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RESEARCH ARTICLE

AVIAN MYCOBACTERIOSIS

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ABSTRACT

Avian mycobacteriosis in poultry is a contagious disease and may also be called avian tuberculosis or avian TB. Several mycobacterial species can be involved in the aetiology of avian tuberculosis. However, avian tuberculosis is most often caused by *Mycobacterium avium* belonging to serotypes 1, 2, 3 and 6 and *M. genavense*. The capacity of *M. avium* to produce progressive disease may be related to cell wall constituents and certain complex lipids present in the cell wall such as cord factor, sulfur-containing glycolipids (sulfatides) or strongly acidic lipids. Clinical signs are not pathognomonic and vary depending on the organs involved. Mortality over a short period may be insignificant but the intermittent loss of adult birds in valuable breeding stock and decreased egg production in layers are detrimental. Avian Mycobacteriosis also has zoonotic importance and commonly affect the immunocompromised human. The various preventive and control measures are present to control this disease but the diagnosis of the disease is difficult and hence it remains uninvestigated in many parts of our country.

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INTRODUCTION

Mycobacteria cause serious infections in animals and human beings. Huge economic losses on farms are caused by selected species of this wide family. A high risk of transmission of infection from animal to human exists. A few decades ago mycobacteriosis in poultry was a huge problem in the industry. The significance of avian mycobacteriosis in domestic chickens has diminished in many countries as a result of the introduction of more intensive production system that encompass better nutrition, shelter and hygiene, and management practices. Nevertheless, avian mycobacteriosis remains a problem in extensive (traditional) production system under which chicken scavenge for survival in unhygienic environment. This predisposes them for various infections including avian mycobacteriosis. The incidence of mycobacteriosis in pet birds is estimated to be 0.5-14% in post-mortem surveys (Lennox 2007). Avian mycobacteriosis in poultry is a contagious disease and may also be called avian tuberculosis or avian TB. It is caused by *Mycobacterium avium*. Although the term mycobacteriosis can be applied to any infection of mycobacteria, the term avian tuberculosis is more used for the disease caused by *M. avium* or related agents, due to its typical tuberculous lesions.

The bacteria *M. tuberculosis* is also occasionally found in birds but its clinical signs are different (Kearns and Loudis 2003). Avian tuberculosis is a chronic persistent infection in a flock and once established induces unthriftiness, decreased egg production and finally causes death.

Mycobacterial species associated with avian tuberculosis: Several mycobacterial species can be involved in the aetiology of avian tuberculosis. Infection in pet and wild birds has been reported with several subspecies of *M. avium* and other species of *Mycobacterium* including *M. tuberculosis, M. bovis, M. fortuitum* and *M. genavense*. Together with *M. intracellulare, M. avium* forms the *Mycobacterium avium* complex (MAC) which is a commonly used term in the literature. Genotypic studies clearly divide *M. avium* and *M. intracellulare* into two distinct species. *M. avium* in turn consists of the subspecies *M. avium subsp. avium, M. avium subsp. paratuberculosis* (causative agent of Johne's disease in cattle) and *M. avium subsp. silvaticum* (Wood-pigeon bacilli). Disease in poultry is mostly attributed to *M. avium* subspecies *avium*. The subspecies *Mycobacterium avium avium* was originally described as the causative agent of bird tuberculosis but was later found to cause disease also in humans.

Till now 28 MAC serotypes have been identified from which the serotypes 1–6, 8–11 and 21 belong to *M. avium* subsp. *avium* (*M.a.a.*) Serovars 7, 12–20 and 25 have been ascribed to *M. intracellulare*. However, no consensus was achieved on other serovars and some isolates cannot be typed. Serotypes 1, 2 and 3 are considered virulent for chickens. Serotypes 1 and 2 are most commonly isolated from domestic birds and serovar 3 is recovered sporadically from wild birds. *M. avium* is resistant to high and low temperatures, dryness, pH changes and many commonly used disinfectants. However the unprotected organism is killed by direct sunlight (Dhama et al. 2011).

Epidemiology: Under favourable conditions virtually all species of birds are susceptible to *M.a.a.* infection, although among domestic birds hens (*Gallus domesticus*) are the most susceptible species (Shitaye et al., 2007). *M. avium* causes avian tuberculosis in probably all avian species especially in waterfowl, galliformes, columbiformes, passerines, psittacines, raptors and ratites. *M. avium* is highly resistant to environmental challenges and can survive in soil for up to 4 years and this makes eradication of the organism difficult. Molecular techniques have been shown to be more suitable for use in the epidemiologic study of *M. avium* complex (MAC) infection. Avian tuberculosis has been reported in domesticated or captive-raised ducks, geese, swans, peafowl, pheasants, quail, partridge, pigeons, doves, turkeys, birds of prey, and other captive and/or wild birds. Pet birds including parrots, cockatoos, budgerigars, finches, fly catchers and canaries have been infected. Avian tuberculosis in chickens caused by *M. avium* serovars 1, 2 and 3 is worldwide in distribution but occurs most frequently in the North Temperate Zone. Tuberculosis causes important death losses in captive wild birds of zoo aviaries. The significance of these findings is emphasized by reports of disease in valuable endangered species (Vanderheyden 1986).

