Review Article

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Review on Fascioliasis in Animals and Human

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ABSTRACT: The digenetic trematode parasite, which is a member of the genus Fasciola and has two species: Fasciola Hepatica and Fasciola Gigantica, is the source of the snail-borne trematode disease known as Fascioliasis. Fasciola gigantica is found in tropical regions of Africa and Asia, whereas Fasciola hepatica is primarily found in temperate zones. 2.4 million people are thought to be affected with fascioliasis worldwide, which has recently been recognized as a significant zoonotic parasitic illness that infects humans. Both species spread by snails belonging to the Lymnaeidae family and pose a concern to domestic ruminants and public health. Ingestion of encysted metacercariae of Fasciola species causes infection in both humans and animals. In Egypt, fascioliasis in humans and animals is endemic and causes both clinical and epidemiological health problems. Economic losses from fascioliasis include mortality, decreased carcass weight, decreased meat and milk output, decreased growth and quality of wool, liver damage, and decreased resistance, which makes animals more susceptible to subsequent consequences and other diseases. The parasite's history, taxonomic position, biology, pathophysiology, epidemiology, geographic distribution, economic losses, diagnosis, and control of infection in humans and animals are only a few of the topics covered by the authors in this work.

KEYWORDS: Fascioliasis, Economic losses, Epidemiology, Snails, Encysted metacercariae, and Egypt.

1. INTRODUCTION

According to the World Health Organization, fascioliasis is a neglected zoonotic disease caused globally by genus Fasciola and can be transmitted by snails. It poses a significant threat to human health in endemic places and results in numerous financial losses in livestock production because of morbidity and mortality. Clinical manifestations of fascioliasis in ruminants include ascites, icterus, anemia, and weight loss [1, 2, 3]. Fascioliasis infected livers showed a number of pathological lesions, including bile duct hyperplasia, dilatation of lymphatic vessels in portal regions with extensive fibrosis, necrosis of the liver parenchyma, and Glisson's capsule thickening with fibrosis [4, 5]. Fascioliasis is a global disease that infects roughly 250 million sheep and 300 million cattle worldwide, causing losses of about $3 billion annually. In addition, around the world 2.6 million people get fascioliasis and 180 million people are at risk of infection. Rising human prevalence of fascioliasis does not seem to be directly associated with high animal prevalence [6, 7, 8, 9]. Fasciola gigantica is more limited to the tropical and subtropical climates of Asia and Africa, whereas Fasciola hepatica is more extensively distributed (from temperate to tropical regions on all continents except Antarctica). For transmission, both Fasciola species require an intermediary host, which is a Lymnaeidae snail [10, 11]. Worldwide, there are about 20 species of lymnaeid snails that serve as intermediate hosts for Fasciola species. The transmission of F. hepatica is caused by Lymnaea truncatula, while the transmission of F. gigantica is caused by Lymnaea natalensis [12, 13]. Furthermore, both Fasciola species are transported by Lymnaea columella in numerous parts of the world [14]. Humans and animals can contract fascioliasis by consuming green vegetables and polluted water that contains Fasciola [15]. Although the most popular method for diagnosing fascioliasis with high specificity and sensitivity is the parasitological method

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of detecting Fasciola eggs in the feces, fascioliasis can also be diagnosed based on history and clinical signs [16, 17, 2]. Furthermore, PCR is an accurate way to distinguish between Fasciola species and sequence analysis of the Nuclear Ribosomal ITS-1 gene is the best method to distinguish between two species of Fasciola [18, 19].

1.1. History:

The name Fasciola is derived from (L. dim. of fascia) which meaning a band of a fillet. Fascioliasis in sheep was initially noticed by [20] described the characteristic feature of the parasite and internal anatomy. Zeder elucidated the life cycle and egg hatching in 1803, but in 1914, the full life cycle was thoroughly explained. Although the first recorded human infection dates to the 17th century, there is an evidence that human fascioliasis can be traced back to Egyptian mummies that were discovered to have Fasciola eggs. Mode of transmission of fascioliasis to herbivores animals was identified in 1892 by Lutz, whereas Sinitsin in 1914 described the route of transmission to humans [21, 22, 23].

