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Perspective of gapeworm infection in birds

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Abstract
Syngamus trachea is a parasitic nematode of thin, red worm, known as a gapeworm which lives in the trachea, and sometimes the bronchi or lungs of certain birds. They can affect chickens but are common in turkeys, waterfowl (ducks and geese) and game birds (pheasants etc.). The resulting disease, known as "gape" or "the gapes", occurs when the worms clog and obstruct the airways. The worms are also known as "red worms" or "forked worms" due to their red color and the permanent procreative conjunction of males and females. Gapeworms are common in young, domesticated chickens and turkeys. Birds are infected with the parasite when they consume the eggs found in the faeces, or by consuming a transport host such as earthworms, snails or slugs. The drug ivermectin is often used to control gapeworm infection in birds.

Keywords: Syngamus trachea, gapeworm, pathogenesis, treatment

Introduction
The production and productivity is reduced due to various bacterial, viral, fungul, protozoan and helminthic diseases in birds [1-4]. Among the helminthic diseases game worm infection in chicken and turkeys plays an important role to reduce the economic losses to poultry farmers. The worms are also known as "red worms" or "forked worms" due to their red color and the permanent procreative conjunction of males and females. Earthworm acts as a transport host for the parasite [5, 6]. Gaping, coughing, panting, gasping and wheezing/hissing are the main clinical indication of the red worm infection. Chicken have a complex respiratory system that is prone to disease and infection. It is for this reason that breathing problems are one of the first consequences of environmental stress. While it is hard to miss a chicken that is coughing or having trouble breathing, it is much more difficult to discern whether a parasite or an infection causes the problem. Gapeworm and chronic respiratory disease (CRD) in chicken have very similar symptoms but require entirely different treatment. Importance of the disease we elaborately discussed about the morphological features, life cycle of the parasite along with pathogenesis and clinical signs caused by this parasite, epidemiology, diagnosis, treatment and control of the disease in the article.

Morphology
The parasites are bright red in colour when fresh hence known as red worm occur in trachea and sometimes in bronchi or lungs of certain birds [5]. The female (5-20 mm) are longer than the male (2-6 mm). Males and females are joined together in a state of permanent copulation forming, a Y shape (*forked worms*) [5, 6]. The mouth opening is wide, without leaf-crowns and the buccal capsule are cup-shaped, with six to ten small teeth at its base. The male bursa has short, stout rays and spicules are equal. Eggs (70-100x43-46µm) are thin shelled, oval, operculated at both ends and expelled in 16-celled stage.

Life cycle
The life cycle of the gapeworm is peculiar in that transmission from bird to bird may be successfully accomplished either directly (by ingesting embryonated eggs or infective larvae) or indirectly (by ingestion of earthworms containing free or encysted gapeworm larvae they had obtained by feeding on contaminated soil).

The eggs of the worms are usually coughed up and swallowed by the host and they passed out in the faeces. The infective larvae (L_3) develop inside the eggs, requiring, under optimal conditions of moisture and temperature, about three days, but under field conditions usually one to two weeks. The larva moult twice in the egg; it retains the cuticle of the previous stage as a sheath and has a short, pointed tail and relatively long oesophagus.

In the preparasitic phase, third stage infective larvae (L3) develop inside the eggs at which time they may hatch. When the female *S. trachea* lays her eggs in the trachea of an infected bird, the eggs are coughed up, swallowed and then passed out in the faeces [5]. Earthworms serve as transport (paratenic) hosts. Larvae have been shown to remain viable for more than three years encapsulated in earthworm muscles. Other invertebrates may also serve as paratenic hosts, including terrestrial snails (*Planorbarius corneus*, *Bithynia tentaculata* and others) and slugs [6]. Infection may occur by one of three ways, first by ingestion of the L_3 in the egg, secondly by ingestion of the hatched L_3, or thirdly by ingestion of a transport (earthworms, snails, slugs, flies, beetles and other arthropods, where larvae encysted) containing L_3. After penetrating the intestine of the final host the L_3 travel to the lungs, probably in blood since they are found in the alveoli 4-6 hours after infection. The two parasitic moults take place in the lungs within five days by which time the parasites are 1.0-2.00 mm long [5, 6]. Here, one ecdysis occurs on third day after infection, after which sexes can be differentiated. The final ecdysis occurs on the 4th or 5th day and the young worms migrate to the larger bronchi. Copulation occurs around days seven in the trachea or bronchi after which the female grows rapidly. The prepatent period is 18-20 days [5, 6].

Pathogenesis

The parasitic phase involves substantial migration in the definitive host to reach the predilection site. Young birds are most severely affected with migration of larvae and adults through the lungs causing a severe pneumonia [5, 6]. Lymphoid nodules form at the point of attachment of the worms in the bronchi and trachea. Adult worms also appear to feed on blood. Worms in the bronchi and trachea provoke a hemorrhagic tracheitis and bronchitis, forming large quantities of mucus, plugging the air passages and, in severe cases, causing asphyxiation [5, 6, 7].

