WORLD JOURNAL OF ADVANCE HEALTHCARE RESEARCH

ISSN: 2457-0400
Volume: 2.
Issue: 1.
Page N. 22-28
Year: 2018

Review Article www.wjahr.com

REVIEW ON DICTYOCAULOSIS AND ITS IMPACT IN EQUINE

Tagesu Abdisa*

Jimma University, School of Veterinary Medicine, Oromia, Ethiopia.

Received date: 13 November 2017 **Revised date:** 04 December 2017 **Accepted date:** 25 December 2017

Corresponding author: Tagesu Abdisa
Jimma University, School of Veterinary Medicine, Oromia, Ethiopia.
Email ID: abdisatagesu@gmail.com, atagish5@gmail.com,

ABSTRACT

Lungworms are parasitic nematode worms of the order Strongylida that infest the lungs of vertebrates. The lungworms in the super family Trichostrongyloidea include several species in the genus Dictyocaulus which infest hoofed animals, including most common domestic species. Dictyocaulus arnfieldi *is the true lungworm affecting donkeys, horses, mules and zebras and Lungworms are widely distributed throughout the world but are particularly common in countries with temperate climates, and in the highlands of tropical and sub-tropical countrie. The epidemiology of lungworm disease is largely concerned with factors determining the number of intensive larvae on the pasture and the rate at which they accumulate. The adult worms are most often found in the small bronchi and their eggs, containing the first stage larvae, hatch soon after being passed in the faeces. The characteristic lesion is similar in both horses and donkeys and is somewhat different from bovine parasitic bronchitis. Despite the prevalence of patent D. arnifieldi infection in donkeys, overt clinical signs are rarely seen; however, on close examination slight hyperpnoea and harsh lung sounds may be detected. Diagnosis is based on clinical signs, epidemiology, presence of first-stage larvae in feces, and necropsy of animals in the same herd or flock. The Baermann technique is the best laboratory examination used to separate lungworm larvae from faecal material. The treatment should be by broad spectrum anthelmintic like ivermectin, fenbendazole. Preventative measure often recommended just prior to their first grazing season, followed by additional treatments depending on the infestation level of the pastures and the residual effect of the administered anthelmintic. The impact of dictyocaulus arnifield in horse population is range from impaired development and performance to death. Therefore, the death of horse may decrease the economy and population of equines.The perspective of this manuscript paper is to overview the fact of lungworm and its impact in equine animals and also the way how to control and prevent lung worm of equines.*

KEYWORD: *Baerman techniques; Dictyocaulus arnifield; Equine; Fenbendazole; Lund worm.*

INTRODUCTION

Equines (Donkeys, mules and horses) play an important role as working animals in many parts of the world, for packing, riding and carting. Equine power is very crucial in both rural and urban transport system. This is because of its cheapness and availability and so provides the best alternative transport means in places where the road network is insufficiently developed and the landscape is rugged and mountainous and in the cities where narrow streets prevent easy delivery of merchandise. In some areas of North West Kenya and Southern Ethiopia, donkey meat is a delicacy and the milk believed to treat whooping cough.[1] Equines are working animals which has been serving as animal burden for long period of time and rendering valuable services mostly as pack animals. The equine animals are serving the people in developing county by transportation and packing the goods throughout the country where the modern transportation is scant, unaffordable and inaccessible.[2] However, the equine animals are suffering from infectious and non infection diseases, the most common infectious disease which disturbing the health of equine are like epizootic lmphangite, strangle, pneumonia, parasitic disease, African horse sickness, glander, ulcerative lymphangite and also non infectious disease like colic and etc.[3]

Lungworms are widely distributed throughout the world providing nearly perfect conditions for their survival and development but are particularly common in countries with temperate climates, and in the highlands of tropical and subtropical countries. *Dictyocaulidae* are known to

exist in East Africa (Ethiopia, Kenya and Tanzania) and South Africa.[4] Even though mules and donkeys have often been described as sturdy animals; they succumb to a variety of diseases and a number of other unhealthy circumstances. Among these, parasitic infestation is a major cause of illness. *Dictyocaulus arnfieldi* is the true lungworm affecting donkeys, horses, ponies and zebras and is found throughout the world. It is relatively well adopted parasite of donkeys (*equus assinus*) but tend to be quite pathogenic in horses, where this parasite is endemic. Donkeys and their crosses (Mules) are the natural hosts for lungworm and the condition in horses is usually found in those that have been in accompany of donkeys and mules.[5]