1.2. Biology of the Agent:

1.2.1. Taxonomy

Phylum platyhelminths involve two classes of parasitic flat worms, (Trematoda and Cestoda). The class Trematoda (flukes) is further divided into two main subclasses, the Monogenia which have a direct life cycle, and the Digenia which need an intermediate host to finish their life cycle. The class Trematoda has many families as Fasciolidae, Schistosomatidae, Paramphistomatidae and Dicrocoeliidae [24]. According to [25] and [24] the taxonomic classification of Fasciola is as the following table

Kingdom:	**Animalia**
Phylum:	**Platyhelminthes**
Class:	**Trematoda**
Subclass:	**Digenia**
Order:	**Echinostomida**
Family:	**Fasciolidae**
Genus:	**Fasciola**
Species:	**F. hepatica, F. gigantica**

Table 1: taxonomic classification of fasciola

1.2.2. Morphological Characteristics

According to the characteristic features of body, length and width, both Fasciola species can be identified morphologically [26, 27, 23]. The adult fluke of F. hepatica is large flattened and leaf-shaped, anteriorly have cone shaped projection followed by a pair of prominent shoulder, with rounded and wider posterior end [28]. When conserved, flukes hange from their original grayish brown color to gray. When the immature fluke enters the liver; it is lancet-shaped and 1-2 mm long. In the bile ducts, flukes reach full maturity and grow to a length of 3.5 cm and a width of 1 cm. F. gigantica is capable of growing up to 7.5 cm in length, therefore being larger than F. hepatica [23]. The adult flukes have two attaching suckers, the oral sucker at the front end and the ventral sucker at the base of the cone. Fasciola species have spine-covered, absorbent tegument on their body surface. The digestive system is simple, start with the oral opening leading to the pharynx, esophagus and a pair of branched intestinal ceca which terminate blindly. Both species are typically hermaphrodites; each worm has branching testes and ovaries. So that Fasciola species are able to reproduce through self- fertilization[24, 29, 23]. Fasciola species produce large, oval, operculated eggs that are yellowish brown in colour. The eggs are 130-145 μm long and 70-90μm wide [28, 23] . Temperature, humidity, and oxygen level are some of the variables that affect how eggs develop in the environment. In both Fasciola species, the eggs require 12–16 days outside the hosts for maturation then the first larval stage (miracidia), hatches in the aqueous medium 4 days after the egg maturation [30, 23].

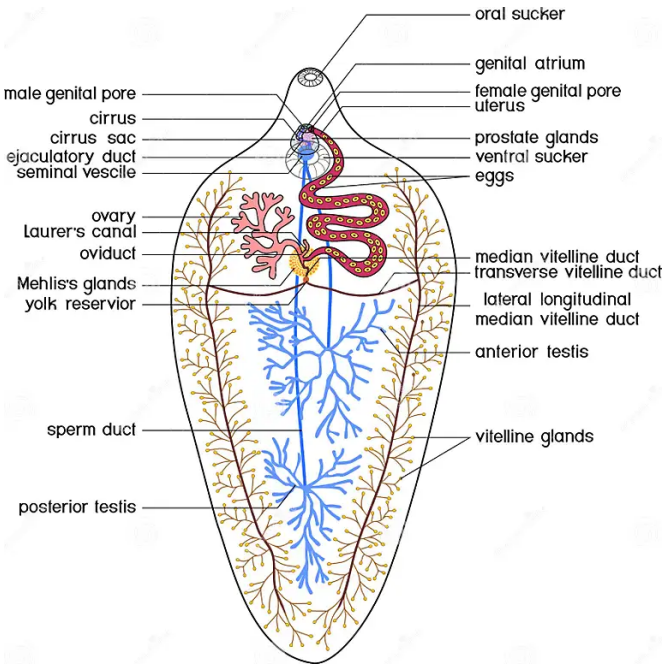

Figure 1: Adult fasciola hepatica fluke diagram

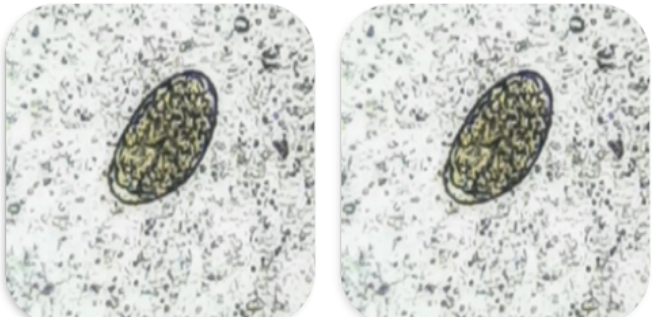

Figure 2: Egg of fasciola hepatica detected in feacal sample in cattle in new Valley governorate,Egypt