In heavy infections migration through the lungs may cause ecchymoses, oedema and even lobar pneumonia and death. In the trachea the adult worms attach themselves to the mucosa and suck blood, with consequent catarrhal haemorrhagic tracheitis and secretion of much mucus [5, 6, 7]. The mucus may occlude the air passages causing difficulty in breathing. The males become deeply embedded with their anterior ends in the wall of trachea of turkeys, causing the development of nodules. The male worm, in the form of lesions, remains permanently attached to the tracheal wall throughout the duration of its life. The female worms apparently detach and reattach from time to time in order to obtain a more abundant supply of food. Lesions are usually found in the trachea of turkeys and pheasants but seldom if ever in the tracheas of young chickens and guinea fowl.

Clinical signs

Pneumonia during the prepatent phase may cause signs of dyspnea and depression, whereas the adult worms and excess mucous in the trachea lead to signs of asphyxia and suffocation with the bird gasping for air [5, 6, 7]. Otherwise we can say blockage of the bronchi and trachea with worms and mucus will cause infected birds to gasp for air. An infected bird often may give its head a convulsive shake and cough in an attempt to remove the obstruction from the trachea so that normal breathing may be resumed. The characteristic signs of ‘gapes’ are those of dyspnea and asphyxia, occurring in spasms on account of accumulation of mucus in the trachea. The bird shakes and tosses its head about and it may cough, or it extends the neck, opens the beak and performs gaping movements. They stretch out their necks, open their mouths and gasp for air producing a hissing noise as they do so. This “gaping” posture has given rise to the common term "gapeworm" to describe *Syngamus trachea* [5, 6]. Death results from asphyxia during such an attack or from progressive emaciation, anaemia and weakness caused by the parasite.

Afore mentioned clinical signs first appear approximately 1-2 weeks after infection. Birds infected with gapeworms show signs of weakness and emaciation, usually spending much of their time with eyes closed and head drawn back against the body. Severely affected birds, particularly young ones, will deteriorate rapidly; they stop drinking and become anorexic. At this stage, death is the usual outcome. Adult birds are usually less severely affected and may only show an occasional cough or even no obvious clinical signs.

Epidemiology

Eggs may survive for up to 9 months in soil and L_3 for years within the earthworm or other transport host. Larval abundance was significantly and positively associated with temperature and relative humidity [8]. Disease is seen most frequently in breeding and rearing establishments where outdoor pens, such as are used for breeding pheasants. Infection may be initiated by eggs, passed by wild birds such as rocks and blackbirds, these may also infect earthworms. Heavy burdens may occur in turkeys kept in straw yards. They may, however, be dangerous carriers of the disease, from which other birds acquire infection. Hen-chicks, goslings and artificially reared game birds, especially pheasants, suffer mostly from the parasites [7].

The effects of *S. trachea* are most severe in young birds especially in game chicks and turkey poults. Adult hens are not usually infected and guinea-fowl are little affected at any age. Turkeys are susceptible to infection at any age and some consider them to be the natural hosts of *S. trachea* hence adults often acting as carriers. Pheasants appear to be chiefly susceptible to infections ensuing in high mortality rates during outbreaks [5, 6]. The rapidly growing worms almost immediately obstruct the lumen of the trachea, causing suffocation. Turkey poults, baby chicks and pheasant chicks are most susceptible to infection. Turkey poults usually develop gapeworm signs earlier and begin to die sooner after infection than young chickens.

Earthworm transport hosts are important factors in the transmission of *S. trachea* when poultry and game birds are reared on soil. The longevity of infective third stage larvae (L3) in earthworms (up to 3 years) is particularly important in perpetuating the infection from year to year [5, 8]. Wild birds may serve as reservoirs of infection and have been implicated as the sources of infections in outbreaks on game-bird farms as well as poultry farms. Pheasants, ruffed grouse, partridges, wild turkeys, magpies, meadowlarks, American robins, grackles, jays, jackdaws, rooks, starlings and crows are the main reservoir hosts distributed different parts of the world. There is also evidence to suggest that strains of *S.*

trachea from wild bird reservoir hosts may be less effective in domestic birds; if they have an earthworm transport host rather than direct infections via ingestion of L_3, or eggs containing L_3.

Diagnosis

A diagnosis is usually made on the basis of the classical clinical signs of "gaping". Subclinical infections with few worms may be confirmed at necropsy by finding copulating worms in the trachea and also by finding the characteristic eggs in the faeces of infected birds. Examination of the tracheas of infected birds shows that the mucous membrane is extensively irritated and inflamed [6, 7]. Coughing is apparently the result of this irritation to the mucous lining. In comparison with CRD, gapeworm is far less likely to be accompanied by sneezing and nasal discharge. Additionally, CRD is generally characterized by a congested, phlegmy sound in the chest. Unlike the rattling or gurgling the gapeworm will produce, stemming from the trachea or throat. Because they affect the upper respiratory tract, gapeworm infestations have similar symptoms to chicken respiratory disease; the two are frequently confused.

