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Ascariasis in poultry: A comprehensive review

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Abstract

Parasitic infections pose a significant challenge in poultry production. *Ascaridia galli* (Syn. *A. lineata*, *A. perspicillum*) is a nematode belonging to the phylum Nematoda that causes frequent infections along with significant morbidity and mortality in poultry birds, thereby leading to huge economic losses. *A. galli* has been detected in most of the species of poultry, including chickens, turkeys, geese, guinea fowl, and a wide variety of wild birds, with chicken being the main host. *A. galli* is commonly found in the small intestine of birds. Clinical symptoms include lack of appetite, ruffled feathers, weight loss, reduced egg production, anaemia, and diarrhoea, ultimately causing death. Upon post-mortem (PM) examination, partial or complete obstruction of the intestinal lumen alone or along with catarrhal to haemorrhagic enteritis is highly evident. Histopathologically, desquamation, hyperplasia of secretory cells and villous atrophy can occur. Treatment with suitable anthelmintics and good management practices are the key components for the treatment, prevention and control of this disease.

Keywords: *Ascaridia galli*, economic loss, parasitic infections, prevention, treatment

Introduction

Backyard poultry production has a long history, and they are being raised for various purposes like egg and meat production by people particularly in rural areas. Domestic chickens have a diverse diet that includes grains, fruits, and insects, which serves as a major source for potential infectious parasitic stages. These diverse dietary habits make them susceptible to various parasitic infections, particularly gastrointestinal parasites (Frantovo, 2000; Oniye *et al.*, 2001) [13, 25]. *Ascaridia galli* is a roundworm classified under the phylum Nematoda, class Chromadorea, order Ascaridida, and family Ascaridiidae (Tarbiat, 2018) [41]. It is characterised by a long, cylindrical body and exhibits sexual dimorphism. Males of *Ascaridia galli* are smaller typically measuring 50–76 mm in length and have a curved tail whereas females range from 72–116 mm and appears straight. They have three prominent lips and an oesophagus without a posterior bulb. Tail in male is equipped with ten pairs of papillae having alae. Notably, there is a round precloacal sucker present with a thick cuticular rim. The sub-equal spicule of the male varies in length from 1 to 2.4 mm. The eggs produced by *Ascaridia galli* are round having smooth shells, and non-segmented when laid first. Eggs typically measures 73–92 µm in length and 45–57 µm in width (Solusby, 1982) [37]. *Ascaridia* nematodes are commonly found in intestine of the birds (Garadaghi, 2011) [14]. It is the most prevalent nematode species causing severe harm especially in the domestic fowl (*Gallus gallus domesticus*). The parasite has a direct life cycle. Infectious eggs hatch in the duodenum of the suitable hosts, and the juvenile larvae initially inhabit the caudal part of the duodenal lumen after hatching and subsequently penetrating the mucosa, leading to haemorrhages. Juvenile worms remain in the duodenal lumen until they reach a mature stage after 28–30 days (Piergili *et al.*, 2005) [12]. The most significant predisposing factors for Ascariasis include young age, pre-infection with coccidiosis, and deficiency of vitamin A and protein in the diet (Gordon and Jordan, 1982) [37]. Ascariasis can be controlled by regular anthelmintic treatment. However, frequent administration of anthelmintics can lead to their resistance (Salam, 2015) [30]. Since, *Ascaridia galli* eggs require moisture for their survival, it is very important to keep the bedding dry and replacing it regularly with the fresh bedding in order to prevent the infection. All these preventive measures hold greater significance particularly to prevent the young birds from getting infected (Jacobs *et al.*, 2003) [17]. Previously, several researchers have explored the pathological changes associated with *A. galli* infection. These include the detection of nematode eggs in the faeces and the presence of large white worms in the poultry droppings of the bird. The primary treatment for *Ascaridia galli* includes anthelmintic drugs, mainly

piperazine.

Ascariasis is commonly ignored by the poultry farmers despite being highly prevalent and cause of significant economic loss in poultry. Therefore, the current review was prepared for the purpose of focusing on the aetiology, pathogenesis, clinical manifestations, diagnosis, treatment and preventive measures so that the infection can be detected in birds and their control measures can be taken well in time.

Historical overview

A. galli nematode was initially described in Germany and since then it has been reported in chickens across several countries worldwide, including Brazil, India, Zanzibar, the Philippines, China, Canada, the United Kingdom, Belgian (the Democratic Republic of the Congo). *A. galli* is now recognised as an infection of poultry birds worldwide with reports from numerous nations spanning temperate, subtropical, and tropical climates (Permin and Hansen, 1998) [27].

Host Range

A. galli can be found in wide range of poultry birds including chickens, guinea fowl, pigeons, geese, turkeys, and several wild birds but chickens act as a primary host for *A. galli* infection (Solusby, 1982; Permin and Hansen, 1998; Shohana *et al.*, 2023) [37, 27, 35].

