Review on Status of Strongylosis in Ethiopia

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ABSTRACT

Strongylosis is a major internal parasite disease of horses caused by nematodes of the strongylidae family that affects more than 80% of the world’s equines. The horse is a host to a large variety of gastrointestinal parasite species, the most important of which are nematodes of the family Strongylidae, sometimes known as strongyle worms or strongyles. These parasites are common and reside as adults in the equids’ big intestines. Strongyle nematodes are distinguished by a well-developed buccal capsule, the shape and size of which are significant for species identification. Strongyle nematodes of equids (horse, donkey, and zebra) are divided into the subfamilies Strongylinae and Cyathostominae, which are also known as giant and little strongyles. *Strongylus vulgaris* (s.vulgaris) is one of the most common and harmful parasites of horses. Large strongyles exhibit substantial pathogenesis that includes verminous arteritis, visceral organ damage, embolism, or thrombosis that leads to death and is primarily related to migratory parasite larvae. Strongylus species larvae are caused by huge nodules in the caecum and colon wall with significant bleeding, and the nodules burst and release the worm into the lumen of the intestine. Heavyweights might cause bleeding, which can kill the animals.

Keywords: Ethiopia, Epidemiology, Strongylosis.

INTRODUCTION

The global equine population is 122.4 million [1]. In the distribution pattern, developing countries account for 98% of all donkeys, 97% of all mules, and 60% of all horses. In Africa, there are approximately 17.6 million equines, including 11.6 million donkeys, 2.3 million mules, and 3.7 million horses [2,3].

Equines (donkeys, mules, and horses) are valuable working animals in many parts of the world, where they are used for packing, riding, hauling, and plowing. Equine power is essential for both rural and urban transportation systems because it is inexpensive and provides the best alternatives in areas where the road network is insufficiently developed, the terrain is rugged and mountainous, and in cities where narrow streets make it difficult to deliver goods [4,5]. Many people in rural areas utilize mules and donkeys to bring food and other goods to settlements [6,7].

Donkeys and mules work long hours under harsh conditions.
these animals are not working for long periods, they are left to wander and graze on waste. These have the potential to negatively impact their welfare and quality of life. They reduce activity, production, and productivity in the animals, primarily in the reduction of body weight or failure to gain weight, and can even increase mortality in severe cases [5,8].

Strongylidae is divided into two subfamilies: Strongylinae and Cyathostominae, which are frequently referred to as giant and little strongyles, respectively. The key distinguishing feature of strongyle nematodes is a well-developed buccal capsule, the shape and size of which are significant for species identification. By comparing DNA sequences, it has been demonstrated that the taxa with small cylindrical buccal capsules are likely to have evolved from those with large buccal capsules [9,10].

Strongyle is diagnosed through fecal examination for the discovery of the strongyle egg, fecal culture for identification of strongyle larvae, and per rectal examination for aneurysm of the cranial mesenteric artery. Anthelmintic medications are administered to horses to clear adult strongyles from the large intestines and to prevent excessive egg and L3 contamination of pastures. Large strongyle control programs that are effective use strategic treatment and better pasture management. Horses are the most usually infected with mixed species of Strongyle [11,12].

Large Strongyle infections are the most lethal among horse gastrointestinal nematodes, with a reported infection rate of 58.5% [13,14]. However, the overall frequency of Strongylus nematodes remained high in herds where anthelmintics were not utilized. Intrinsic characteristics such as age and gender have been discovered to influence the occurrence of Strongyle infection and egg excretion. Ethiopia has a less contemporary transportation system in rural areas and some towns. Equines are the most commonly employed mode of transportation for both industrial and agricultural products in both rural and urban areas. Helminth parasites were causing significant damage to equine species and had significant economic negative feedback. When a large number of equines graze in the free pasture, equine strongylosis is contaminated and spreads. This improves their performance in the control, management, healthcare, and housing systems of horse species [10,15]. As a result, the purpose of this review is to examine the epidemiology, life cycle, etiology, clinical symptoms, and status of Equine Strongylosis in Ethiopia.

EQUINE STRONGYLOSIS

Etiology

Equine strongyles have a well-developed bacterium capsule on the dorsal wall, where there may be median thickening called the dorsal gutter, which carries the duct of the dorsal esophageal gland. The internal margin of the buccal capsule typically bears leaf-like cuticular structures known as the leaf crown or corona radianta [16].

