International Journal of Veterinary Sciences and Animal Husbandry

ISSN: 2456-2912
VET 2022; 7(5): 26-34

www.veterinarypaper.com
Received: 09-04-2022
Accepted: 11-05-2022

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Review on epidemiology, pathogenesis, treatment, control and prevention of gastrointestinal parasite of poultry

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DOI: https://doi.org/10.22271/veterinary.2022.v7.i5a.439

Abstract
Gastrointestinal tract is one of the organ systems which infected by different pathogenic organisms specifically the predilection sites for different parasites (helminthes and protozoa) which absorb the nutrient and predispose to another infection. The aim of this review was to provide an overview of the published information regarding the epidemiology and the diagnostic approaches of chicken helminthes infection and coccidiosis. The disease is the main constraints of poultry production in different parts of world and gastrointestinal parasite is the most common infections of poultries directly and predisposes to others different infection. The common internal parasitic infections occur in poultry include cestodes, nematodes and coccidian that may cause considerable damage and great economic loss to the poultry industry due to malnutrition, decreased feed conversion ratio, weight loss, lowered egg production and death in young birds. Furthermore, parasites can make the flock less resistant to diseases and exacerbate existing disease conditions. Poultry coccidiosis generates economic losses due to mortality, reduced body weight plus the expenses related to preventive and therapeutic control**.** More research and investigation related to epidemiology and risk factors of gastrointestinal parasite should apply to increase the poultry production.

Keywords: Antihelminths, chicken, gastrointestinal, helminths, poultry

1. Introduction
Poultry includes all domestic birds kept for the purpose of human food production (meat and eggs) such as chickens, turkeys, ducks, geese, ostrich, guinea fowl and doves and pigeons. The common internal parasitic infections occur in poultry include cestodes, nematodes and coccidian that may cause considerable damage and great economic loss to the poultry industry due to malnutrition, decreased feed conversion ratio, weight loss, lowered egg production and death in young birds **(**Puttalakshmamma, 2008, Attree *et al*, 2021) [27, 6]. Furthermore, parasites can make the flock less resistant to diseases and exacerbate existing disease conditions (Gary and Richard, 2012, Katoch *et al*., 2012) [10, 12].

The study showed that parasitic infestations are usually conjoint. The concurrent infestations with two or more parasites, especially those with gastrointestinal predilection, heighten their role in early chick mortality and other productivity losses among the adults. This is particularly true of conjoint infestations with helminthes and coccidia whose combined effects on host metabolism could be devastating (Nnadi and George, 2010) [20]. Furthermore, parasites can make the flock less resistant to diseases and exacerbate existing disease conditions (Gary and Richard, 2012) [10] and (Katoch *et al*., 2012) [12].

Poultry coccidiosis generates economic losses due to mortality, reduced body weight plus the expenses related to preventive and therapeutic control. It is probably the most common disease in modern poultry production, where confinement rearing is practiced (Lorenzoni, 2010; Abadi *et al*., 2012 Blake *et al*., 2020) [1, 7]. Helminths infestations are known to cause interference with host metabolisiom resulting in poor feed utilization and reduced growth rate as well as size and age at maturity and these have been described as common characteristics of village chickens (Nnadi *et al*., 2007) [21].

The concurrent infestations with two or more parasites, especially those with gastrointestinal predilection, heighten their role in early chick mortality and other productivity losses among the adults (Ybañez *et al*., 2018) [36]. This is particularly true of conjoint infestations with helminthes and coccidia whose combined effects on host metabolism could be devastating (Nnadi and George, 2010) [20].

Study on the use of anthelmintics showed that to reduce losses in backyard poultry farming, the strategic deworming schedule has to be followed, so as to ensure better productivity and financial gains to the poultry owners (Katoch *et al*., 2012) [12].

2. Common Gastrointestinal Parasites

There are several species of helminthe parasites in domestic birds i.e. in poultry or chickens. Some of the helminthe parasites in domestic birds and their predilection site in the body can be listed as follows.

Oesophagus and crop: *Capillaria contorta, Gongylonen ingluvicola,* and *Ornithostrongylus quadriradiatus*

Proventriculus: *Dispharynx* spp., *Echinuria* spp. and *Tetromeres* spp.

Gizzard: *Amiiloslomum anseris***,** *Cheilospirura* spp., *Histiocephalus* spp. and *Streptocara* spp.

Small intestine: *Amoebotaeniu sphenoides***,** *Ascaridia columbav***,** *Ascaridia dissimilis***,** *Ascaridia galli, Copillaria caudinflato***,** *Capillaria obsignata***,** *Choanotaenia infindibulum***,** *Davainea proglottina***,** *Filicollis* spp., *Hartertia* spp., *Polymorphus* spp., *Raillietina echinobothridia***,** *Strongyloides avium* and *Trichosrrongylus tenuis.*

Large intestine: *Heterakis gallinarum***,** *Heterakis isolonche,* and *Trichostrongylus tenuis* (Urquhart *et al.,* 1996 and Sufian *et al*., 2022) [32, 33].