Life Cycle and transmission

The life cycle of *Ascaridia galli* is a direct with the involvement of only one host. In the life cycle, sexually developed adult worms live in the small intestine of the host, where they lay eggs. Eggs are then excreted into the environment through host faeces (Shohana *et al.*, 2023) [35]. The oval-shaped eggs of *Ascaridia galli* are protected by three layers: the vitelline membrane, which is the inner permeable layer; a thick protective covering; and a thin albuminous layer (Perry and Wharton, 2011) [28] that prevents the egg from desiccation and maintains them in the environment for an extended period of time. Rather than hatching outside in the environment, the eggs of *Ascaridia galli* develop larvae within them. These larvae continue to moult until they reach the third larval stage (L3), which is the infective stage. The development of egg into the infectious L3 stage takes around 11 to 12 days (Tarbiat *et al.*, 2018) [40]. The primary route of transmission for *Ascaridia galli* is the consumption of feed or water contaminated with eggs. Additionally, mechanical transmission can also occur through earthworms which act as a transport host by ingesting the eggs containing the L3 larvae. Ingestion of earthworms by the final host can transmit the infection to them (Perry and Wharton, 2011) [28]. Inside the earthworms, larvae remain infectious for approximately 96 hours. Once eggs are ingested by the host, various factors such as pH, temperature, and carbon dioxide level initiate the process of egg hatching within 24 hours (Tarbiat *et al.*, 2015) [39]. Hatching occurs in the proventriculus region resulting in the release of L3 larvae into the cranial part of the jejunum (Ferdushy *et al.*, 2012) [11]. Subsequently, these larvae progress through various developmental stages until they reach sexual maturity and start laying eggs that are excreted in the faeces.

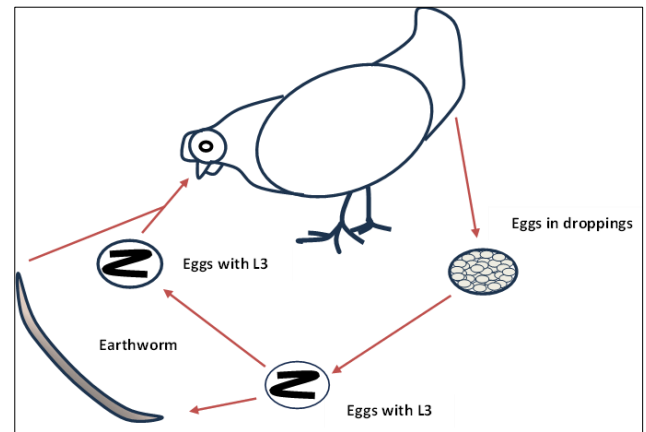


Fig 1: A schematic overview of life cycle of *Ascaridia galli*

Prevalence of Ascariasis

Parasitic infections pose a significant challenge for free-ranging poultry, especially chickens (*Gallus gallus domesticus*), whether raised in the backyard (Rabbi *et al.*, 2006) or free-ranging systems (Sherwin *et al.*, 2013) [33]. Kumar *et al.* (2015) [18] screened 58 poultry farms in the state of Uttar Pradesh and Uttarakhand in India and reported a 15.52 percent farm-wise prevalence of *A. galli*. Another study conducted from August 2017 to July 2019 by Ara *et al.* (2021) [3] in the Kashmir region of India revealed the prevalence of *A. galli* infection of 32.97 percent in the first year and 35.34 percent in the second year. A meta-analysis study conducted by Shifaw *et al.* (2021) [34] analysed 191 publications from the year 1942 to 2019 to assess the prevalence of helminth infections in poultry. The analysis revealed a pooled prevalence of 79.4 percent, with infection rates ranging from 4 to 100 percent. More than 30 helminth species were identified in poultry populations, with *Ascaridia galli* being the highly prevalent (35.9%). Notably, birds raised in free-range and backyard systems exhibited a higher pooled prevalence of 84.8 per cent and 82.6 per cent respectively which was higher than the prevalence of birds raised in cage production systems (63.6%).

Pathogenesis

Pathological sequelae are induced by both larval and adult worms (Daş *et al.*, 2012; Luna-Olivares *et al.*, 2015) [7, 22] which undergo development progressively through the mucosal and histotrophic phases (Luna-Olivares *et al.*, 2012) [21]. In the first week of infection, the majority of larvae reside in the cranial part of the jejunoileal region. However, as the infection progresses, the parasites move caudally. The majority of the larvae primarily develop in the lumen, whereas a few larvae remain stunted and associated with the mucosa. Eventually, each subpopulation relocates caudally in the gastrointestinal tract (Ferdushy *et al.*, 2013) [10]. The histotrophic larval stage may last for 3–54 days until the larvae progress to the final stage within the lumen. After three days of infection, the majority of larvae (63%) are located in the lumen in contact with the epithelium of Lieberkühn crypts whereas 37 percent are found in the tunica mucosa (Luna-Olivares *et al.*, 2015) [22].