Adult Dictyocaulus worms are slender, medium sized roundworms which have a whitish to grayish color. This parasite has both digestive system and nervous system but have no excretory system.[6] Animals become infected with lung worm infection mainly while grazing, but infection can also happen indoors through contaminated hay or bedding.[7] The major pathologic changes occur in equine after infection can be divided into prepatent, patent and post patent phase. However, the pathogenic effects of lungworm depends on their location within the respiratory tract, the number of infective larvae ingested, the animal immune status, body condition of the animals, on the nutritional status and age of the host.[8,9]

Body condition can be considered as a major risk factor to lung worm infection in equines. This is due to the fact that, poorly nourished animals appear to be less competent in getting rid of infection although it is not unusual for well-fed animals to succumb to the disease provided the right environmental conditions are made available.[10] Lung worm infection may have different severity among different age groups. Hence, the older and younger animals are taught to have higher sensitivity as they have decreased immunity to combat infections.[11]

LUNG WORM IN EQUINE

General Description of Lungworm Parasites

Lungworms are parasitic nematode worms of the order Strongylida that infest the lungs of vertebrates. The name is used for a variety of different groups of nematodes, some of which also have other common names; what they have in common is that they migrate to their hosts' lungs or respiratory tracts, and cause bronchitis or pneumonia.[12] The lungworm will gradually damage the airways or lung tissue by inciting an inflammatory reaction inside the tissue. Ultimately, the Parasites survive and reproduce in the respiratory tissues. The most common lungworms belong to one of two groups, the super family Trichostrongyloidea or the super family Metastrongyloidea, but not all the species in these superfamilies are lungworms.[13]

The lungworms in the super family Trichostrongyloidea include several species in the genus *Dictyocaulus* which infest hoofed animals, including most common domestic species. Different species are found in cattle and deer (*D. viviparus*), donkeys and horses (*D. arnfeldi*), and sheep and goats (*D. filaria*). These animals have direct life-cycles. The lungworms in the super family Metastrongyloidea include species that infest a wider range of mammals, including sheep, goats and pigs but also cats and dogs. These include *Metastrongylus apri*, found in pigs; *Oslerus osleri* found in dogs; and *Aelurostrongylus abstrusus* found in cats. Some of these have indirect, and complex, life-cycles; several of them involve slugs or snails as intermediate hosts, where the habit of sniffing at slug trails, or even licking them, causes the parasite egg to enter the dog's respiratory tract. In the case of *A. abstrusus* the cat is normally infected by eating a bird or rodent that has itself eaten the original host.[14]

Dictyocaulus arnfieldi is the true lungworm affecting donkeys, horses, mules and zebras and is found throughout the world.[15] It is a relatively well adopted parasite of donkeys but tend to be quite pathogenic in horses, where this parasite is endemic.[6] The first three lungworm listed above belong to super family Trichostrongylidea and have direct life cycle; others belong to Metastrongylidea and, except *F.osleri* and *C.aerophila* have indirect life cycle. Diseases caused by the three *Dictyocaulus* species are of most economic importance. The cattle lungworm *Dictyocaulus viviparous* is common in Northwest Europe and is the cause of severe outbreaks of “husk” or “hoose” in young grazing cattle. The lungworm of sheep and goat, *Dictyocaulus filarial* is less pathogenic but does cause losses especially in Mediterranean countries, although it also recognized as a pathogen in Australia, Europe and North America. *Dictyocaulus arnfeildi* can cause severe coughing in horses and because patency is unusuall in horse (but not in donkeys) differential diagnosis in disease due to other respiratory disease can be difficult. *Mullarius capillaries* present worldwide and, while usually nonpathogenic in sheep, can cause severe signs in goats. Other lungworm infections generally cause occasional sporadic infection on many species in many countries.[9]