***Source*:** (Puttalakshmamma, 2008) [27].

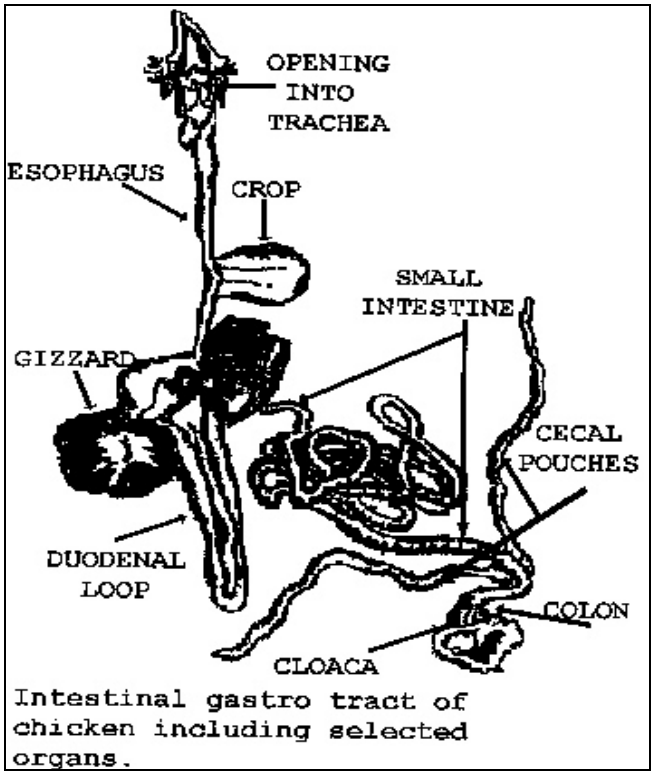
Fig 1: Intestinal Contents showing Nematode and Cestode parasite.

The presence of the cestode, *Davainea proglotina* is noteworthy because of its association with haemorrhagic enteritis which could complicate anaemia of ectoparasite origin. The presence of *Heterakis gallinae* also poses the danger of enhanced transmission of *Histomonas meleagridis* to both susceptible turkeys and other poultry through shedding of the eggs in the environment (Nnadi and George, 2010) [20].

Table 1: Common Helminths of Poultry

Parasite	Host	Intermediate host or life cycle	Organ infected	Pathogenicity
Nematodes				
Ascaridia galli	Chicken, turkey, duck, quail	Direct	Small intestine	Moderate
Capillaria caudinflata	Chicken, turkey, duck, game birds, pigeon	Earthworms	Small intestine	Moderate to severe
Capillaria contorta	Chicken, turkey, duck, game birds	None or earthworms	Mouth, esophagus, crop	Severe
Capillaria obsignata	Chicken, turkey, goose, pigeon, quail	Direct	Small intestine, ceca	Severe
Heterakis gallinarum	Chicken, turkey, duck, game birds	Direct	Ceca	Mild, but transmits agent of histomoniasis
Oxyspirura mansoni	Chicken, turkey, guinea fowl, quail	Cockroaches	Eye	Moderate
Strongyloides avium	Chicken, turkey, quail, goose	Direct	Ceca	Moderate
Syngamus trachea	Chicken, turkey, pheasant, quail	None or earthworm	Trachea	Severe
Tetrameres americana	Chicken, turkey, duck, game birds, pigeon	Grass-hoppers, cockroaches	Proventriculus	Moderate to severe
Trichostrongylus tenuis	Chicken, turkey, duck, pigeon	Direct	Ceca	Severe
Cestodes				
Choanotaenia infundibulum	Chicken	House flies	Upper intestine	Moderate
Davainea proglottina	Chicken	Slugs, snails	Duodenum	Severe
Raillietina cesticillus	Chicken	Beetles	Duodenum, jejunum	Mild
Raillietina echinobothrida	Chicken	Ants	Lower intestine	Severe, nodules
Raillietina tetragona	Chicken	Ants	Lower intestine	Severe