1.3. Life Cycle

Fasciola species like the majority of trematodes have a composite life cycle that requires an intermediate host (a freshwater snail of the Lymnaeidae family) where the liver flukes reproduce asexually and a mammalian definitive host where they reproduce sexually [31, 32, 33]. The following phases make up F. hepatica's life cycle: 1. Immature Fasciola eggs (Fig. 2) are discharged in the final host's feces. 2. In water, eggs develop and hatch into miracidium. 3. Miracidium moves in the water until it comes into contact with the aquatic snail (Lymnaeideae-Fig. 4), which is its first intermediate host and dwells in puddles, lagoons, or canals. 4. The parasite develops as a sporocyst, mother redia, daughter redia, and cercaria inside the snail. 5. The movable cercariae emerge from the snail, swim in the water, and attach themselves to the second intermediate host, which could be the water's surface or vegetation. They then lose their tail and encysted as metacercariae. 6. The final host contracts infection after eating the plant that contains the infecting metacercariae. 7. Metacercariae are excreted in the duodenum, releasing juvenile flukes that move through the intestinal wall to the peritoneal cavity, enter the liver, penetrate the bile duct, maturate into adults, and lay eggs in the feces after 8-10 weeks. A single fluke can lay up to 25,000 eggs every day, As illustrated in Fig. 3 [34]

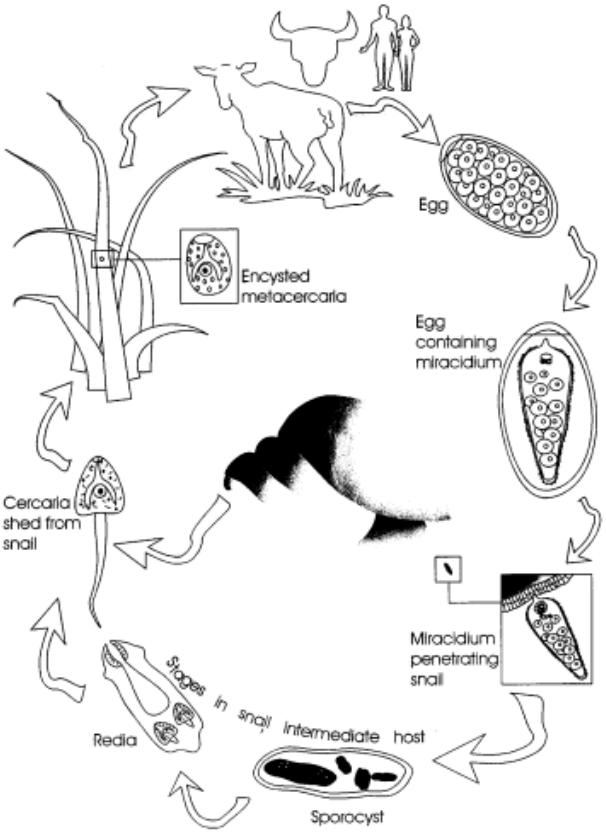

Figure 3: Life cycle of fascia

1.4. Host Range

1.4.1. Definitive Hosts:

The parasite that causes fascioliasis mainly affects domestic and wild ruminants, such as sheep, cattle, buffaloes, goats, and camels. Equine, swine, and rodents are examples of non-ruminant herbivores animals that can contract

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the infection. This parasite can infect humans and use them as accidental hosts [35, 23].

1.4.2. Intermediate Hosts:

The intermediate hosts of Fasciola spp. are freshwater snails belonging to the Lymnaeidae family. The genus Lymanea has a number of snail species that can spread fascioliasis in humans and animals, including Galla, Fossaria, and Pseudosuccinea. Geographical location may affect the species of snails for F. hepatica and F. gigantica [23].

