AVIAN TUBERCULOSIS

K. B. M. Saiful Islam, Ph.D.

OTHER NAME

Avian Tuberculosis — Avian Mycobacteriosis — Mycobacterium avium complex (MAC) disease — Mycobacterium avium-intercellulare complex (MAIC) disease.

Tuberculosis occurs worldwide in birds as a contagious, chronic, bacterial disease caused by members of the Mycobacterium avium complex which currently consists of four subtypes:

M. avium subsp avium is fully virulent for birds and small mammals.

M. avium subsp hominissuis is found in the environment but some are virulent in birds.

M. avium subsp paratuberculosis affects ruminants and other animals.

M. avium subsp silvaticum is rarely found and can be virulent in birds.

Avian tuberculosis is a slowly developing disease that leads to anorexia, emaciation, dyspnoea and death after a few months. After being established it can persist in the environment and bird populations for years.

ETIOLOGY

M. avium, the causative agent of avian tuberculosis, considered as “atypical mycobacteria”, comprises-

aerobic, nonspore-forming and nonmotile rod shaped bacteria that vary in length from 1–3 µm and cords are not formed, unlike M. tuberculosis.

They are weakly Gram-positive and stained specifically by acid fast (Ziehl-Neelsen) staining method, due to high levels of lipids in mycobacterial cell wall.

M. avium is highly resistant to environmental challenges and can survive in soil for up to 4 years.

TRANSMISSION

The most important source of the organism is the infected host.Also infectious are the items contaminated with the droppings of infected birds, such as litter, pastures and pens, equipment and implements, Dust.

And also the hands, feet and clothing of workers.

Eggs are of a minor importance in the epidemiology of the disease, and chicks hatched from eggs laid by tuberculous hens are not infected.

Lack of hygiene influences the appearance of the disease due to the organism being highly resistant in the environment. It is relatively resistant to many antimicrobials and disinfectants, but sensitive to ionic detergents.

INCUBATION PERIOD

The disease has a long incubation period and a protracted course and if appreciable, the symptoms can prolong for weeks or months. Because of the chances to become established through a longer exposure, the disease is less prevalent in young fowls and lesions are less severe in them when compared to adult birds. Usually the losses are experienced more in older stocks of age group 18–20 months.

CLINICAL SIGN

Subcutaneous masses in distal part of neck and sternal region.

There is a generally progressive loss of condition and lethargy, although appetite remains good.

Affected birds will show marked emaciation and atrophy of the sternal muscles with a prominent keel.

The face and comb are pale and sometimes jaundiced.

There is persistent diarrhoea with soiling of the tail feathers.Sometimes birds will show a hopping motion, usually unilateral.

Sudden death can also occur due to rupture of the liver or spleen and internal haemorrhage.

The classical presentation is characterised by chronic and progressive wasting and weakness.

Avian tuberculosis in birds is primarily an intestinal and hepatic disease with dissemination to other organs including the lungs, air sacs, spleen, bone marrow, and skin.

The disease process can be divided into three phases: latency, lesion development, and period of cachexia.

During cachexia, massive tubercles with large numbers of bacilli develop.

In the classic form of infection the tubercles or granulomas develop in multiple organs.

Asecond form is manifested with lesions in the intestinal tract;

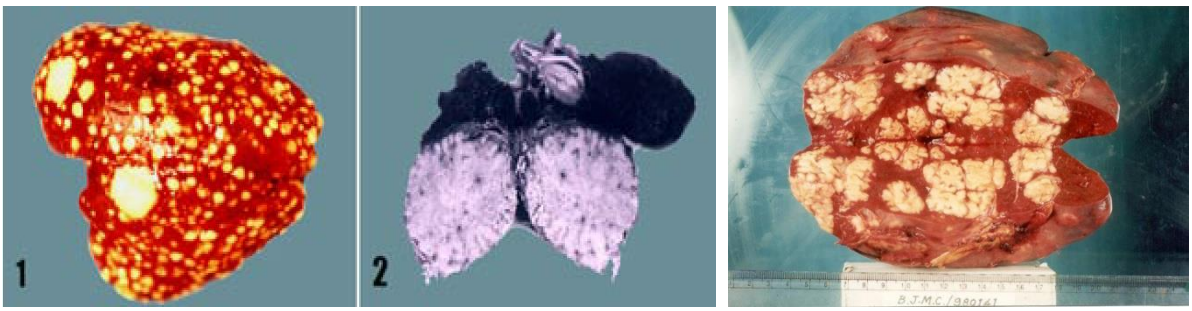
A third type of infection often experienced as a nontuberculous one, mainly seen in finches, canaries, and psittacines.

