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Avian trichomonosis with special reference to pigeon

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Abstract
Canker, the avian trichomonosis is a protozoan disease caused by *Trichomonas gallinae*, occurs in pigeon especially affecting the upper digestive tract, mainly the crop and esophagus. The significant clinical manifestations of the disease are caseous accumulation in the throat and loss of body weight with high morbidity and low to high mortality depending upon the variable pathogenicity of the organisms. Transmission is via oral secretions in feed and water, and crop milk. The epidemiology of the disease depends on transmission way of etiological agents, immune status of the birds, age of the birds, managements of the pigeons. Hence, the infections are more in young as compare to adults and more prevalent among domestic pigeons than wild doves. The history, post-mortem findings, microscopic examination of mucous or fluid from throat, molecular technique and culture technique can be applied for diagnosis of the disease. The effective treatment and proper managemental practice helps in prevention of the disease.

Keywords: *Trichomonas gallinae*, pigeon, diagnosis

Introduction
Avian trichomonosis is caused by *Trichomonas gallinae*, a flagellate protozoan which has four anterior flagella and an undulating membrane on one side without posterior flagellum. *T. gallinae* is a cosmopolitan parasite of pigeons and doves (Soulsby., 1982) [1] Other birds such as domestic and wild turkeys, chickens, raptors (hawks, golden eagle, etc.) may also become infected [2]. The disease in pigeons is commonly called canker while in birds of prey is called frounce. The parasite inhabits the upper digestive tract, mainly the crop and esophagus, but it may also infect the liver, lungs, air sacs, internal lining of the body, pancreas and bones and sinuses of the skull [4]. Transmission of the disease occurs via oral secretions in feed and water, and crop milk [3]. The caseous accumulation in the throat and loss of body weight is the common characteristic of the disease. The morbidity of the disease may be high with low to high mortality due to variable pathogenicity of the organisms [8]. The young birds are mostly susceptible to trichomonosis.

Etiology
Both *T. gallinae* and a newly recognized species, *T stableri*, are the causative organisms of trichomonosis. These flagellated protozoa live in the sinuses, mouth, throat, esophagus, liver, and other organs. The most virulent strains are Jones' Barn (JB) strain followed by TG strain, and YG strain.

Transmission
The transmission of the causative organisms of trichomonosis from one bird to another occurs in one of three following ways:

Infected parent feeding young
Pigeons and doves transmit the infection to their offspring in contaminated pigeon milk. So transmission occurs when infected older pigeons/ doves (carriers) feed "pigeon milk" to newly hatched squabs [4]. Adult birds, which do not show signs of disease, may carry the infection for a year or more and are a constant source of infection for their young [9].

Contaminated drinking water

Contaminated water and food by regurgitated saliva or droppings or crop secretions from an individual infected with the parasites is probably the most important source of infection for chickens, turkeys, and songbirds [8]. Because the trichomonads do not survive for long outside the bird, transmission must occur rapidly. Wild pigeons and other birds may be an important source of introducing the infection to domestic birds.

Infected bird is a prey meal for another bird:

An infection may be transmitted and established in a raptor that has fed on an infected prey bird. This type of transmission is more common to raptors [3]. Raptors acquire the disease when consuming infected birds as prey. Although under most conditions the parasite is not viable in the environment for long, bird feeders and baths are thought to be potential sites of transmission.

Epidemiology

The trichomoniasis is more prevalent among domestic pigeons and wild doves than among domestic fowl, although severe outbreaks have been reported in chickens and turkeys [3]. Younger birds are usually affected more seriously than older, mature chickens. So the disease is mostly occurred in young birds with high morbidity and varies mortality due to the variable pathogenicity. Some tricho monad strains cause high mortality in pigeons and doves. Hawks may become diseased after eating infected birds and commonly show liver lesions, with or without throat involvement. The severity of the disease varies depending on the strain of *T. gallinae*, immune status of the bird, and age. There are numerous strains of *T. gallinae*, ranging from some which cause no clinical signs to highly pathogenic strains. Columbi forms are the most common host of *T. gallinae*, with most wild and almost all domestic pigeons and doves being infected [9].

