently and as a combination of two or more of them. These methods involve reduction in the number of intermediate snail hosts by chemical or biological means, strategic application of anthelmintics, reduction in the number of snails by drainage, fencing and other management practices and reduction in the risk of infection by planned grazing management [24].

Controlling intermediate host (Snail)

Control of parasitic diseases is crucial to improve the productivity of the animals. In most fasciolosis endemic areas, the control of the intermediate snail host population offers a good opportunity for the reduction of transmission and is generally effective when combined with one or more other methods such as chemotherapy or environmental sanitation [92,93]. Although eradication of the snail hosts is the most effective method of total fluke controls this, however, is often very difficult in low-lying, wet areas with a mild climate. Snails multiply extremely rapidly and hence eradication is almost impossible in irrigation areas. There are different types of snail poison available that are safe for stock but need care and precision in their application. Other useful methods of fluke control include biological control of the intermediate host, fencing the waterlogged area and so on [14,19,94].

The use of molluscicides for the control of snail intermediate hosts is a potential tool for the control of fluke infections. Before considering chemical control of snails, it should be noted that many habitats are topographically unsuitable for the use of molluscicides and it is often very difficult to apply them effectively. Whereas, they are not species-specific, may destroy edible snails highly valued as food in some communities and expensive [94].

A great number of chemicals have been used as molluscicides in the past, but at present Niclosamide (Bayluscide or mollotor) and copper sulfate are used in different part of African Countries [95]. Brown [96] indicated that molluscicidal properties have been demonstrated in extracts from a variety of plants. A substance 'Endod' or toxins derived from the fruits of shrubs *Phytolacca dodecandra* [97] as cited by Brown [95]. Substance such as 'Endod' might provide means of snail control less costly to developing countries than synthesized by molluscides but the production naturally molluscides on a commercial scale has yet to achieved. Tadesse and Getachew [98-100] from their finding they indicated that 'Endod' used for the control of fasciola transmitting snails particularly *L. truncatula* and *L. natalensis*.

Controlling by use of chemicals (Chemotherapy)

Effective control of most trematode infections is based on strategically applied chemotherapy [94]. Combination of chemotherapy, intermediate host control, sanitation and Environmental manipulation are believed to be more efficient but very expensive. A flukicidal drug of choice must fulfill the following: it must act against both immature and mature flukes, it must not be toxic to the recipient animal and it must be cheap and available. Chemotherapy with drugs remains the most cost-effective way of treating parasitic diseases, and is usually at the heart of any major control campaign. Compared to environmental engineering, drug treatment is very cheap [99]. The drugs to be used against flukes should ideally destroy the migrating immature flukes as well as adults in the bile ducts. Several drugs are now available for the treatment of fasciolosis, which are against the adult flukes, and the parenchymal stages. These include Rafoxanide, Nitroxynil, Brotanide, Closantel and Albendazole. Diamphentide that kills all immature flukes even a day old once and the Triclbendazole (TCBZ) are highly effective against all stages of fluke. It is one of the widely used drugs worldwide for the control of fasciolosis [99,100]. Chemotherapy normally reduces the prevalence and intensity of infection as measured by fecal egg counts [101].

Environmental sanitation

Draining swamps, building sewage systems and providing clean water supplies are used to control water-borne /including snail borne/ helminths but it is very expensive compare to chemotherapy [94,99]. Strategies for the treatment and prophylaxis of infections with *Fasciola* are developed based on Epidemiological data. Effective treatment during the prepatent period for an extended duration could eliminate Fasciola infection or reduce contamination of pasture to a very low level, requiring less frequent treatments for a considerable time [32,94].

Retardation of immature flukes, which survive treatment, appears to be applicable to all anthelmintics and the degree of retardation depends on the efficacy of the drugs against the immature stages. This phenomenon has a great advantage in strategic control by reducing early pasture contamination with eggs. Less frequent strategic treatments with a possible yearly rotation of anthelmintics or anthelmintic combinations that are effective against both immature and adult flukes has been reported to provide the best method of successful control of fasciolosis [102].