Morphology of Dictyocaulusarnfieldi

Adult *Dictyocaulus* worms are slender, medium sized roundworms, up to 8 centimeter long. Females are about one third longer than males. They have a whitish to grayish color. As in other roundworms, the body of these worms is covered with a cuticle, which is flexible but rather tough. The worms have a tubular digestive system with two openings, the mouth and the anus. They also have a nervous system but no excretory organs and no circulatory system, i.e. neither a heart nor blood vessels. The female ovaries are large and the uteri end in an opening called the vulva. Males have a copulatory bursa with two short and thick spicules for attaching to the female during copulation. The eggs of *Dictyocaulus arnfieldi are approximately* 60x90 micrometers. They

have an ovoid shape and contain a fully developed L1 larva (**Figure 1**).[7] Lungworm larvea are slender and 25 to 70 millimeters long. The *D. arnfeildi* larvae resemble those of *D.viviparous* but the tail ends in a small spine.[9]

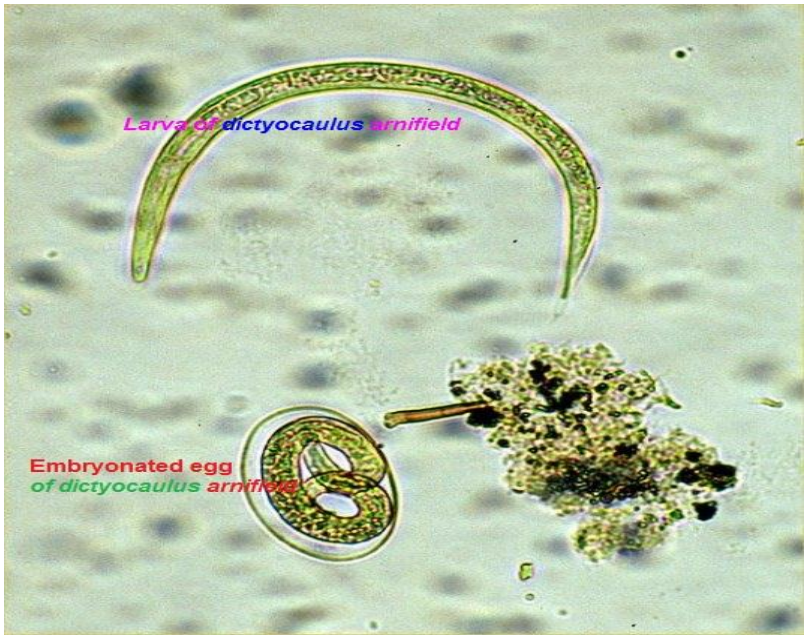

Figure 1: Egg and larva of *Dictyocaulus arnifieldi* (Source: (https:// www.vetstream.comtreatequislabtestfeces-parasitology).

Epidemiology

Lungworms are widely distributed throughout the world but are particularly common in countries with temperate climates, and in the highlands of tropical and sub-tropical countries.[16] However, it occurs more frequently as a clinical problem in some warmer areas such as Mediterranean countries, the Middle East and India. Lungworms are also distributed in the highland and semi-lowland areas of Kenya, north eastern and south western Tanzania, Ethiopia, Lesotono and republic of South Africa.[17]

The epidemiology of lungworm disease is largely concerned with factors determining the number of intensive larvae on the pasture and the rate at which they accumulate. The third stage larvae are long living in damp and cool surroundings. Warm and wet summers give rise to heavier burdens in the follow autumn and spring. Horses are not the favorite host of this parasite and do not usually transmit the disease to other horses. In most instances, horses acquire this disease when pastured with donkeys.[18]

Under optimal condition the larvae may survive in the pasture for a year. They are quite resistant to cold although it generally delays their maturations. They can withstand temperature of 4-5 degree Celsius; Larvae can over winter in cold climates.[19] Most outbreak of verminous pneumonia occur during cool season specially autumn and early winter because the larvae stages of the causative worms tolerate and prefer low temperatures.[4]

The natural host of the parasite is donkey, and comparably, large numbers of parasites can accumulate in the lungs of this host without clinical signs. Donkeys and mules can act as a reservoir for horses.[20] *Pilobolus* fungi may play a role in the dissemination of *D. arnifieldi* larvae from faeces, as *D. viviparus. D. arnfieldi* is found worldwide, particularly in areas with heavy rainfall.[17]