***Source*:** (Zirintunda *et al*, 2022) [37] (Kenneth, 2013) [13]



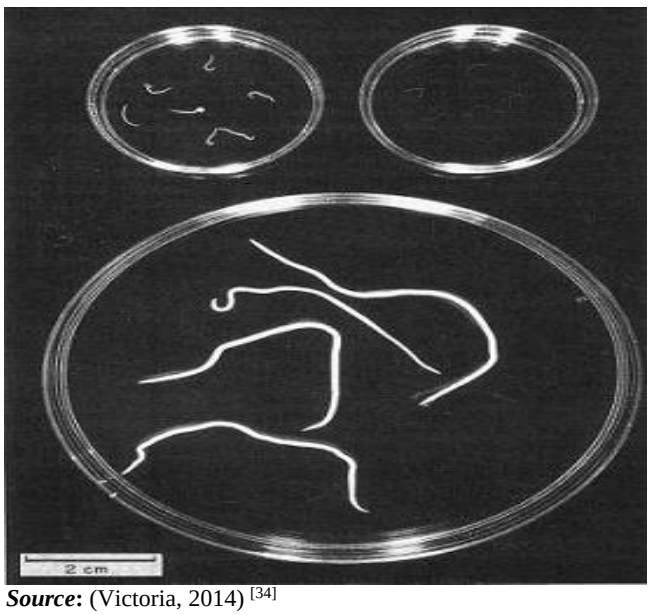
***Source*:** (Jacobs *et al.*, 2012)

Fig 2: Common predilection site of GIT poultry parasite

2.1 Nematode Parasites

Nematodes are the most common and most important helminth species in poultry. More than 50 species have been described in poultry (Abebe *et al*., 2016) [2]. Of these the majority causes pathological damage to the host. Nematodes belong to the phylum Nemathelminthes, class Nematoda. The nematodes of poultry are parasitic, unsegmented worms. The shape is usually cylindrical and elongated, but the cuticle may have circular annulations, be smooth, and have longitudinal striations or ornamentations in the form of cuticular plagues or spines. All worms have an alimentary tract. The sexes are separate. The life cycle may be direct or indirect including an intermediate host (Permin and Hansen, 2003) [22].

The nematodes or roundworms are the most common internal parasites of breeders. These include *Ascaridia galli* (intestine), *Heterakis gallinarum* (ceca) and various *Capillaria* species (crop→intestine) found through the digestive tract and *Syngamus* trachea or gape worm found in the lungs and trachea. As a group, the nematodes are characterized by being long spindle shaped worms varying in color from off-white to creamy yellow (Leeson and Summer, 2009) [15]. *Capillaria contorta* and the *Capillaria obsignata* are parasites of the crop and intestine respectively. *Ascaridia galli* occurs in the jejunum and *Heterakis gallinarum* in the cecum. *Tetrameres americana* is a 3 mm spherical nematode beneath the mucosa of the proventriculus. *Cheilospirura hamulosa* – a 2.5 cm nematode beneath the mucosa (koilin layer) of the ventriculus (Simon and Emeritus, 2005; Abrham and Abeba 2021) [29, 5].



***Source*:** (Victoria, 2014) [34]

Fig 3: Common gastrointestinal nematodes of poultry; Large dish: Ascarids, top left: Heterakis, top right: Capillaria

2.1.1 Life cycle

In the Nematoda, the sexes are separate and the males are generally smaller than the females which larva eggs or larvae. During development, a nematode moults at invervals shedding its cuticle. In the complete life cycle, there are four moults, the successive larval stages being designated L1, L2, L3, L4, and finally L5, which is the immature adult. One feature of the basic nematode life cycle is that immediate transfer of infection from one final host to another rarely occurs. Some development usually takes place either in the faecal pat or in a different species of animal, the intermediate host, before infection can take place. In the common form of direct life cycle, the free living larvae undergo two moults after hatching and infection is by ingestion of the free L3**.** There are some important exceptions however, infection sometimes being by larval penetration of the skin or by ingestion of the egg containing larva (Urquhart *et al.*, 1996) [32, 33]. In indirect life cycles, the first two moults usually take place in an intermediate host and infection of the final host is either by ingestion of the intermediate host or by inoculation of the L3, when the intermediate host feeds. After infection, two further moults take place to produce the L5 or immature adult parasite. Following copulation a further life cycle is initiated. In the case of gastrointestinal parasites, development may take place entirely in the gut lumen or with only limited movement into the mucosa (Urquhart *et al.*, 1996) [32, 33].

The life cycle of *A.galli* is simple and direct. Eggs in the droppings become infective in 10–12 days under optimal conditions. The infective eggs are ingested and hatch in the proventriculus, and the larvae live free in the lumen of the duodenum for the first 9 days. They then penetrate the mucosa, causing hemorrhages, return to the lumen by 17–18 days, and reach maturity at 28–30 days. Levels of infection are often underestimated, because early larval stages are barely visible and can remain for long periods within intestinal tissues, whereas adult stages in the lumen are generally fewer in number. Maturation of larval stages can be hampered by adult worm numbers, thereby increasing the time larval stages remain in intestinal tissues and continue to cause damage. The life cycle of *H.gallinarum* is similar to that of *A.galli*. Earthworms may ingest the eggs of the cecal worm and serve as a source of infection when ingested by poultry. Litter beetles may also serve as a mechanical vector. The life cycle of *Capillaria* may be direct (*C obsignata*), require an intermediate host such as earthworms (*C.caudinflata*), or be either direct or use earthworms (*C.contorta*). Larval development in the egg takes 8–15 days depending on temperature. Worms reach maturity in 20–26 days after ingestion by the final host (Kenneth, 2013) [13].