Treatment

Livestock are often treated with flukicides, chemicals toxic to flukes, including bromofenofos, triclabendazole and bithionol [103,104]. Ivermectin, which is widely used for many helminthic parasites, has low effectivity against *F. hepatica*, as does praziquantel [105,106]. For humans, the type of control depends on the setting. One important method is through the strict control over the growth and sales of edible water plants such as watercress. This is particularly important in highly endemic areas. Some farms are irrigated with polluted water; hence, vegetables farmed from such land should be thoroughly washed and cooked before being eaten [12].

Public health importance of Fasciolosis

Fasciolosis occasionally affects humans, thus Considered as a zoonotic infection [107,108] but it is a disease of sheep, goat, and cattle mainly [9]. Studies carried out in recent years have shown human fasciolosis to be an important public health problem

[109]. The incidence of human cases has been increasing in countries of five contents. It has been reported from countries in Europe, America, Asia, Africa and Oceania [34,110]. A global analysis show that the expected correlation between animal and human fasciolosis only apparent a basic level. High prevalence in human infections is not found in areas where fasciolosis is a great veterinary problem. For instance, in South America, hyper endemics and mesoendemics are found in Bolivia and Peru where the veterinary problem is less important, while in countries such as Uruguay, Argentina and Chile, human fasciolosis only sporadic or hypo endemic [2,111].

F. hepatic may be acquired by man, but not directly from cattle. The person must ingest the metacercaria in order to become infected [34]. The most common transmission route is ingestion of watercress although; depending up on the geographic location and a variety of edible aquatic plants can be vehicles of transmission [33]. The degree of pathogenicity of Fasciola hepatica to man depends on many factors, particularly the number of worms present and the organ infected, mechanical and the damage are characteristics [112]. The symptoms in Humans vary depending on whether the disease is chronic or acute. During the acute phase, the immature worms begin penetrating the gut, causing symptoms of fever, nausea, swollen liver, skin rashes, and extreme abdominal pain. The chronic phase occurs when the worms mature in the bile duct, and can cause symptoms of intermittent pain, jaundice, and anemia [113]. Global estimate prevalence is between 1.7 and 2.4 Million human infection worldwide and a further 180 Million at risk of infection [33]. The distribution of the disease is predominantly rural, being associated with cattle and sheep breeding [112].

Conclusion and Recommendations

Bovine fasciolosis is an economically important parasitic disease causing great loss of revenue through reduction in productivity of animal in terms of lowered growth rate, meat and milk production, fertility, feed efficiency and draught power in Ethiopia. The disease aggravation depends on distribution of Lymnaeid species snails which are the intermediate hosts of the fluke in areas where the cattle and sheep raised. The young parasite that developed on small intestine of definitive host can cause mechanical damage to the liver tissue up on its migration for feeding that result on bacillary hemoglobinuria and other abnormalities in the cattle. The diagnosis may be conducted by demonstration of fasciola egg from fecal sample in the laboratory and examination of infected animal liver after slaughter. The disease prevention and control involve controlling snail or intermediate hosts, programed use of anthelmintics, facilitating environmental sanitation and good management practices of herds and their grazing conditions. Many reports in Ethiopia show that fascioliasis is mainly an animal disease, causing a great economic burden in the highland areas of the country. There are only a few reported cases of the disease in humans that reported from the areas where the animal fasciolosis are fierily reported. Therefore, the bovine fasciolosis in Ethiopia has significant impact on the economy of the country. So that, the following recommendations are forwarded based on the above conclusion;

- ✓ Control of snails (intermediate host for Fasciola species) is highly recommended to control and prevent the disease.
- ✓ Every animal owner should be able to regularly treat their animals with the appropriate anthelmintics and awareness should be created on the prevention and control methods of fasciolosis.
- ✓ Government bodies should have created effective policies in collaboration with veterinarians to control and prevent the disease throughout the country.
- ✓ Solid epidemiological investigations should be conducted on the current status of bovine fasciolosis in the country.

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