Life Cycle

The detailed life cycle is not fully known, but is considered to be similar to that of bovine lungworm, *Dictyocaulus viviparus* except in the following respect. The adult worms are most often found in the small bronchi and their eggs, containing the first stage larvae, hatch soon after being passed in the faeces.[17] *Dictyocaulus* worms have a direct lifecycle, i.e. there are no intermediate hosts involved. Adult females lay eggs in the airways of infected hosts. These eggs are transported to the pharynx within respiratory secretions. From the pharynx these eggs are coughed out, into the mouth to be swallowed or directly to the outside. Those that are swallowed release the L1 larvae in the gut, which are shed in the faeces. Once in the environment, L1-larvae develop to infective L3 larvae in about 1 week. These larvae show a low motility and remain close to the droppings. Animals become infected mainly while grazing, but infection can also happen indoors through contaminated hay or bedding. Once ingested and in the host's gut infective larvae penetrate into the gut's wall and reach the lymphatic nodules where the molt to L4 larvae. Through the thoracic duct and the jugular vein they reach the heart and are pumped to the lungs. Once in the lungs they are stopped in the lung capillaries, cross their wall and reach the bronchioles, bronchi or the

trachea where they complete development to adult worms (**Figure 2**).

The prepatent period (time between infection and first larvae shed with the faeces) lasts about 4 weeks. However, larvae in the lungs may become arrested (dormant, hypobiotic, inhibited) for up to 5 months. These larvae resume development at early spring and re-infect the pastures during the next season.[7]

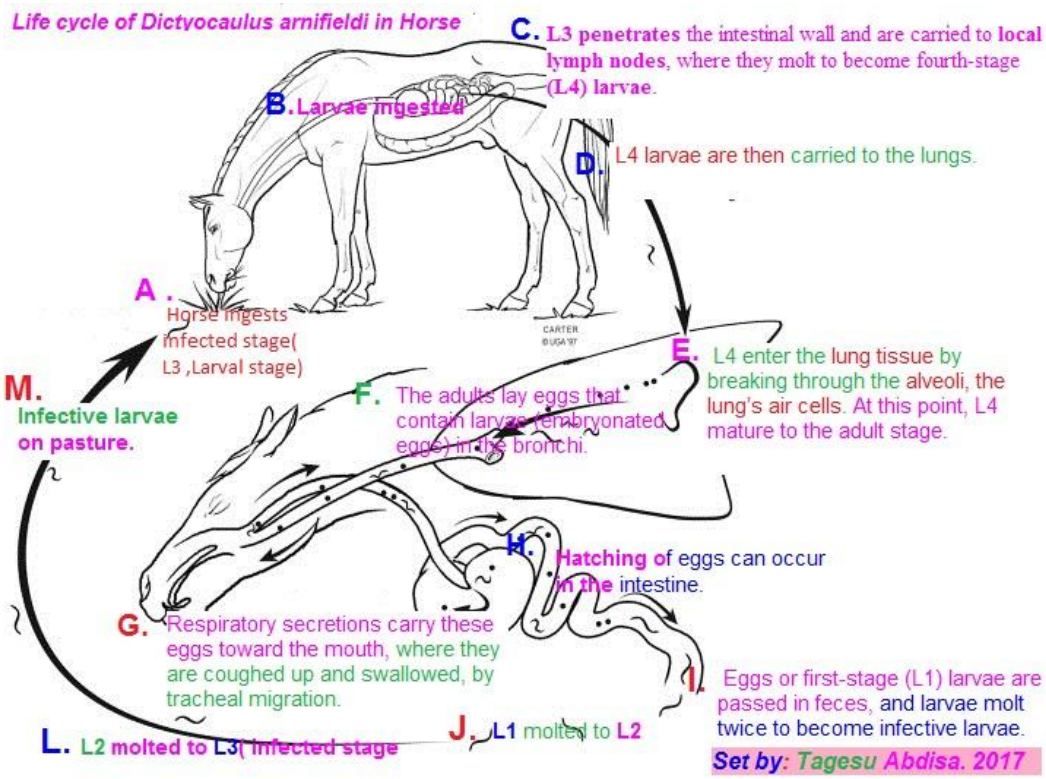

Figure 2: Life cycle of equine lungworm (*D.arnfeildi*).