Indication of gapeworm in chickens includes:

- Gaping (stretching the neck and shaking the head, in an attempt to dislodge the worms),
- Coughing,
- Open-mouthed breathing or panting,
- Gasping for breath
- Wheezing or hissing

Molecular approach may also be the diagnostic tools applied as advance diagnosis. The implementation of a restriction enzyme analyse (RFLP) can provide a fast tool for species diagnostics of *S. trachea* [9]. Experimental infection can also be employed to diagnose the disease. Larvae of *S. trachea* injected into the veins, skin, peritoneum, musculature, and trachea of turkeys successfully infected the host [10].

Treatment

Several compounds have been shown effective against *S. trachea* under experimental conditions as under

1. Flubendazole is the only licensed anthelmintic for use in poultry and game birds. Continuous medication of pen-reared birds has been recommended, but is not economical and increases the possibility of drug resistance.
2. Methyl 5-benzoyl-2-benzimidazole was 100% efficacious when fed prophylactically to turkey poults.
3. 5-Isopropoxycarbonylamino-2-(4-thizolyl)-benzimidazole was found to be more efficacious than thiabendazole or disophenol.
4. Thiabendazole-@0.3-1.5g/kg orally has been used successfully to treat chickens and turkeys [5].
5. Continuous feeding of 0.05% thiabendazole in food for 7 days is effective in pheasants and partridge and @6 mg/kg of food for 48 days may prevent infection in pheasants.
6. Mebendazole and Fenbendazole are effective @ 0.01% in the feed for 7-14 days [5].
7. The level of control with three treatments of cambendazole on days 3-4, 6-7, and 16-17 post-infection was 94.9% in chickens and 99.1% in turkeys.
8. Levamisole at the dose rate of 0.04% in feed for 2 days or 2 g/gal drinking water for 1 day each month has proven effective in game birds.
9. Fenbendazole @20mg/kg for 3-4 days is also effective.
10. Even ivermectin injections may be effective in treating resistant strains but ivermectin will not kill adult gapeworm.

How to treat gapeworm

Healthy adult chickens can cope with a certain level of endoparasites before their health is affected. However, if birds are showing perceptible symptoms of gapeworm infection it is necessary to treat them with a dewormer. The treatment will prevent permanent damage to their respiratory system and eventual death. To treat gapeworm infection, the following steps could be followed

1. Treat the flock

If one bird is showing the signs of gapeworm infection, your whole flock will be carrying the parasite and should be treated with appropriate anthelmintics.

2. Treat the flock again

Gapeworms spread through the ingestion of eggs that are coughed up by infected birds or present in their faeces. Therefore, it is essential to treat birds for gapeworm twice at one to two week interval depending on the anthelmintic to be used. Interestingly, the first treatment will kill adult worms infecting the birds, while the second treatment will eradicate any worms which have hatched from eggs or been ingested since the first treatment.

3. Reduce the risk of reinfection

After the second deworming, it is imperative to reduce the menace of reinfection by cleaning and, where possible, disinfecting the cage and/ or run within 24 hours of treatment. This will destroy any remaining eggs and reduce the risk of infection.

4. Implement a regular deworming program

We suggest you deworm your chicken twice a year to prevent parasitic loads that may badly affect bird health. Regular deworming will also decrease the risk baffling CRD with gapeworm, ensuring that when your chickens fall ill, they will obtain the accurate treatment.

Prevention

a) Young birds should not be reared with adults, especially turkeys, and to prevent infection becoming established runs or yards should be kept dry and contact with wild birds prevented [6].
b) Severe out breaks of gapeworm infection are less likely to occur if the birds are not kept for long periods on the same ground.
c) Moist localities where earthworms, slugs and snails occur should be avoided if possible.
d) Infected pens or yards may be treated with D-D (a mixture of 1, 3-dichloropropylene and 1, 2-dichloropropane) or Dow fume W-40 (42% ethylene bromide in petroleum extendor) at the rate of 1.5 L/16 m^2.
e) In the artificial rearing of pheasants, gapeworms are a serious hazard. Confinement rearing of young birds has reduced the problem in chickens compared to a few years ago. However, this parasite continues to present an occasional problem with turkeys raised on range.
f) Treatment with carbaryl, tetrachlorvinphos of the soil or litter to kill intermediate hosts may be beneficial.

Extreme care should be taken to ensure that feed and water are not contaminated. Treatment of range soil to kill ova is only partially successful.

g) Changing litter can reduce infections, but treating floors with oil is not very effective.

Conclusion

Gapeworm causes of respiratory distress in birds feeding earthworm intermediate host. Diagnosis is made by the presence of blood-red nematodes in the trachea and large bipolar nematode ova in faeces. Benzimidazole anthelmintics used at standard avian doses is effective but preventive measures help in reducing the spread of the disease.

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