Some birds show respiratory signs and sudden death may occur, dyspnoea is less common, and granulomatous ocular lesions and skin lesions have been reported.

In advanced cases weight loss, increased thirst, respiratory distress, fatigue, and decreased egg production.

If a jerky hopping gait is observed due to unilateral lameness then it should be assumed that there could be the presence of tubercular lesions in bone marrow of the leg bones or joints.

The principal lesions is Greyish-white to greyish-yellow nodules. Before the intestinal tract is opened, the ulcerated areas appear as tumour-like masses attached to the gut wall.Typical caseous lesions, without calcification, are always found in the liver and spleen, with considerable enlargement of the organs.



DIAGNOSIS

The clinical signs are strongly suggestive.

Gross lesions include: granulomatous lesions in the intestine, liver, spleen and bone marrow present as irregular grey-white nodules, firm and caseous, with a pale yellow centre.

The lungs are more commonly affected in peafowl.

Ziehl-Neelson staining of smears or sections and demonstration of acid-fast bacilli also supports the diagnosis.

Immunological tests are useful in recognising infected live birds, and include:

Tuberculin test: In chickens, avian tuberculin is injected into the wattle, and the test read 48 hours later. The two wattles are palpated simultaneously and a positive reaction is recognised by a hot, soft and oedematous swelling in the injected wattle. Sensitivity is 80% in chickens. In other species it is not as reliable and the agglutination test is preferred.

Agglutination test: a drop of antigen is mixed with a drop of blood from the bird and a positive reaction corresponds to agglutination within 1 minute. It has a poor specificity.

ELISA: antibodies against M. avium can be detected in serum. A positive result can indicate previous exposure to the organism, latent infection or active infection.

TREATMENT

therapeutic regimen should include rifabutin and ethambutol, and later azithromycin or clarithromycin can be administered concurrently. Birds that respond poorly to therapy should have either a fluoroquinolone or an aminoglycoside added to the regimen. An alternative or additional drug that may prove useful, especially in birds with a marked inflammatory response, would be clofazamine. All these drugs may be curative at a total daily dose of 85 mg/kg for clarithromycin, 43 mg/kg for azithromycin, 56 mg/kg for rifabutin, 56 to 85 mg/kg for ethambutol, and 6 to 12 mg/kg for clofazamine.

CONTROL

Antimicrobials are generally ineffective against the organism, and drug therapy is usually not economically feasible.

In commercial poultry flocks, relatively rapid turnover of populations, together with improved general sanitation, has largely eliminated the incidence of avian tuberculosis.

In an outbreak, to eradicate and maintain freedom from infection:

All infected material must be removed by destroying infected birds and thoroughly cleaning the housing and equipment.

Stock should only be introduced if it is free from infection: negative serological tests or absence of clinical signs.

Infection should be prevented from entering the stock by preventing contact with wild birds and maintaining high biosecurity levels.

Freedom from infection should be monitored with serological testing or post-mortem investigation.

The weight of infection can be reduced by only keeping stock in their first laying season and monitoring for infection and disposing of any positive cases.

Avian tuberculosis in zoos is difficult to eradicate. New additions to the aviary should be quarantined for 2-3 months. The movement of ratites through sales and the long life of these animals have made tuberculosis a major concern for ratite producers. Isolation of ratites purchased at sales is essential to prevent the introduction of tuberculosis into established flocks.

REFERENCES

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