Pathogenesis

The characteristic lesion is similar in both horses and donkeys and is somewhat different from bovine parasitic bronchitis. In the caudal lung lobules particularly, there are raised circumscribed areas of over-inflated pulmonary tissue 3.0-5.0cm in diameter .On section, at the centre of each lesion is a small bronchus containing lungworms and mucopurulent exudate. Microscopically, the epilhelium is hyperplastic with an increase in the size and number of mucus-scrolling cells while the lamina propria is heavily infiltrated and often surrounded by inflammatory cells, predominantly lymphocytes.

The pathogenic effect of lungworms depends on their location within the respiratory tract, the number of infective larvae ingested and the immune status of the animal. This may be divided in to four phases.[17] The penetration phase: In phase of penetration the larvae are moving toward the lung, but no any apparent pulomonary lesion.[21] The Prepatent phase: The small bronchioles are blocked by exudates, which obstruct the airways, and this may result in the collapse of the lung tissue distal to the blockage. Towards the end of this phase bronchitis develops, characterized by mucus containing immature lungworms in the air ways, which may only be seen with the aid low-power microscope, and by cellular infiltration of the epithelium.[22] During the prepatent phase of *D. viviparus* infection, the main lesion is blockage of bronchioles by an infiltrate of eosinophils in response to the developing larvae; this results in obstruction of the airways and collapse of alveoli distal to the block. Clinical signs are moderate unless large numbers of larvae are ingested, in which case the animal may die in the prepatent phase with severe interstitial emphysema and pulmonary edema.

In the patent phase, the adults in the segmental and lobar bronchi cause bronchitis, with eosinophils, plasma cells, and lymphocytes in the bronchial wall; a cellular exudate, frothy mucus, and adult nematodes are found in the lumen. The bronchial irritation causes marked coughing, and the entire reaction leads to increased airway resistance. A major component of the patent stage is development of a chronic, nonsuppurative, eosinophilic, granulomatous pneumonia in response to eggs and first-stage larvae aspirated into alveoli and bronchioles. This is usually in the caudal lobes of the lungs and is severe when widespread; in combination with the bronchitis, death may result. Interstitial emphysema, pulmonary edema, and secondary bacterial infection are complications that increase the likelihood of death. Survivors may suffer considerable weight loss.[13,14,24]

Heavily infected animals, whose lungs contain several thousand developing worms, may die, due to respiratory failure of following the development of severe interstitial emphysema and pulmonary oedema.[14,17]

Clinical Sign

Large numbers of larvae entering in to lungs and many adult worms in the bronchi can cause irritation of the mucousa. Mucus is produced, causing difficulty in breathing, severy coughing,tachypnea, unthriftiness in the older horses and loss of appetite. Severy bronchitis is often accompanied by chronic pneumonia, pulmonary edema and secondary bacterial infection. Heavy infections may cause death in the foals.[14] Despite the prevalence of patent *D. arnifieldi* infection in donkeys, overt clinical signs are rarely seen; however, on close examination slight hyperpnoea and harsh lung sounds may be detected. This absence of significant clinical abnormality may be partly a reflection of the fact that donkeys are rarely required to perform sustained exercise. Infection is much less prevalent in horses. However, patent infections may develop in foals and these are not usually associated with clinical signs. In older horses infections rarely become patent but are often associated with persistent coughing and an increased respiratory rate.[17] Donkeys usually show no disease signs and can be silent carriers and shedders of this parasite, which causes clinical signs in horses.[23]

Diagnosis

Diagnosis is based on clinical signs, epidemiology, presence of first-stage larvae in faeces, and necropsy of animals in the same herd or flock. Diagnosis is based on clinical signs, epidemiology, presence of first-stage larvae in feces, and necropsy of animals in the same herd or flock. Bronchoscopy and radiography may be helpful. Larvae are not found in the faeces of animals in the prepatent or postpatent phases and usually not in the reinfection phenomenon. ELISA tests are available in some laboratories. Bronchial lavage can reveal *Dictyocaulus arnfieldi* infections in horses.[24]

Verminious pneumonia is easily confused clinically with bacterial bronchopneumonia with acute and chronic interstitial pneumonia and with viral pneumonia. The disease usually occurs in outbreak form in summer and autumn.[19] The diagnostic methods of lungworms are described as the following ways in details.