Figure 4: Lymnaeidae species of Lymanea group

2. Epidemiology:

2.1. Geographical Distribution

Fascioliasis is one of the most prevalent helminthic disease affecting both domestic and wild herbivore mammals throughout the world. America, Australia, New Zealand, Africa, Asia, Europe, Columbia, Venezuela, Bolivia, Cuba, Ecuador, Egypt, England, France, Iran, Peru, and Portugal are among the temperate places of the world where F. hepatica can be found. Conversely, F. gigantica is primarily found in tropical and subtropical areas of Asia, the Middle East, and Africa [22]. Intermediate hybrid forms of F. hepatica and F. gigantica have been reported in overlapped regions with both species [36]. There are 51 countries in the world where fascioliasis is known to be present [37]. Fascioliasis is a significant animal health issue that affects both rural and urban regions worldwide. It presents in over 50 nations, particularly those where ruminants are raised [38]. Due to the rise in infected individuals and animals worldwide, fascioliasis has become more common in humans over the past 20 years [39]. It is estimated that 2.6 million people worldwide are infected with fascioliasis, and another 180 millions are at risk of contracting the disease [7]. There have been reports of human fascioliasis in South America, Australia, Europe, Africa, and the Far East additionally; several regions have been identified as endemic for human fascioliasis [40]. The prevalence of fascioliasis infection in children in Peru ranged from 0% to 20% [41]. Human Fascioliasis has been recorded in twelve African nations, including Egypt, Algeria, Morocco, Tunisia, Angola, South Africa, Ethiopia, Ghana, Senegal, Tanzania, and Nigeria [42].

2.2. Factors that Influence the Agent:

Numerous factors influence the transmission of fascioliasis, including climatic conditions including rainfall and temperatures, mammalian hosts, snail habitat, snail food, and the presence of vector snails and parasites [43, 44]. The eggs development in the environment is also influenced by changing in the temperature, humidity and oxygen tension [23]. Fasciola species can survive in sheep for 8-11 years and metacercaria can survive in the environment for up to 12 months and up to eight in wet harvested hay [34] The severity, timing outbreak, and distribution of fascioliasis are influenced by changes in climate because temperature and moisture affect several stages of the parasite's life cycle [45]. Therefore, climate change with high rainfall and milder temperatures has been predicted to increase the risk of fascioliasis [46]. Temperature between (10 and 30°C) and abundant rain act as favorable media for development of the Fasciola in the external environment and inside the snails [34]

2.3. Source and Mode of Transmission of Fascioliasis

It is possible to contract fascioliasis through food or drink. Humans and animals can become infected by consuming infectious encysted metacercariae that are attached to aquatic or semi-aquatic plants (like watercress), drinking water polluted with encysted metacercaria, or consuming metacercaria that may be attached to the surface

of food utensils which have been cleaned by polluted water[47, 23]. Animals typically become infected when they graze in snail-contaminated areas [34] . Numerous vegetable species are consumed raw in salads in Egypt, including non-aquatic plants like Lactuca sativa (Elkhas), Eruca sativa (Elgargeer), Portulaca oleracea (Elregla), Allium porrum (Elkorrat), and Petroselium sativum (bakdoones), which are grown along the banks of water channels and have been found to contain attached liver fluke metacercariae, which raises the risk of human infection in Egypt [48] .

2.4. Economic Significance

Worldwide, fascioliasis causes significant economic losses that are linked to high morbidity, liver condemnation, decreased meat, milk, and wool production, slowed growth, and other complication like decreased fertility, abortion or even mortality. Over 2000 million dollars are thought to be lost each year as a result of fascioliasis in livestock worldwide. The US economy may lose roughly $3 billion a year due to fascioliasis [49, 50]. Fascioliasis has a direct economic impact on livestock production due to liver condemnation because beef liver is a good source of protein for humans and contains certain vitamins like copper, riboflavin, vitamin B12, and vitamin A [51]. Regarding economic significance of fascioliasis in Egypt, Fascioliasis costs roughly $221 USD per cow per year in the Nile Delta region of Egypt [52], In a single year, 7.99% of livers in Aswan governorate are condemned because of fascioliasis, causing total economic losses of about $152718 [51]. Fascioliasis economic losses can be estimated through calculation of the reduction in body weight, mortality of the infected animals, and treatment cost for fascioliasis are 301.55 Egyptian pounds (EGP), 46.22 EGP for each sheep with 4800 EGP for three dead sheep [53]. In 2018, 2019, and 2020, the estimated total percentages of liver condemnation among slaughtered cattle and buffaloes owing to fascioliasis in all Egyptian provinces were 0.98, 0.89, and 0.66%, respectively [54].