After completion of the histotrophic stage, the larvae migrate to the lumen of the gut and develops into adult worms. Once the female reaches maturity, they lay large number of eggs which are continuously passed in the droppings.

The microscopic lesions of *A. galli* infection in poultry are primarily characterised by intestinal villi hypertrophy and inflammatory cell infiltration, particularly eosinophils, lymphocytes, and macrophages. The histotrophic phase of larvae is characterised by the necrosis of Lieberkühn crypts' (Malatji *et al.*, 2019) [23]. In addition, infection with a significant number of adult nematodes causes occlusion of the small intestinal lumen accompanied by intussusception of the intestine due to hypermotility and causing death (Daş and Gaulty, 2014) [8]. Adult worms can travel via the lumina propria of the large intestine and cloaca to the oviduct, where they get incorporated within the eggs (Solusby, 1982) [37]. Sometimes, *A. galli* secretes toxins along with its secretory and excretory (E/S) secretions, which effects the enzymatic activity of the gut and restrict the absorption of nutrients through the intestinal wall (Daş *et al.*, 2010) [9]. Young birds are more susceptible to infection than older birds due to the less number of goblet cells in the intestinal epithelium. Additionally, dietary deficiencies of vitamin A, B, and B₁₂, along with minerals and proteins can result in severe infection. Chickens over three months old are more resistant to infection due to the notable increase in goblet cells within the intestinal lining during this period (Solusby, 1982) [37].

Clinical Signs

Infection of *A. galli* can lead to severe illness or even death in birds (Balqis *et al.*, 2013) [4]. Illness resulting from Ascariasis often presents symptoms like anaemia, hypoglycaemia, weakness, anorexia, drooped wings, ruffled feathers, decreased egg production, diarrhoea, filthy vent, and weight loss (Permin and Hansen, 1998; Anwar and Rahman, 2002; Skallerup *et al.*, 2005; Schwarz *et al.*, 2011; Brar *et al.*, 2016) [27, 2, 36, 31, 5].

Pathology and Lesions

A. galli infection often presents significant macroscopic and microscopic lesions. Macroscopically, infection is characterised by a thicker intestinal wall along with severe haemorrhagic patches, and oedema. Microscopically, the infection shows an accumulation of lymphoid cell populations mixed with eosinophils (Levkut *et al.*, 2022) [20]. Haemorrhagic enteritis occurs when a large number of juvenile parasites invade and penetrate the mucosa of the duodenum or jejunum. Larvae embedded in the epithelium causes severe haemorrhages and extensive damage to the glandular epithelium and also lead to the proliferation of mucus-secreting goblet cells, which causes fusion of mucosal villi. Furthermore, mature worms can damage the epithelial lining by exerting pressure on the villi, ultimately leading to mucosal necrosis. Larvae measuring up to 7 mm in length can also be found within the mucosa (Solusby, 1982; Permin and Hansen, 1998; Hinrichsen *et al.*, 2016) [37, 27, 16]. In some cases, Ascariasis can lead to ulcerative proventriculitis (Brar *et al.*, 2016) [5]. Epithelial desquamation is observed frequently in duodenal villi which act as a barrier in preventing the penetration of L3 larvae into the duodenal mucosa. Additionally, there is hyperplasia in the intestinal villi to compensate for the loss of a number of cells. Fusion of the majority (90%) of the duodenal, complete jejunal, and ileal villi has also been reported (Balqis *et al.*, 2013; Zalazar *et*

al., 2006) [4, 43]. A high number of *A. galli* can lead to the degeneration and necrosis of epithelial cells in the small intestine, resulting in reduction of surface area of the villi. In areas where hyperplasia of villi occurs, the number of goblet cells, inflammatory cells, and mast cells increases significantly, which prevents the further penetration of *A. galli* larvae into the mucosa (Balqis *et al.*, 2013; Zalazar *et al.*, 2006) [4, 43]. Sometimes, live or calcified parasites can also be found in the albumin of eggs (Solusby, 1982) [37].