The following are the scientific classifications of equine strongyles:
- Kingdom: Animalia
- Phylum: Nematoda
- Class: Chromadorea
- Order: Strongylida
- Family: Strongylidae
- Genus: Strongylus
- Species: S. vulgaris, S. edentatus, S. equinus, S. westeri [17].

Morphology

Strongylus worms are reddish because they have consumed blood. Strongylus vulgaris can grow to be up to 25 mm long, S. edentatus up to 40 mm, and S. equinus up to 50 mm. Female worms are longer than male worms. As with other roundworms, the body of these worms is covered with a flexible, but durable cuticle. These species’ cuticles are striated in a circular pattern. The worms have a tubular digestive system with two openings, the mouth and the anus. All species have a well-formed, fairly spherical buccal capsule with basal teeth to cut the host’s tissues (Figure 1) [18].
They eat blood and tissues from the organs they pass through. These worms are plug feeders, which means they tear out a little bit of the tissue in the organs where they live or migrate. They have a neurological system as well, but no excretory organs or circulatory system, i.e. no heart or blood arteries. The female ovaries are bigger, and the uterus terminates in a hole known as the vulva. Males have a copulatory bursa with two spicules that allow them to connect to females during copulation. The eggs are ovoid (Figure 2), tiny (45 x 80 micrometers), thin-shelled, and contain a 16-cell morula [19,20].

**Figure 1.** Close-up of *Strongylus vulgaris* mouthparts [19].

**Figure 2.** Egg of a Strongylus species [19].

**Life cycle of equine Strongylosis**

All Strongyle nematode species have direct life cycles, however, they are relatively convoluted due to somatic migration of larval stages [21]. The quantity of eggs in female uteri varies significantly amongst Strongylus species. The quantity of eggs varies by 50-100 times different species [22]. Adult female Strongyle nematodes lay eggs, which are discharged in the feces into the external environment, where they hatch into first-stage larvae (L1s) at 12-39 °C with enough moisture. The minimum temperature for eggs to hatch is 7-8 degrees Celsius. L1 develops from the egg on the ground per pasture, matures and molts to L2, and finally to L3 [11].

The L3 stage is the infective stage, and it takes around ten days to two weeks to mature from the moment the egg is passed. L3s have a protective coating on the outside and are more resistant to cold and dehydration than L1s and L2s. Dehydration may hinder L3s from exiting the feces and interacting with herbage. Most larvae climb no higher than 10 cm from the soil surface and can move 15 cm horizontally; after rain, L3s migrate most efficiently from excrement to adjacent herbage [2,15].

They proceed to the small intestine when the host consumes L3, lose their external covering, and begin the internal phase of development. The removal of the protective layer is triggered by physiological/biochemical circumstances in the
host’s gut. Large Strongyle nematode larvae emerge from the sheath by an anterior cap, whereas small Strongyle nematode larvae escape via a longitudinal slit in the esophagus [11,22]. Using an artificial intestinal fluid containing trypsin, pancreatin, sodium bicarbonate, and sodium dithionite, the outer covering was removed at 38°C in 3 hours [23].

The internal phase of large Strongyle nematode larval development includes somatic movement, whereas small Strongyle worm larvae burrow into caecum and colon glands and become encysted with no further migration.2004 [24,25]. The next molting happens in the submucosa, ie L4, on day 4 or 5. The L4s gradually migrate up the artery system of the intestine, working against the flow of blood. The larvae have reached the cecal and ventral colic arteries by the eighth day. When these larger arteries are reached, the migration path is identified by a twisting thread of fibrin on the intima, and larvae can be observed in mural thrombi by day 14. Between 11 and 14 days, the ileocecal and cranial mesenteric arteries are reached. By the 19th day, larvae can be detected in nearly any part of the vascular system, but they are always most prevalent in arteries adjacent to the origin of the cranial mesenteric artery [20].

Molting to the fifth stage (L5) can occur as early as 9 days and as late as 120 days. Most larvae at this stage are pre-adults, measuring up to 18 mm in length. S. vulgaris larvae often stay in the artery location until they molt to the fifth stage, while many fourth-stage larvae are carried away before the final molt. The size of the larvae and the shove from the blood flow are critical elements in the separation of larvae from arterial lesions. Pre-adult larvae travel to the tiny arteries on the serosal surface of the large intestine and the terminal small intestine [25].