3. Pathogenesis:

The pathogenesis of fascioliasis starts when young flukes enter the hepatic tissues. Although the pathogenic course is similar among different hosts, the severity may vary based on the parasitic growth stage, the amount of ingested metacercariae, and Fasciola species [22, 23] . There are two stages to pathogenesis: the first is the hepatic parenchymal phase (Acute Fascioliasis), which happens when large, immature larvae penetrate the liver in short period of time , invade the liver parenchyma, cause severe liver damage and hemorrhage, and then cause abrupt death, especially in sheep. The ductal phase, also known as chronic fascioliasis, follows when a small number of flukes enter the liver over a long period of time (weeks or months), during which time adult flukes damage the biliary mucosa by reaching the bile ducts. Acute and chronic fascioliasis can occasionally coexist together [55, 56].

4. The Disease in Animals:

4.1. Clinical Signs of Fascioliasis in Animals:

Fascioliasis in animals is characterized by significant morbidity and mortality [57]. Clinical signs of fascioliasis in animals depend on infectious dose (amount of ingested metacercariae).and divided into 4 types [58, 23]: Acute Fascioliasis Type I: occur due to ingestion of high amount of infective metacercaria in short time. Animals in this type may be susceptible to sudden death without any previous signs due to hepatic hemorrhages. In addition, animals may suffer from reduced food uptake, lethargy, icterus, Ascites, abdominal pain, severe peritonitis with dyspnea and secondary bacterial infection of liver by Clostridium noyvi may occur and lead to necrotic hepatitis [59, 60] . Acute Fascioliasis Type II: occur due to ingestion of infective metacercaria (1000-5000). Animals die but show pallor, and ascites types [58, 23]. Sub-acute Fascioliasis: caused by moderate number of ingested metacercariae (800–1000). Infected animals suffer from

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lethargy, anemia, weight loss and reduced reproductive performance [61, 58]. Chronic Fascioliasis: caused by ingestion of low amount of metacercariae in long period of time (200-800). This type is characterized by cholangitis and bile duct obstruction due to reproduction of mature flukes in the bile ducts. Clinically this type is characterized by development of bottle jaw, weakness, diarrhea, pallor due to anemia which is related to the blood feeding activity of adult flukes. Leukocytosis and eosinophilia were also noticed [62, 63].

4.2. post mortem Lesions in Animals

There were many characteristic lesions on liver as congestion, enlargement, fibrosis, hemorrhage and necrosis. The bile ducts are dilated, thickened and calcification of the wall. Enlargement of gallbladder also found. Hepatic tissue was creamy or whitish in color while hepatic lymph nodes are dark brown in color. Encapsulated parasites are often seen in the lung [64, 65, 66] .

4.3. Fascioliasis Prevalence in Animals in Egypt:

The prevalence of fascioliasis among slaughtered animals in Elmahalla Elkubra city in Gharbia governorate was 0.2% [67], 14.7% among slaughtered sheep in Menofia province [68]. 30.88% in slaughtered animals at El-Kharga abattoirs in New Valley governorate [65], 27.4% in Sharkia governorate [69], 20% in slaughtered cattle at El-Kharga abattoirs in New Valley governorate [70], 0.26% in Menofia governorate [71], 23.3% in cattle in El-Minia governorate, 86.3% of condemned liver in Abu Simbel, Aswan governorate was due to fascioliasis [51], the overall prevalence of ovine fascioliasis in Beheira, Kafr-Sheikh, Sharkia, Menofia and Gharbia governorate in Egypt was 17.87% [53].