Pathogenesis

The organisms invade the oropharyngeal mucosa causes inflammation and ulceration [6, 11]. In pigeon squabs, the early lesions appear as small white to yellowish areas in the mouth cavity, especially the soft palate in 3-14 days after infection. A mass of necrotic materials are found in the mouth and esophagus later this may extend into the skull and sometimes through the surrounding tissues of the neck to involve the skin [3]. The yellowish lesions in esophagus and crop are become rounded, raised, with a central conical caseous spur, often referred to as "yellow buttons." The lesions increase in size and number and extend to the esophagus, crop and proventriculus [6]. The lesions may develop into large, firm necrotic masses that may block the lumen. The lesions of the digestive tract do not extend to beyond the proventiculus. The crop may be covered by a yellowish, diphtheritic membrane and which gradually extend to the proventriculus. Simultaneously, the organisms invade the pharyngeal glands, penetrate the underlying tissues and reach the liver and form abscess which may be the cause of death. In liver, the lesions are found with vary from a few small, yellow areas of necrosis to almost complete replacement of liver tissue by caseous necrotic debris.

Adhesions and involvement of other internal organs appear to be contact extensions of the liver lesions. The visceral form of the disease involves the liver and gastrointestinal tract, causing organ dysfunction. More velogenic strains can cause caseated abscessation of the oropharynx [4]. Eventually these space occupying lesions obstruct the esophagus and trachea resulting in emaciation and asphyxiation.

Clinical signs

One of the main characteristics of the disease is the development of a canker, or caseous plaque in the oral cavity (mouth), esophagus (throat), and crop [11]. Trichomonosis causes severe damage to the tissues of the mouth, throat, crop and esophagus and affected birds may drool saliva, regurgitate food, have difficulty in swallowing food and water, demonstrate laboured breathing and/or have a swollen neck or throat [4]. In addition to showing signs of general illness (i.e., lethargy, poor flight ability and fluffed up feathers), affected finches are frequently observed to have matted wet plumage around the face and beak, presumably due to regurgitation. Affected birds are also commonly very thin as the damage to the tissues of the throat and esophagus makes eating and drinking painful and difficult. Erosion of the papillae on the palatal flaps is a good sign of infection [10]. Infected pigeon squabs show an initial depression with ruffled feathers and later they become weak and emaciated. There is accumulation of a greenish fluid, or cheesy materials, in the mouth and crop and this may emanate from the beak [11].

Diagnosis

1. History - of pigeons and doves drinking or eating from the same sources as chickens.
2. Post-mortem findings: lesions of trichomonosis are characteristic but not pathognomonic; those of pox, fungal disease, *Salmonella*, and other infections can be similar [5]. Moreover, trichomonosis has sometimes been confused with histomoniasis because of the similarity in liver lesions.
3. Microscopic examination: the confirmatory diagnosis should be done by microscopic examination of a smear of mucous or fluid from the throat or secretions to demonstrate the presence of trichomonads.
4. Molecular technique: based on the internal transcribed spacers of nuclear ribosomal DNA of *T. gallinae* the PCR can be performed to diagnose the disease [7].
5. Culture technique: Trichomonads can be cultured easily in various artificial media such as Diamond's media, 0.2% Loeffler's dried blood serum in Ringer's solution, or a 2% solution of pigeon serum in isotonic salt solution. Good growth is obtained at 98.6°F (37°C). Antibiotics may be used to reduce bacterial contamination.

Treatment

Successful treatments include 2-Amino-5-nitrothiazole (30mg/kg daily for 7 days for homing pigeon and 45 mg/kg body wt for other birds), carnidazole (10 mg/kg body wt), metronidazole (60 mg/kg body wt), and dimetridazole (50 mg/kg body wt, PO; or in the drinking water at 0.05% for 5–6 days).

Management and prevention

The feeders and baths play a significant role in dissemination of the disease so the following precautions are recommended to avoid spread of the disease and to keep birds healthy.

1. Don't allow pigeon's access to the chicken feeders and waterers.
2. Confiscate bird feeders and baths for at least two weeks to disperse birds and reduce the likelihood of transmission during the outbreak of unknown trichomonosis.

3. Clean your bird feeders and baths regularly with a weak solution of domestic bleach (5% sodium hypochlorite).
4. Feeders should be rinsed well and dried before re-use.
5. Only use bird feeders that prevent the seed from getting wet.
6. Bird seed that is exposed to rain and becomes wet is a more suitable environment for the potential survival of the parasite.
7. Do not use table feeders. Sick birds sitting directly on bird seed are more likely to contaminate it with *T. gallinae*.

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