Clinical diagnosis

Typical signs and symptoms are heavy coughing (often paroxysmal), accelerated and/or difficult breathing and nasal discharge. Affected animals lose appetite and weight. Severe infections can also cause pneumonia (lung inflammation), emphysema (over inflation of the alveoli), and pulmonary edema (liquid accumulation in the airways). Adult livestock usually develops resistance and if re-infected may not show clinical signs but continue shedding larvae that contaminate their environment.[7]

Fecal examination

The Baermann technique is used to separate lungworm larvae from faecal material.[25] The principle is that the larvae migrate actively from faeces into the aqueous phase of the Baermann apparatus consisting of a glass/plastic funnel held in a retort stand. A rubber tube is attached to the funnel, its bottom constricted by a clip. A sieve (aperture 250mm) is placed on top of the funnel, which has been partially filled with water. A double layer of gauze is then placed on top of the sieve and a faecal sample placed on it before slowly filling the funnel with water to immerse the faecal sample. The sample stays overnight at room temperature during which time the larvae migrate out of the faeces through the sieve to the sediment at the neck of the funnel. The following morning the clip is released and water together with larvae in the neck of the funnel are collected into a Petri dish for microscopic examination. A simple adaptation of the above technique is to suspend faeces enclosed in gauze in a glass filled with water and allowed to stay overnight. The larvae leave the faeces, migrate through the gauze and settle at the bottom of the glass. This is collected in a Petri dish and examined under a low power microscope.[25]

Serological diagnosis

Enzyme Linked Immuno Sorbent Assay (ELISA) test can demonstrate antibodies from five weeks after the animals have been exposed and it may be use full in identifying infected animals when heavy burdens of worms do not generate and larvae in the feces. This time need to perform an ELISA depends on the availability of antigen-coated microstate-plates. If such plates can be provided; the result can be obtained with four hours after the serum has been prepared. If not, plates have to be coated with antigen for up to 16 hours.[26]

Post mortem findings

The morphological change in the lungs include wide spread areas of collapsed tissue of dark pink color, hemorrhagic bronchitis with much fluid filling all the air passed and enlargement of the regional lymph nodes. Histologically, the characteristic lesions are edema, eosinophilic infiltration, debris and larvae in the bronchioles and alveoli. The bronchial epithelium is hyperplasic and heavily infiltrated by inflammatory cells, particularly eosinophils.[27]

Differential diagnosis

On a clinical basis, bacterial pneumonia is considered as the first tentative diagnosis. Other probable tentative diagnoses are considered such as chronic hypersensitivity pneumonitis, chronic obstructive pulmonary disease, fungal pneumonia, immune mediated interstitial or vascular disease and unusual drug reactions as well as foreign body in the trachea.[28]

Treatmnet

The equine which has been infected by dictyocaulosis can be treated by addminstration of anthelmentic like, the

benzimidazoles (fenbendazole) and macrocyclic lactones (ivermectin). Fenbendazole can against all stages of *D. arnifield.*[14]

Control and Preventions

Routine deworming of horses and donkeys may help prevent cross infection when kept together. Pastures that housed donkeys may be infected with lungworm larvae. As a result, horses and donkeys should not be grazed together.[23] Reducing pasture contamination with infective larvae is a key preventative measure that can be achieved to a large extent with adequate management measures.

Rotational grazing with a change interval of 4 days and keeping the paddocks empty for atleast 40 days significantly reduces pasture contamination. This is due to the fact that larvae are susceptible to dryness and won't survive more than 4 or 5 weeks on pasture if they do not find an adequate host. Alternate grazing with sheep and/or horses may be considered, since *Dictyocaulus* species are quite host-specific. The longer the absence of the specific host, the higher will be the reduction of its specific lung worm. Keeping the pastures as dry as possible and keeping equines away from places excessively humid are additional key measures to reduce the exposure of livestock to infective larvae. Preventative measure often recommended just prior to their first grazing season, followed by additional treatments depending on the infestation level of the pastures and the residual effect of the administered anthelmintic.[7]