5. The Disease in Humans (Zoonotic Importance of Fascioliasis):

5.1. Clinical Signs of Fascioliasis in Humans:

The incubation period begins when infectious metacercariae are consumed and ends when the first symptoms show up. This phase in humans may last two to three months or longer depending on the infectious dose (the quantity of consumed metacercariae) and the host's immune response [8, 23] . Acute phase: two to four months. This phase is characterized by the presence of fever, which can be mild or moderate in temperature but can reach 40°C or 42°C. It can be irregular and rise in the evening because of tissue damage caused by worm migration through the duodenal wall. Gastrointestinal disorders include diarrhea, anorexia, nausea, vomiting, and loss of appetite, however constipation and vomiting are rare. Chest pain, coughing, dyspnea, and hemoptysis are examples of respiratory symptoms. Hepatomegaly, splenomegaly, hepatic abscess, jaundice, and ascites are examples of liver lesions. It is possible to find increased liver enzymes, urticaria, anemia, weight loss, pruritus, and eosinophilia [72, 73, 74, 8]. Chronic Period: eosinophilia was the only noticeable symptom, and it may last for years without showing any symptoms [75]. Adult flukes can cause blockage and cholangitis and cholecystitis when they are present in the bile ducts. Additional symptoms include jaundice, pruritus, biliary colic, epigastric pain, and the frequent formation of tiny, numerous stones in the gall bladder or bile duct [76, 77] . Ectopic Fascioliasis: Fasciola in regions other than the liver or bile duct. Inguinal nodes, cervical nodes, kidney, bowels, muscles, and infrequently the spinal cord, orbital tissue, brain, breast, bone marrow, thigh, heart, stomach, caecum, appendix, pancreas, spleen, lungs, and inguinal nodes are examples of ectopic locations. After consuming raw, contaminated livers without cooking, Fasciola known as "Halzoun" can enter the pharynx and cause edema, dysphagia, and dyspnea. As a result, the person may die from asphyxia [78, 74, 23].

5.2. Prevalence of Fascioliasis among Humans in Egypt:

With 830,000 cases, Egypt has the highest prevalence of fascioliasis among MENA nations, followed by Yemen

and Iran. The habitat of farmers who immerse vegetables in canals after harvesting them to keep them fresh until all picking is completed is the cause of the highest incidence of fascioliasis in Egypt [79, 80]. In Behera governorate, the prevalence of human fascioliasis was 11.5% by serological examination (IHA) and 5.38% by stool examination [81]. According to coprological examination [30] reported that there was no fasciolid egg was detected in 150 human fecal samples examined in Qena governorate. Additionally, only two of the 23 hepatic fascioliasis cases that had already been infected at Assiut University Hospitals had Fasciola eggs detected by routine fecal analysis [82]. In Dakahlia Governorate, prevalence of human fascioliasis was 0.62% by fecal examination and 0.79%, 1.13% by IHA and ELISA respectively [83]. The infection rate among school children in EL-Behera and Alexandria governorates was 1.4% [84].

6. Diagnosis of Fascioliasis:

Numerous techniques can be used to diagnose fascioliasis at different phases of the disease. While the best way to detect anti-Fasciola antibodies in serum samples during the acute and ectopic phases of fascioliasis is through serological testing, the best way to detect Fasciola eggs in biliary fluid or stool is during the chronic phase, though it can be challenging to find eggs in stool samples because of intermittent elimination [85]. Clinical symptoms and history of fascioliasis incidence on the farm are the main factors used to make the diagnosis. If risk factors like exposure to endemic area or consumption of raw or untreated aquatic plants are present, clinical suspicion may increase. Although signs of fascioliasis are not specific for diagnosis, certain combinations with risk factors may suggest the infection [40, 47, 86]. Numerous techniques, including molecular approaches, parasitological examination, and serological investigation, have been used to diagnose fascioliasis [87]. There is no gold standard test for diagnosis of fascioliasis but detection of eggs in fecal samples is the mainstay of diagnosis of Fasciola infection and is the most used method especially in endemic countries. Multiple stool samples must be taken to confirm infection because excretion of eggs is intermittent [86, 23]. Fecal examination is convenient to use for individual diagnosis or community surveys since it is straightforward, affordable, and simple to do, making it appropriate for use in both the field and the lab. Nevertheless, the detection of eggs in feces has serious limitations, including low sensitivity due to irregular egg shedding in feces, inability to detect fascioliasis during the prepatent period, and limited use as an early diagnostic tool until 3-4 months after infection. Additionally, it is challenging to distinguish between F. hepatica and F. gigantica eggs using this method in every areas have both Fasciola [88, 16]. Serological tests like ELISA, IHA, and IFA are effective at every stage of the disease and may handle issues with fecal examination, such as poor egg shedding caused by low infection rate and the absence of eggs in stool samples during the acute stage, and ectopic fascioliasis also the biliary phase. Conversely, this approach has certain drawbacks, including the inability to differentiate between F. hepatica and F. gigantica infections, as well as between old and current infections; the occurrence of serological cross-reactions; the ineffectiveness of this method for quantification; and the fact that many tests eventually require specific technical training [16, 89]. It becomes impossible to differentiate between F. hepatica and F. gigantica just by focusing on their morphological characteristics when dealing with small specimens and when intermediate forms are present. Therefore, molecular methods are required for species confirmation and to differentiate the intermediate forms [90, 26, 23].It is now possible to identify Fasciola species and hybrid forms because of developments in molecular techniques. Nowadays, variety of molecular markers, including as ITS1, ITS2, 28S rRNA, COX1, and NAD1, are utilized to identify Fasciola species [91, 92]. Numerous molecular techniques, including PCR, nested PCR, real-time PCR,