2.1.2 Epidemiology

In Acaridae, adult birds are symptomless carriers, and the reservoir of infection is on the ground, either as free eggs or in earthworm transport hosts. *H. gallinnarum* is widespread in most poultry flocks and is of little pathogenic significance in itself, but is of great importance in the epidemiology of *Histomonas.* In contrast *H. isolonche* in game birds occurs as a clinical entity (Urquhart *et al.*, 1996) [32, 33].

2.1.3 Pathogenesis, clinical signs and pathological lesions

Infection of the crop (*Capillaria contorta*) and the intestine (*Capillaria obsignata*) will result in severe emaciation and mortality in both immature and producing flocks. In floor-housed breeders and commercial layers reduction in egg production occurs. Ingluvitis (inflammation of the crop) is associated with *C. contorta*. Mucosal thickening and focal enteritis occurs with *C. obsignata.* Extensive A. *galli infection* may reduce egg production in floor housed breeders and commercial layers. Death may occur due to intestinal obstruction in birds which are immunosuppressed or are affected by an intercurrent debilitating condition (Simon and Emeritus, 2005 and Suhail *et al*., 2013) [29, 31].

2.1.4 Diagnosis of nematode parasites

In infections with adult Ascaridia worms, the eggs will be found in faeces, but since it is difficult to distinguish these from *Heterakis* eggs, confirmation must be made by post-mortem examination of a casualty when the large white worms will be found. In the prepatent period, larvae will be found in the intestinal contents and in scrapings of the mucosa. *H. gallinarum* infection is usually only diagnosed accidentally, by the finding of eggs in faeces or the presence of worm at necropsy. *H. isolonche* infection is diagnosed at necropsy by the finding of caecal nodules containing adult worms, and if necessary, confirmed microscopically by examination of the spicules (Urquhart *et al.*, 1996) [32, 33]. Parasitism can be diagnosed by examination of mucosal scrapings and fecal flotation, which reveal characteristic bi-operculated ova (Simon and Emeritus, 2005) [29]. When viewed under the microscope, nematodes have transverse grooves running across the body, but unlike the tapeworms they do not physically segment and so only the complete worms are found in the intestine or feces. Female worms produce eggs which are deposited in the feces (Leeson and Summer, 2009) [15].

2.1.5 Prevention, treatment and control for nematode parasites

Worms may become a problem in conditions of overcrowding and inadequate nutrition, particularly a deficiency of vitamin A which will make birds more susceptible. The best defense against worms is good management and good diet. When worms are present the most efficient way to control them is to break the life cycle in some way and so prevent constant reinfestation. Since worm eggs are either ingested by birds directly or via an intermediate host infestation can be significantly reduce by preventing contact between birds and droppings for example keeping them on wire. A rotational system of poultry runs will reduce the number of viable eggs in the soil; few will remain viable if the run is left vacant for 8 months (Lois, 1996) [16].

Once a year, or after a heavy worm infestation, the birds should be removed from the run and the ground covered with quicklime at a rate of 0.5 kg per square meter. After three weeks the whole run should be dug over to ensure that the worm eggs are killed. In the shed and run make sure that there are no damp, dark patches that will provide an environment for worm eggs to survive and become infective. Areas around water troughs present a particular risk. Wild birds may introduce worms into a previously clean pen and must be excluded if complete control of worms is to be achieved. It may be difficult to prevent birds having contact with the intermediate hosts of parasite but removal of breeding places for house flies will help. Spraying for flies, ants or termites that the insecticides used may be taken in by the birds and cause poisoning or residue problems in eggs and meat (Lois, 1996) [16].

Treatment for nematodes can be done with Piperazine, Fenbendazole in feed or levamisole or ivermectin in drinking water (Simon and Emeritus, 2005) [29]**.** When birds are reared

on a free-range system, and ascaridiosis is a problem, the young birds should, if possible, be segregated and reared on ground previously unused by poultry. Since the nematode may also be a problem in deep litter houses, feeding and watering systems which will limit the contamination of food and water by faeces should be used. In either case treatment with piperazine salts levamisole or a benzimidazole, such as flubendazole, can be administered either in the drinking water or the feed (Urquhart *et al.*, 1996**)** [32, 33]. The traditional worming compounds, used in the feed or water, have been piperazine and hygromycin. Hygromycin is usually used at around 750g/tone feed, while piperazine use is at 2-3kg/tone feed. Birds can also be treated individually if desired, with about 100mg piperazine. The traditional wormers are narcotics that paralyze, but do not kill the worm. The worms lose their attachment, and are passed out with the feces. At this stage, the eggs can still be infective, and so effective treatment must involve 2 or 3 dosages of the wormer, each some 7-10 days apart (Leeson and Summer, 2009) [15].