Diagnosis

Effective and timely diagnosis forms the basis of control of parasitic infections. Routine diagnosis of Ascariasis can be made by finding the eggs of the nematode in the faeces either through floatation technique, or the parasite can also be found in the intestine at the time of necropsy (Soulsby, 1982) [37]. Adult worms of *A. galli* can be readily detected during post-mortem examination in the small intestine whereas identification of the initial larval stages poses a greater challenge since it needs expertise. Furthermore, because of the tissue-related phase in the life cycle of *A. galli*, conventional methods like wet-sieving fails to detect the early infection. Therefore, the development and use of advanced diagnostic techniques including point-of-care testing having good diagnostic sensitivity and specificity is very important for accurate and timely detection of infection before they further spread into the flock. Oladosu *et al.* (2022) [24] developed a non-invasive ELISA technique to detect *A. galli* coproantigen in the droppings of poultry birds using polyclonal antibodies. The test has diagnostic sensitivity and specificity of 93 percent and 100 percent respectively. In addition to faecal samples, samples of serum and yolk can also be used for testing parasite specific IgY antibodies using serological techniques such as ELISA (Sharma *et al.*, 2018; Dao *et al.*, 2019) [32, 6]. Recently, Panich *et al.* (2023) [26] developed a loop-mediated isothermal amplification assay in association with a lateral flow dipstick (LAMP-LFD) test for identification of internal transcribed spacer (ITS-2) that aids in the visual detection of *A. galli* eggs in faecal samples. Furthermore, a test based on duplex digital droplet (dd) PCR was developed by Tarbiat *et al.*, (2021) [42] for detecting the relative abundance of DNA copies of internal ITS-2 in faecal samples. This test can detect as well as differentiate between the eggs of *A. galli* and *Heterakis gallinarum* and have higher detection rate as compared to the conventional floatation technique.

Treatment

Ascaridia galli infections can be effectively treated with piperazine drug available in different salts for administration in feed or drinking water. 94 to 100 per cent effectiveness has been reported by administration of piperazine adipate at the dose rate of 300–440 mg/kg in feed and 440 mg of piperazine citrate per liter of water for 24 hours. Similarly, piperazine carbodithioic acid in same dose rate is equally effective in treating *A. galli* infection. Phenothiazine can also be used with dose up to 2200 mg/kg but it has varied effects. Furthermore, *Ascaridia* infections can be successfully treated with alternative drugs like mebendazole (administered at 2 g in 28 kg of feed), haloxon (@ of 30 g per 50 g of feed), and tetramisole (provided as a 10% solution in drinking water). Hygromycin B, when dosed at 8 g/tonne of feed for 8 weeks, was also found effective in controlling the infection (Soulsby, 1982; Lalchandama, 2010; Al-Quraishi *et al.*, 2020) [37, 19, 1].

Prevention and Control

Maintaining strict hygiene in poultry yards and houses is very important for preventing the parasitic infections. Extra care should be provided to young birds, particularly when they are housed in deep-litter systems where *A. galli* infections are more prevalent especially in moist conditions. Additionally, maintenance of proper ventilation and moisture level of litter is also important. Before the new batches of chicks are introduced into the flock, it is advisable to pile up the litter in the litter house for several days for its optimal heating and sterilization. This is a proactive approach that is helpful in significant reduction of risk of parasitic infections in poultry (Soulsby, 1982) [37]. Furthermore, an appropriate stock of birds should be maintained to prevent overcrowding. To enhance the parasitic control, it is beneficial to separate birds into different age groups rather than adopting the "all in-all out" approach. This is very important as older birds may act as a carrier of various parasitic infections without displaying any overt clinical signs. To minimise the presence of infectious parasitic eggs, a key practice involves the regular changing of litter in pens, whether the birds are housed indoors or outdoors. This should be done at least once a week. Additionally, it's imperative to keep the floor dry to inhibit parasitic infestation. The most practical and cost-effective approach for preventing and managing *Ascaridia galli* infection is the implementation of regular deworming by following the proper dosage and administration protocols. This comprehensive strategy is essential in maintaining the health and productivity of poultry (Permin and Hansen, 1998) [27].

Conclusion

In the recent times, there has been a significant increase in poultry production owing to its low startup costs and ability for efficient conversion of nutrients into animal protein. However, despite its promising growth, the poultry industry faces a notable challenge in parasitic infections control which can lead to high economic losses particularly in low-income and developing countries. To address this challenge effectively, people associated with the poultry industry must possess knowledge about common parasitic infections and understanding of the mechanisms behind poultry parasitic infections that can pave the path for potential increases in production through the use of more advanced strategies for detection, prevention, and control of parasitic infections. The dynamics of helminth transmission have evolved, especially with the rapid expansion of commercial free-range production system. Consequently, it is imperative to gather epidemiological evidence that sheds light on the presence, spread, and underlying variables influencing these transmissions. This knowledge is essential for effective implementation the control measures. Therefore, facilitating access to accurate and timely diagnostic methods along with raising the awareness among poultry producers, becomes paramount and crucial for early interventions and the overall successful management of Ascariasis infection in the poultry.

Contribution of Authors

All the authors have made an equal contribution in the manuscript. They have reviewed the final manuscript and have given approval for publishing.

Conflict of Interest

The authors declare no conflict of interest.

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