Economic Impact of the Disease

Although equines play a significant role in the economy of the country, the government lives look development programmes and those of aids agencies are aimed towards in meat, milk, and egg and wool production. Equines have been completely neglected or omitted from the pastoral livestock programmes. This is because of the contribution of equines power in the agricultural system and their role in the productions not yet well recognized and magnified.[29] Internal parasites are one of the greatest limiting factors to successful horse rising throughout the world. All horses at pasture become infected and suffer a wide range of harmful effects ranging from impaired development and performance to death despite the availability of large array of modern anthelmintic, parasite controls often fail to safeguard horse health. The main reason for these break downs are errors the choice of anthelmintic and in the time of treatment.[30]

CONCLUSION AND RECOMMENDATION

Lungworm is the one of the parasite which cause verminous pneumonia in domestic animals. The lungworm is known as dictyocaulus genus, dictyocaulus arnifield in equine animals. The clinical sign is not observes as well but, it occur rarely and lung worm can cause the secondary bacterial nd virasl complication disease like septicemic pneumonia and viral synchyal disease which cause alveolar cell degeneration and animals fail to breath. The dictyocaulus can cause the disease called dictyocaulosis, infestation of worm in lung of equines, then the equine animals fail to breath and circulatory system is disturbed. The major economic importance of this disease is death of animals, cost of drug and laboratory examination. The animals can be treated by administration of broad spectrum anthelmintic drugs like fenbendazole, ivermectin and levamisole givne orally. The lung worm can be prevented by deworming and pastural rotation of animals frompasture to pasture. The main objectivety of this review paper is to give some understanding on the lung worm knowledge, its lifecycle, epidemiology and control and prevention. Depending on the above conclusion the following recommendation should be forwarded:

The equine animals should be dewormed. The management practice in feeding pasture of animals should be practice in good manner. The animals should be rotate from one pasture area to other in order to decrease the infestation with larvae of lung worm. The community should have to have knowledge about the lung worm and its impact on equine. The government should have to install the anthelmintic drugs for veterinary sectors. The veterinary sector should have to control and prevent distribution of this disease

ACKNOWLEDGEMENT

First of all, I praise my Almighty God (God Father) through the name of his son Jesus Christ, for he loved, guided and helped me in all my life. I would like to express my appreciation for my family for they support me until all my academic education. Lastly, I have great appreciation and thanks for Jimma University College of agriculture and veterinary medicine for they support me in computer and internet access.

REFERENCE

1. Fred, O. and K. Pascal,(2006): Extension approaches to improving the welfare of working equines. Kenya Network for Dissemination of Agricultural Technologies (KENDAT), Nairobi, Kenya, 1-28.
2. Abayneh, T., Gebreab, F.B.Zekarias and G.Tadesse, (2002). The potential role of Donkeys inland tillage in central Ethiopia. Bulletin of Animal Health and production in Africa, 50: 172-178.
3. Abdisa T (2017) Review on Equine Epizootic Lymphangitis and its Impact in Ethiopia. J Vet Med Res, 4(5): 1087
4. Hansen, J. and Perry, B. (1996): The Epidemiology, Diagnosis and control of Helminthes parasites of ruminants, ILRAD, Nairobi, Kenya, 83.
5. Rose, F.R. and R.D.S. Hodgson, (2000).Manual of Equine practice. 2 Ed., USA, Saunders, 224.
6. Bowman, D.D. (2003): Georgis, Parasitology for Veterinarians, 8th Ed., USA, New York Saunders, 166-168.