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and polymerase chain reaction-restriction fragment length polymorphism (PCR-RFLP), are available for diagnosing fascioliasis [93, 23].

7. Treatment of Fascioliasis in Animals and Man:

Once animals diagnose with fascioliasis, fasciolicide treatment should be applied to the all herd, including apparently healthy animals and animals that shed small numbers of eggs. Treatment must be repeated every three months. Flukicides can be used for treating the disease or to prevent outbreak of disease. Because chemical products are eliminated in the milk and be harmful for humans and calves health, so only cows in dry stage and cattle have not yet calved can be treated [40, 34]. The best medication for treating fascioliasis in both humans and animals is triclabendazole since it works against all phases of the disease and affects both young and adult flukes. However, regular and prolonged usage of triclabendazole can result in triclabendazole resistance. Other anthelminthic medications, such as rafoxanide, oxyclozanide, albendazole, closantel, and nitazoxanide, are also used to treat fascioliasis. These medications are good substitute for triclabendazole, particularly in cases of chronic fascioliasis [94, 23]. Fasciola is resistant to most of flukicidal medications; however, a combination of ivermectin and artemether can kill 100% of Fasciola in two days. Furthermore, a single dose of oxfendazole, which is exclusively used in veterinary medicine, can kill Fasciola in ten days and showed excellent effectiveness against F. hepatica in sheep [95, 96, 97]. Treatment for human fascioliasis is recommended to shorten the duration of symptoms and avoid consequences from either acute or chronic fascioliasis. An effective medication for treating both acute and chronic fascioliasis in humans is triclabendazole. Due to difficulties in getting and the fact that triclabendazole resistance in humans infected with fascioliasis which connected to resistance in animals, triclabendazole is currently only registered for use in treating humans in four countries. As a result, new fasciolicidal medications must be developed [98, 99, 86]. Fasciola must be removed during endoscopic retrograde cholangiopancrea-tography in the biliary phase [23] . Triclobenzole treatment failure in humans was first documented in a cattle farmer in the Netherlands. Four cases from Chile, one from Turkey, and seven from Peru followed. Triclobenzole resistance is undoubtedly a significant zoonotic emerging problem [100].

8. Prevention and Control of Fascioliasis:

Livestock treatment is the main management method used in both developed and developing countries. Although there are number of medications that can be used to treat animals, including triclabendazole, albendazole, closantel, and clorsulon, the most commonly used flukicide is triclabendazole. Combining medications from various chemical classes is one of the most important ways to maintain antiparasitic efficacy and stop the development of resistant parasites. Combination of two or more medications typically used to treat sheep liver fluke infections [101, 94]. Reducing the numbers of the intermediate snail host by draining wet pasture and applying molluscicides can help prevent fascioliasis, together with the proper use of anthelminthic medication. Sanitation campaigns, pasture handling, preventing rainwater accumulation, keeping cattle away from pasture in water plants, and, lastly, public health education and awareness-raising regarding the dangers of consuming raw water plants are additional options that may aid in the control of fascioliasis [1, 2]. Fascioliasis may be prevented by keeping the animals away from places where the aquatic snails (Lymnaea) present, drain the soil to eliminate the moisture and eradication of the snail, avoid harvesting of the forage with humidity or add 2% of common salt to the hay grass before consumption or even delay its consumption for 2-3 months to be sure that the metacercariae die, remove grass from wet areas to avoid the risk of infection of the animals and do not allow infected animals with fascioliasis to graze in irrigated grassland to avoid contamination

with eggs of Fasciola. Finally, do not use the manure as fertilizer before dried or treated with copious amounts of lime to destroy the eggs and miracidia of the Fasciola [34].

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