Epidemiology

Strongylus is a frequent horse disease that can be fatal if control measures are not implemented. Egg deposition increases in the spring and continues high throughout the summer in places with cold winters and moderate summers. Temperatures are conducive to larval growth, and significant contamination with infective larvae may occur in late summer, early summer, and early autumn when young susceptible horses are present. S. vulgaris larvae can be found throughout the winter. When the summers are hot and dry, only a tiny percentage of strongyle eggs develop into larvae, which may be short-lived, but constant re-infection maintains pasture contamination high. The start of sickness after ingestion of a high number of larvae is dependent on the parasite’s maturation period in the host and whether the pathogenic stage is immature or adult [14,27].
Pathogenesis

*S. vulgaris* is the most pathogenic of the giant Strongylus nematodes due to its extended migrations through the mesenteric artery system and its branches before maturing in the cecum and colon (at least 4 months). Larval migration damages the smooth endothelial surfaces of arteries, creating a focal point for clot formation. These clots (thrombi) are caused by inflammation and a thickening of the artery walls. The larval stages of large and small Strongyles are responsible for the damage they produce. Small Strongyles have small buccal capsules and feed on the mucosa superficially [28,29].

Intussusception is occasionally seen as colic and can be caused by a thickened cranial mesenteric artery on the mesenteric plexus. *S. vulgaris* larvae are significant due to their placement in the artery, namely, the anterior mesenteric artery, which provides blood to the large intestine [30]. Their adhesion to the vessel lining generates a roughened lining, creating an optimal place for fibrin and leucocytes to be removed out of circulation thrombi. Both large and small strongyles cause harm to the intestinal wall lining at the attachment. They may ulcerate as they move from place to place. Bleed or become infected with germs. Infections appear to be more severe in immature and young horses, with sufficient worms causing anemia, edema, digestive disruption, and emaciation [29,31].

Clinical Signs

Prepatent durations (the time between infection and the detection of eggs in feces) for members of this genus range from 6 months for *S. vulgaris* to 10-12 months for *S. edentatus*. *S. vulgaris* causes poor hair coat, reduced performance, weight loss, anemia, chronic low-grade fever, verminous arthritis, and colic [30]. Ill-thrift to death [32], diarrhea, and anorexia are common [33]. Strongyle parasite infection is common in working horses, which can lead to stumpy performance and a shorter life expectancy [34]. Hematological changes include decreased TEC and packed cell volume (PCV) [35], decreased hemoglobin concentration, and eosinophilia [33].

Although there do not appear to be any clinical indications that can be clearly attributed to mucosal-feeding adults of all three Strongylus species, it is widely believed that they can have a major impact on afflicted horses [30,33]. Horses afflicted with big Strongylus show general clinical indications of pale mucous membranes, poor weight gain, and even weight loss, as well as dull, glaring coats [35]. These clinical indications, however, are common in most parasite infections of the digestive tract. The “wormy horse” is often classified as “unthrifty” because of its poor performance and appearance. Because of the widespread use (some would argue misuse) of anthelmintics and the adoption of parasite management programs by veterinarians and the horse-owning public, horses like these are now relatively uncommon in the United States. Large Strongylus in the colon is usually blamed for anemia, emaciation, poor coat, and poor performance [36,37].

Diarrhea is a more prevalent symptom of small Strongyle infections than large Strongyle infections. These clinical indications are caused by adult worms’ eating habits, in which they seize a portion of mucosa with their huge jaws and devour it, causing significant bleeding at the bite site and the creation of an ulcer [31]. Necropsies reveal that there are significantly more ulcers than adult giant Strongyle nematodes in the cecum and colon, implying that these worms feed and then travel to a new location. It also means that mucosal tissues are their primary source of nutrition and that blood is only consumed as part of the mucosal meal. This suggests that the giant Strongyle nematode is more correctly defined as a mucosal feeder than a bloodsucker [38,31,37].

Fever in *S. vulgaris* infection is caused by tissue damage or a poisonous chemical produced by larvae. The most consistent alteration in early *S. vulgaris* infection would be a rapid increase in total white blood cell (WBC) counts. During the first three weeks, these values skyrocket to levels ranging from 17,000 to 22,700/mm3 [39]. After the second week, eosinophil counts will rise but show no change in acute infection. Increases in serum total protein and globulin fractions occur as early as the first week after infection. Thrombus formation can block arteries, producing infarction of intestinal walls and/or intermittent lameness, and is frequently linked with clinical indications of extreme pyrexia, anorexia, severe colic, and mortality [25,39].