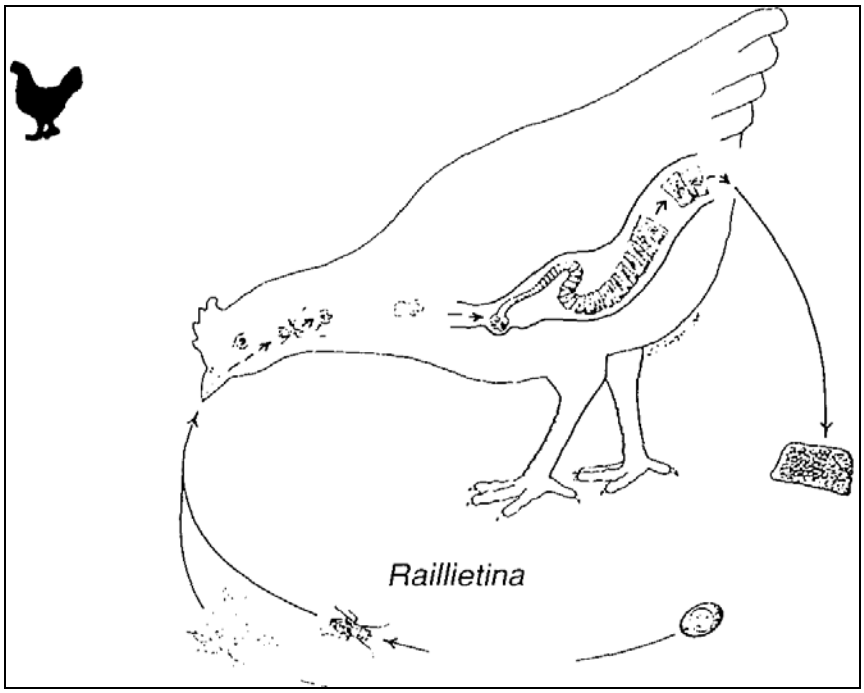
2.2 Cestodes (Tapeworms) Parasites

Tapeworms belong to the phylum Platyhelminthes, class Cestoda. The tapeworms of poultry are all endoparasitic, hermaphroditic worms with a flat, long segmented body without an alimentary tract or body cavity. Poultry tapeworms may reach a length of 30 - 50 cm. They have a scolex (the head) followed by a neck. The rest of the body is called the strobila consisting of a number of proglottids (segments) developing from the neck. Each segment contains a set of reproductive organs. The number of segments differs between species. The segments furthest away from the neck mature and are detached from the body. These gravid segments contain numerous eggs which are released to the environment with the faeces. Poultry reared under free range conditions are likely to be infected with cestodes (tapeworms). All tapeworms of poultry have indirect life cycles with intermediate hosts such as earthworms, beetles, flies, ants or grasshoppers. The intermediate hosts are essential to perpetuate the life cycle and infections are therefore rare in indoor systems. More than 1400 tapeworm species have been described in domesticated poultry and wild birds. The pathogenicity of the majority of these tapeworms is unknown. A great number are harmless or have a mild pathogenicity. Few species cause severe reactions in the host (Permin and Hansen, 2003) [22].

The most commonly diagnosed cestodes include: *Davainea proglottina* - a 4 mm cestode located in the duodenum. *Choanotaenia infundibulum* - a 25 cm cestode located in the distal duodenum and jejunum. *Raillietina tetragona* - a 25 cm cestode located in the distal jejunum. *Raillietina echinobothridia* - a 30 cm cestode of the jejunum resulting in nodular granulomas and catarrhal enteritis (Simon and Emeritus, 2005) [29].

2.2.1 Life cycle

The typical life cycle of cestodes is indirect with one intermediate host. With few exceptions, the adult tapewom is found in the small intestine of the final host, the segments and eggs reaching the exterior in the faeces. When the egg is ingested by the intermediate host, the gastric and intestinal secretions digest the embryophore and activate the onchosphere. Using its hooks, it tears through the mucosa to reach the blood or lymph stream or, in the case of invertebrates, the body cavity. Once in its predilection site the onchosphere loses its hooks and develops, depending on the species, into one of the larval stages, often known as metacestodes. When the metacestode is ingested by the final host the scolex attaches to the mucosa, the remainder of the structure is digested off, and a chain of proglottids begins to grow from the base of the scolex (Urquhart *et al.*, 1996) [32, 33]. Mature tapeworms can be up to 25cm in length and the continual shedding and regrowing of body segments, leads to a continual drain on the nutrient reserves of the bird (Leeson and summer, 2009) [15].