7. Junquera, P. (2014): *Dictyocaulus* species, parasitic lungworms of Cattle, Sheep, Goats and other Livestock. Biology, prevention and control. *Dictyocaulusfilaria, Dictyocaulusviviparus, Dictyocaulusarnfieldi*. In: Merck Veterinary Manual, Merck and Co, Inc, Whitehouse Station, N.J., USA, Last Updated on Friday, January 03, 14: 28.
8. Feseha, G., A. Mohammed and Yilma, J. (1991): Vermicular endoparasitism in donkeys of DebreZeit and Menagesha, Ethiopia. Donkeys, mules and horses in tropical agricultural developement. Proc. Colloq. On donkeys, mules and horses. Eds. D. Fielding and R.A. Pearson, University of Edinburgh, Center for tropical veterinary medicine: UK, 156-160.
9. Fraser, C. M. (2000): The Merck Veterinary Manual. A hand book of Diagnosis Therapy and Disease prevention and control for the Veterinarians 8th Ed., Merck and Co; Inc, Rahaway, NIT, USA, 851-852.
10. Tihitna, S., Basaznew, B., Mersha, C. and Achenef, M. (2012): Ocuurrence of Lungworm Infection in Equines and their Associated Risk Factors. *Global Veterinaria*, 8: 35-38.
11. Andersen, S. and Fogh, J. (2010): Prevalence of lungworm (*D. arnfieldi)* in donkeys in Denmark and in horses in herds together with donkeys. *Nord Veterinary Medicine*, 33: 484-91.
12. Taylor, M. A., R.L. Coop and R.L.Hall, (2007). Veterinary parasitology.3rd edition. USA. Blackwell publishing, 874.
13. Susan, E. Aillo, (2005). The Merck Veterinary Manual ninth ed. Merck and Co., INC. White house station, USA.
14. Kahn, Cynthia (Ed). (2005). The Merck Veterinary Manual. Ninth Ed. Merck and co., INC., white house station, U.S.A. 215-256.
15. Smith, P.B. (2009): Large animal internal medicine, 4th Ed., USA, Mosby Elsevier of Lalitpur District, 232-321.
16. Armour, J., (1980).The Epidemiology of Helminth Disease in Farm Animals. Veterinary Parasitology, 6: 7-46.
17. Urquhart, G.M., Armur, J.I., Dunn, A.M. and Jenninngs, and F.W. (1999): Veterinary parasitology 2nd Ed., 39-40.
18. Blood, D.C., Radiostitis, O.M., Gray and Hincheeliff, K.W. (2000): Veterinary Medicine a text of book of the disease of Cattle, Sheep, Pigs and Horses. 8th Ed., Harcourt Publisher Ltd, London, 1246-1253.
19. Blood, D.C. and Radostits, O.M., (1999). Veterinary Medicine, A text book of the disease of Cattle, Sheep, Pigs, Goats and Horses, 7th Ed., Bailliere Tindal, London, UK, 1064- 1066.
20. Beelitz, P., Gobel, E. and Gothe, R. (1996): Endoparasites of donkeys and horses kept together in Upper Bavaria. Range of species and intensity of infestation. Tier arztliche Praxis, 24: 471-475.
21. Gibbs, H. C., (1986). Effects of parasites on animal and meat production. In: S.M. Gaafar, W.E. Howard and R.E. Marsh, eds. *Parasites, Pests and Predators.* Amsterdam: World Animal Science, Elsevier, 7-25.
22. Blood, D. C, O. M. Radostitis, and C. C. Gay, (1994).Vetrinary Medicine. A Text Book of The Disease of Cattle, Sheep, Pigs and Horses.8th ed. Harcourt Publisher Ltd, London, 1246-1253.
23. Johnson, M., MacKintosh, G., Labes, E., Taylor, J. and Wharton, A. (2003): "*Dictyocaulus* species. Cross infection between cattle and red deer". *New Zealand Veterinary Journal*, 51: 93-98.
24. Stuart, M.T, (2012). An overview of Lungworm Infection.In: Merck veterinary manual, Merck and Co; Inc, Whitehouse Station, N.J., USA, Last full review/revision on March.
25. Kaufmann, J., (1996). Parasitic Infections of Domestic Animals: A Diagnostic Manual.
26. Boon, J., Kloosterman, N. and Vanderlander, T., (1984). The incidence of *D. viviparous* infection of cattle, Netherland, Surveys of sera collected in the field. Veterinary quarterly, 6th Ed., 13-17.
27. Reinecke, R.K., (1989). *D.viviparous*, Veterinary Helminthology, professional publisher (Pty) Ltd, Pretoria, Pp: 178-180.
28. Burks, B.S. (1998): Parasitic pneumonitis in horses. *Compendium on ContinuingEducation for the Practicing Veterinarian*, 20: 378-383.
29. Belay, M., (2005). Preliminary study on helminithosis of equines in South and North Wollo zones. *Ethiopian Veterinary Journal*, 9: 39-50.
30. Power, D.G. (1992). The health of horse, Great Britain, 67-82.