Diagnosis

Symptoms have no diagnostic value. Traditionally, diagnosis has relied on the use of fecal flotation [40]. Because morphological differentiation between Strongyle eggs of different species is impossible, fecal samples are cultivated to facilitate the growth of L3s, which can then be collected for investigation and the species easily identified [41]. A method for recognizing mucosal larval stages would be useful in larval diagnostics. A copro-antigen ELISA demonstrated moderate to strong diagnostic sensitivity and specificity, as well as a favorable connection with worm counts [41,42].

The blood image changes associated with *S. vulgaris* are similar to those seen in bacterial infections. Blood biochemical and hematological parameters can be altered in a subset of sick horses. Hypoalbuminemia is a typical
observation in naturally infected horses, and it is most likely owing to increased intestinal permeability. Natural infections have also been linked to an increase in γ-globulin levels in the blood. Serum fructose amines (glycated serum proteins) were shown to be significantly lower in horses with experimental cyathostomin worm infection [43,44]. The eggs of the three Strongyle species are impossible to differentiate. To isolate the larvae, coproculture is frequently required, which is time-consuming. A commercially available test kit has not yet resulted from research to build accurate and simple diagnostic tools. The clinical history, clinical manifestations of the disease and identification of Strongyle eggs in the feces of infected animals using the direct smear method are used to make the diagnosis [13].

### Table 1. The distribution of equine Strongylosis in Ethiopia

<table>
<thead>
<tr>
<th>S.No</th>
<th>Prevalence of Equine Strongyles</th>
<th>Country(area) in Ethiopia</th>
<th>Author name and year of publication</th>
</tr>
</thead>
<tbody>
<tr>
<td>1</td>
<td>100%</td>
<td>Wonchi, Oromia</td>
<td>[4]</td>
</tr>
<tr>
<td>2</td>
<td>100%</td>
<td>Highland of the whole province, Amahara</td>
<td>[1]</td>
</tr>
<tr>
<td>3</td>
<td>100%</td>
<td>Dugdabora, Oromia</td>
<td>[1]</td>
</tr>
<tr>
<td>4</td>
<td>98.20%</td>
<td>Western highland of Oromia</td>
<td>[45]</td>
</tr>
<tr>
<td>5</td>
<td>87.70%</td>
<td>Gondar, Amhara</td>
<td>[46]</td>
</tr>
<tr>
<td>6</td>
<td>68.10%</td>
<td>Mersa Town of South Wollo Zone, Amhara Regional State, Ethiopia</td>
<td>[31]</td>
</tr>
<tr>
<td>7</td>
<td>46.10%</td>
<td>Bati Town, East shoa, Oromia, Ethiopia</td>
<td>[7]</td>
</tr>
<tr>
<td>8</td>
<td>48.17%</td>
<td>in and around Shone Town, Hadhya zone, Southern Ethiopia</td>
<td>[59]</td>
</tr>
<tr>
<td>9</td>
<td>54.20%</td>
<td>Hosaena District, Southern Ethiopia</td>
<td>[5]</td>
</tr>
<tr>
<td>10</td>
<td>67.19</td>
<td>Alage District, Ethiopia</td>
<td>[12]</td>
</tr>
<tr>
<td>11</td>
<td>53.13%</td>
<td>Mekelle City, Northern Part of Ethiopia</td>
<td>[42]</td>
</tr>
</tbody>
</table>

### Treatment

To prevent excessive contamination of pastures with eggs and L3s, equines are typically treated with anthelmintic medications to clear adult Strongyles from the large intestines. Thiabendazole is commonly used, and numerous other medications, including benzimidazole, tetrahydropyrimidines, and organic phosphorus compounds, have been developed or approved for use in adult horses [47,48].

Benzimidazoles (e.g., thiabendazole, cambendazole, fenbendazole, and oxibendazole), pyrantel, and macrocyclic lactones such as ivermectin and moxidectin [24,49]. In the 1990s, treatment intervals for adult horses were 8 weeks for ivermectin and 4-6 weeks for other anthelmintics. Many combinations of macrocyclic lactones (abamectin, ivermectin, moxidectin), including ivermectin combined with pyrantel (tetrahydropyrimidine) and ivermectin combined with praziquantel (pyrazinoisoquinolin derivative), a pharmaceutically formed generic paste containing ivermectin 4% were tested for their effectiveness to control gastrointestinal nematodes of horses [50,51]. Ivermectin formulations with similar efficacies had varying EPG reductions [49,52].