***Source*:** (William, 2001)

Fig 4: Life cycle of Tapeworms

2.2.2 Epidemiology

Cestodes require an intermediate host (eg, insects, crustaceans, earthworms, or snails). Floor layers, breeders, and broilers are infected with *Raillietina cesticillus* by ingestion of the intermediate host, small beetles that breed in contaminated litter. Cage layers in unscreened houses may become infected with *Choanotaenia infundibulum* by eating its intermediate host, the house fly. Litter beetles in proximity may also serve as intermediate hosts. More than 3,000 of the microscopic tapeworm *Davainea proglottina* have been recovered from a single bird. Several species of slugs and snails serve as intermediate hosts, and >1,500 infective parasites have been recovered from a single slug (Kenneth, 2013) [13].

Gut of domestic fowl is a safe haven for many cestode parasites, but the tapeworms belonging to the genus *Raillietina* are the most prevalent avian helminth parasites throughout the world. *R. echinobothrida* is the most important species in terms of prevalence and pathogenicity, particularly in the domestic fowl, *Gallus domesticus* (Permin and Hansen, 2003) [22]. The cestode inhabits the small intestine and causes stunted growth of young chicken, emaciation of the adult, and decreased egg production of the hen (McDougald, 2003) [18]. Suhail *et al*, (2013) [31] found that prevalence and magnitude of variation of cestode infection was high in indigenous comparison to exotic and reasoned that poor management in the indigenous layers could be the reason. Luka and Ndams, (2007) [17] encountered cestode parasites including *Raillietinatetragona*, *R. echinobothrida*, *R. cesticillus*, *Choanotaenia infundibulum* and. Out of which *Hymenolepis carioca* was the most prevalent and *R. cesticillus* the least.

2.2.3 Pathogenesis and Clinical Signs

Gross lesions caused by the cestode infection in indigenous and exotic layers included nodule formation on the intestinal mucosa, thickening, and ulceration, pale and rough mucosa of intestines. Histopathological lesions were the villous atrophy, catarrhal enteritis, granuloma formation in duodenum, desquamation of villi and submucosal glands, congestion, inflammatory reaction and vacuolation of epithelial cells (Anwar *et al.*, 1991) [3].

Cestodiasis results in emaciation in mature flocks, especially if severe infestation is exacerbated by malnutrition or immunosuppression (Simon and Emeritus, 2005) [29]**.** *Davainea proglottina*, the most pathogenic cestode of poultry. This is the most pathogenic of the poultry cestodes, the doubly armed scolex penetrating deeply between the duodenal villi. Heavy infections may cause haemorrhagic enteritis, and light infections retarded growth and weakness (Urquhart *et al.*, 1996) [32, 33]. The gross lesions of cestode infected guts included nodule formation on duodenal mucosa in *Raillietina echinobothrida* infection, pin point haemorrhages with *Raillietina tetragona* infection. Rough and pale mucosa of duodenum was observed in *Amoebotaenia cuneata* infection. No gross lesions were seen on the guts infected with *Choanotaenia infundibulum*, *Hymenolepis carioca* and *H. contaniana* (Anwar *et al*., 1991) [3].

In conditions of heavy infestation, *R. echinobothrida* is listed as one of the most pathogenic tapeworms, causing conspicuous intestinal nodules in chicken, with characteristic hyperplastic enteritis associated with the formation of granuloma (Kumar, 2007) [14]. The symptom is termed 'nodular tapeworm disease' in poultry. Intestinal nodules often result in degeneration and necrosis of intestinal villi and ultimately lead to death. Cestodes interfere with the metabolisms of certain compounds: they absorb glucose and galactose and stored them as glycogen as well as absorbed amino acids, polypeptides and protein (Cheng, 1973) [8]. Infection of chickens with 20 or 50 cysticercoids of *Raillietina tetragona* had no effect on the total protein content of the liver, but did cause a slight decrease in content of intestinal tissue (Suhail *et al.*, 2013) [31].

2.2.4 Diagnosis of Cestode Parasites

Numerous cestode species may occur in the intestinal tract and can be diagnosed at postmortem or by examination of feces (Simon and Emeritus, 2005) [29].

2.2.5 Prevention, treatment and control for cestode parasites

Control over infection simply relies upon breaking the reproductive cycle of the tapeworm, by eradicating the intermediary hosts. Slug and snail bait, usually containing metaldehyde, must therefore be applied around the perimeter of the house. Chemical treatment of infected birds is possible, but a number of these require 24h prior starvation of the bird, and so this naturally disrupts egg production in mature birds. Products such as praziquantel are effective against tapeworms, while most of the common chemical treatments used for roundworms are ineffective (Leeson and summer, 2009) [15]. Niclosamide in feed is recommended as treatment option (Simon and Emeritus, 2005) [29]. Control of Davainea and Raillietina depends on the treatment of infected birds with a suitable anthelmentic such as niclosamide and butynorate and the destruction of slugs and snails when possible (Urquhart *et al.*, 1996) [32, 33].

2.3 Coccidian Parasites

Protozoa within the subphylum Sporozoa are characterized by occurring intracellularly and having an apical complex at some stage of their development. The trophozoites have no cilia or flagella. Reproduction involves both asexual (schizogony) and sexual (gametogony) phases. Eimeriidae are mainly intracellular parasites of the intestinal epithelium. Important species of Eimeria in chickens may include *Eimeria tenella*, *E. necatrix*, *E. brunetti*, *E. maxima*, *E. mitis* and *E acervulina*. Identification can be made at microscopic level, either by examining the faeces for the presence of oocysts or by examination of scrapings or histological sections of affected tissues. In domestic poultry, coccidiosis can conveniently bc divided into caecal and intestinal coccidiosis (Urquhart *et al*, 1996) [32, 33]. Protozoan parasites of the genus *Eimeria* cause coccidiosis. *Eimeria* are obligated intracellular parasites with complex life cycles including sexual and asexual stages (Lorenzoni, 2010).

2.3.1 Life cycle

The life cycle of *Eimeria* starts with the ingestion of mature oocysts. Each infective oocyst is formed by four sporocysts and in turn each sporocyst contains two sporozoites. Bile salts and chymotrypsin stimulate the release of the sporozoites from the oocyst. Once freed, the sporozoites invade intestinal cells beginning the asexual development stage called schizogony. After a variable number of asexual cycles, gametes are formed and the sexual stage of development begins (gamogony). The sexual phase terminates with the production and release of oocysts into the intestinal lumen. Once in the environment, oocysts must sporulate to become infective. Sporulation process usually takes from 2 to 3 days depending on environmental conditions (Waldenstedt *et al*., 2001).

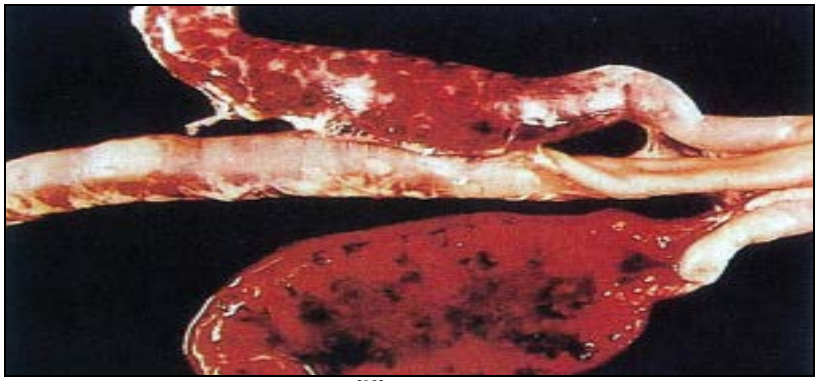
2.3.2 Epidemiology

Modern poultry production practices including rearing animals in high densities facilitate the distribution of this disease within poultry houses. Between poultry houses, the disease is transmitted by mechanical carriers like insects (black beetle) and wild birds. While *Eimeria* eggs (oocysts) can be mechanically transported by wild birds, these parasites are host specific and thus wild birds do not serve as a biological reservoir (Lorenzoni, 2010). The sporulated oocyst is the infective stage of the life-cycle. Infected, recovered chickens shed oocysts representing a problem in multi-age operations. Oocysts can be transmitted mechanically on the clothing and footwear of personnel, contaminated equipment, or in some cases, by wind spreading poultry-house dust and litter over short distances. Factors contributing to outbreaks of clinical coccidiosis include:- litter moisture content exceeding 30% due to ingress of rain or leaking waterers; immunosuppression (Marek's disease, IBD, mycotoxins); Suboptimal inclusion of anticoccidials or incomplete distribution (poor mixing) in feed; and Environmental and managemental stress such as overstocking, inoperative feeding systems, inadequate ventilation (Simon and Emeritus, 2005) [29].

2.3.3 Pathogenesis, clinical signs and pathological lesions

The histopathological examination of caecal scrapings of farm birds infected with Eimerian oocysts revealed necrosis of villous epithelium, massive haemorrhages and infiltration of inflammatory cells in the lamina propria and submucosa (Puttalakshmamma, 2008) [27]. Coccidiosis is generally acute in onset and is characterized by depression, ruffled plumage, and diarrhea. Birds infected with E. tenella show pallor of the comb and wattles and blood-stained cecal droppings (Simon and Emeritus, 2005) [29].