The efficacy stages of the studied medicines varied against *S. edentatus*, *S. equinus*, and *S. vulgaris*. The generic paste (ivermectin 4%) was less effective than the conservative medications. On day 14 after treatment, the efficacy of oxafex, ivomec, and farbendazl was determined to be 94.7, 98, and 81%, respectively. On the 28th post-medication day, it was 100%, 96%, and 86%, respectively [47]. To prevent the development of anthelmintic resistance, it is now recommended to drastically lower treatment intensity [53]. A nonsteroidal anti-inflammatory medication must also be used in cases of severe enteropathy. For horses, a single intravenous dose of 0.6 mg/kg body weight meloxicam once a day is indicated [54]. The usual therapy at numerous intervals should be abandoned, and parasite control should be maintained with significantly fewer anthelmintics [48,55].

### Prevention and Control

During the 6-month period following infection, which includes the worst season for Strongyle transmission, prevention by routine deworming of horses is unnecessary in all regions. During this time, environmental factors primarily limit the development of new parasites. Even if horses have a high egg count during that time, just a small percentage of those eggs develop into adulthood [56].
a result, the climate achieves parasite control goals, and compound treatment is not required [56,57]. Because even a small number of infective larvae can be fatal, foals should be checked for Strongyle eggs regularly and treated with appropriate anthelmintics. Climate change cannot clear pastures efficiently from one grazing season to the next [21]. Herbal substances such as anthelmintics against strongylosis are yet to be investigated. Overstocking is an important measure to lower the risk of infection because if too many animals share the same pastures, horses would choose to eat grass polluted by dung, increasing the risk of eating infected larvae. Each animal should ideally be given 2 to 3 acres (0.8 to 1.2 hectares) of land. Too-humid pastures should be drained if possible; the dryer the pasture, the lower the survival of infective larvae and the risk of infection for the horses. Manure should be removed regularly, and pastures should not be fertilized with new manure. Water tanks should be cleaned regularly, and grazing near them should be avoided. Because they are damp and regularly frequented, they are heavily polluted with infective larvae [42,58,59].

Stable hygiene is essential for preventing pests indoors. They must be cleaned regularly, dung must be removed every day, and bedding must be replaced regularly. Humidity must be kept as low as feasible, for example, through appropriate ventilation. Alternate grazing with livestock that is not vulnerable to Strongylus infection (cattle, sheep) may also be explored, however, livestock can carry other parasites that affect horses. Before being allowed to share pastures and premises with other horses, horses entering a farm must always be examined for pre-existing infections (e.g., through adequate fecal inspection) or treated with a broad-spectrum anthelmintic. When in doubt, quarantine measures should be considered [19,56].

CONCLUSION
Equines are crucial animals for developing countries’ life systems, particularly in Africa. These creatures are especially useful in transportation systems. Equines give limitless services to humans, so they are routinely neglected and mistreated. Strongyle infection in equines is widespread and should be considered one of the most important equine diseases; specifically, large strongyles are the most important parasites of equines and are more common in untreated equines, exerting a significant economic impact when they are raised. Infections caused by big strongyles are very frequent in polluted surroundings. These parasites will remain the most dangerous parasitic helminths in underdeveloped countries. Based on the conclusions stated above, the following recommendations are made:

- To avoid excessive pasture pollution, strategic treatment and enhanced pasture management should be used.
- Any new animals should be separated from a treated group for 48-72 hours before being brought to the group, and anthelmintics should be administered.
- Equines should be kept apart in terms of housing and grazing.
- It is recommended that the government support an equine health promotion program.

DECLARATION
ETHICS APPROVAL AND CONSENT TO PARTICIPATE
Not applicable.

CONSENT FOR PUBLICATION
Not applicable.

AVAILABILITY OF DATA AND MATERIALS
All the datasets generated or analyzed during this study are included in this manuscript.

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All authors have nothing to disclose in this work.

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All authors contributed to data collection, study conception, reference search, manuscript writing, and editing, and all authors have approved the submission of the final manuscript.

REFERENCES