In poultry, *Eimeria* affect the intestine making it prone to other diseases (necrotic enteritis) and reducing the ability of this organ to absorb nutrients. Other pathological Lesions which may be attributed to the different Eimeria species of *E. acervulina* and *E. mivati* can be produces 1-2mm areas of hemorrhage interspersed with white foci visible through the serosa of the distal duodenum and proximal jejunum. *E. necatrix* causes *severe* distention of the mid-jejunum with hemorrhages in the mucosa and red-stained fluid in the lumen. *E. maximais* causes distention of the mid-jejunum with hemorrhages in the mucosa. *E. tenella* can causes hemorrhagic typhlitis (inflammation of the cecum). *E. brunette* pathologically causes for hemorrhages of the mucosa of the distal jejunum and colon. Fibrinonecrotic enteritis may occur in chronic cases (Simon and Emeritus, 2005) [29].



***Source*:** (Simon and Emeritus, 2005) [29].

Fig 3: Severe *Eimeria tenella* infection showing hemorrhagic ceca

2.3.4 Diagnosis of coccidian parasites

Diagnosis is best based on postmortem examination of a few affected birds. Although oocysts may be detected on faecal examination, it would be wrong to diagnose solely on such evidence for two reasons. First, the major pathogenic effect usually occurs prior to oocyst production, and secondly, depending on the species involved, the presence of large numhers of oocysts is not necessarily correlated with severe pathological changes in the gut. At necropsy the location and type of lesions present provide a good guide to the species which can be confirmed by examination of the oocysts in the faeces and the schizonts and oocysts present in scrapings of the gut (Urquhart *et al.*, 1996) [32, 33]. Gross lesions of *E. tenella*, *E. necatrix* and *E. brunetti* are diagnostic. Microscopic examination of intestinal and cecal scrapings reveals oocysts. To confirm a diagnosis in a commercial operation, the following specimens should be submitted to a laboratory: Intestine from a sacrificed, affected bird preserved in 5% potassium dichromate for culture and identification of Eimeria sp.; Intestine showing gross lesions in 10% formalin for histological examination; Representative feed samples for anticoccidial assay; and Litter samples for oocyst counts (Simon and Emeritus, 2005) [29].

2.3.5 Prevention, treatment and control for coccidian parasites

Sulphonaniide drugs are the most widely used and it is recommended that these are given for two periods of three days in the drinking water, with an interval of two days betwcen treatments. Sulphaquinoxaline. Sometimes polentiated with diaveridine, or sulphadimidine arc the drugs of choice. Where resistance has occurred to sulphonamides, mixtures of amprolium and ethopahate have given good results (Urquhart *et al.*, 1996) [32, 33]. Administration of amprolium solution, 0.024% of the active ingredient in drinking water for 3 - 5 days. Sulfonamides (sulfamethazines, 0.1% for 2 days, 0.05% for 4 days or commercial combinations of sulfa drugs) in drinking water. Administration of water dispersable vitamin A and K supplements may enhance recovery (Simon and Emeritus, 2005) [29].

Prevention of avian coccidiosis is based on a combination of good management and the use of anticoccidial compounds in the feed or water. Thus, litter should always be kept dry and special attention given to litter near water fonts or feeding troughs (Urquhart *et al.*, 1996) [32, 33]. Management procedures which limit saturation of litter include: Appropriate

installation and management of watering systems e.g. Nipple drinkers reduce spillage of water onto litter compared to bell and trough drinkers; Acceptable ventilation rate; Maintaining recommended stocking density; Providing adequate feeding space; Inclusion of anticoccidials in diets at recommended levels will prevent clinical infection; Chemical and ionophoric anticoccidials for broilers in shuttle programs; Synthetic coccidiostats for breeders and floor-reared commercial egg production flocks which allow the development of premunity. Anticoccidial vaccines are appropriate for replacement breeding stock and roasters (Simon and Emeritus, 2005) [29].

3. Conclusion

Gastrointestinal of poultry is commonly affected by different parasite which includes, helminths such as nematodes (round worm) cestode (tapeworm) and protozoal infection (coccidiosis). Those parasites are made a great impact on poultry health. Thus a control and prevention method should apply to increase poultry productions.

4. Declarations

Acknowledgement

We would like to thanks Journal of world poultry research (JWPR) to publish the paper.

Competing interest

The authors declare that they have no conflicts of interest.

Author's Contribution

Sufian A, Sadik Z, Teyib Abaf, generated the idea, paper write-up and completed the paper. Both Rashid H, Lenco A, and Suresh PN have taken part in the paper write-up, and edition of the manuscript. All authors read and approved the final version of the manuscript and conceived the study.

Funding

This work has been not funded by anybody.

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