Transmission: Faecal discharges contain tubercle bacilli from lesions of the liver and mucosa of the gallbladder expelled through the common bile duct. *M. avium* bacilli remained viable in carcasses buried 3-feet deep for 27 months. Virulent strains of *M. avium* have been found to survive in sawdust for 168 days at 20°C and 244 days at 37°C. *M. avium* has been isolated from eggs of naturally infected chickens but hatched chicks failed to develop avian tuberculosis. *M. avium* does not survive in eggs after 6 minutes of boiling and in preparation of scrambled eggs 2 minutes of frying was sufficient to kill the bacteria (Dhama et al. 2011).

Pathogenesis: Ingestion of the bacillus results in intestinal infection and eventual bacillemia. Bacillemia allows for the transfer of bacilli from the intestine to the liver directly. The bacillemia, which probably occurs intermittently, and perhaps, early in most instances also provides for a generalized distribution of lesions. No tissue with the possible exception of the central nervous system appears to be exempt from infection. The disease process is divided into three periods a latency period, a lesion development period and a cachexia period. The latency period occurs for the first 7 days of the infection. During this period there are no microscopic lesions but DTH reactions increase in intensity with time. The lesion development period occurs from days 8–17 post infection. Bacilli multiply in lymphoid sheaths during this time. Serum antibody titers develop, the thymus atrophies and small tubercles with few bacilli develop. Cachexia lasts from day 18 until death.

The DTH contributes to accelerated tubercle formation and is in part responsible for cell-mediated immunity in tuberculosis. Activated macrophages that lack sufficient subcellular microbiocidal components to kill virulent tubercle bacilli are destroyed by the intracellular growth of the organism and a lesion develops.

A combination of toxic lipids and factors released by virulent *M. avium* may

- Cause disruption of the phagosome,
- Inhibit phagolysosome formation,
- Interfere with the release of hydrolytic enzymes from the attached lysosomes,
- Inactivate lysosomal enzymes released into the cytoplasmic vacuole.

Toxic oxygen metabolites are not responsible for killing activated macrophages. However the significance of hydrogen peroxide activated oxygen radical(s) and nitric oxide in resistant macrophages of birds exposed to virulent *M. avium* remains to be elucidated (OIE 2014). More recently *M. avium* has been shown to induce caspase-1 activity in macrophages and may serve as a mechanism for its pathogenicity.

Clinical Signs: Clinical signs are not pathognomonic (Mondal 2015). In avian TB the clinical sign vary depending on the organs involved. Birds with the intestinal form of tuberculosis often present with chronic wasting disease. In majority of cases of tuberculosis in birds especially in the initial phase of infection clinical signs are not grossly observable. However in advanced cases birds may develop symptoms like progressive weight loss, depression, white diarrhoea with soiled feathers, increased thirst, respiratory distress, fatigue and decreased egg production. Feathers are often dull or ruffled and combs wattle, ear lobes often appear pale, thinner and dry. In advanced infections the bird will be less lively than its pen mates, will fatigue easily and may be depressed. Although appetite usually remains good, progressive and striking loss of weight commonly occurs evident as atrophy of breast muscles with a prominent keel. In many instances the bird reveals a unilateral lameness and walks with a peculiar jerky hopping gait. Paralysis from tuberculous arthritis can sometimes occur. With advanced emaciation nodular masses can be palpated along the intestine. However, the hepatomegaly that many tuberculous birds possess may make this procedure difficult or impossible.

Macroscopic Pathology: Tuberculous (TB) lesions due to avian mycobacteriosis can vary according to the bird species infected. Lesions can be tuberculoid or non-tuberculoid. Lesions are only occasionally characterized by classical tuberculous lesions and thereby "bird tuberculosis" is more correctly called mycobacteriosis in birds. According to Prukner-Radovcic et al. (1998) three forms of disease manifestations have been described: the classic tuberculosis (a disease with widespread tubercle formation in most organs), the intestinal form (intestine with typical intestinal lesions only) and the so called non-TB form which is almost macroscopically unrecognisable. *Mycobacterium avium* subsp. *avium* (*M.a.a.*) of serotypes 1, 2 and 3 and genotype *IS901+* and *IS1245+* causes a chronic infection in several species of birds that is characterised by granuloma formation in various organs.

Recently scientists have stated that the principal lesions of tuberculosis in birds are seen in intestine where affection often presents with studded greyish-white to greyish-yellow nodules. Lesions of avian tuberculosis in chickens are characterized by pinpoint to several centimeter, irregular greyish yellow or greyish white nodules in spleen and liver. Due to this spleen takes irregular "knobby" appearance. Involvement of liver and spleen results in enlargement which can result in fatal hemorrhage from rupture. The pulmonary lesions, which are a striking feature of tuberculosis in other species, are rarely observed in birds. Pulmonary avian tuberculosis is only seen occasionally as in case of tuberculosis of pigeons and water fowl (Marco et al. 2000). For turkeys, ducks and pigeons, lesions predominate in the liver and spleen but occur also in many other organs.

Microscopic pathology: The basic lesion of *M. avium* infection consists of multiple granulomas with a central caseous necrosis. In larger nodules the central area of the granuloma may have coagulative or caseous necrosis.In large nodules only the multinucleate giant cells may persist as a mantle around the necrotic core. Immediately peripheral to the multinucleate giant cells is a collection of both epithelioid and histiocytic macrophages. A fibrous capsule consisting of fibrocytes and minute blood vessels also occurs near the outer portion of the peripheral area. The outermost region of the granulomas is encapsulated by fibrous connective tissue, macrophages, some lymphocytes and an occasional granulocyte. Calcification of the tubercle rarely occurs in fowl. Amyloid deposition in the surrounding parenchymal elements has been reported in liver, spleen, and kidney.

Infection to other species: *Mycobacterium avium* can not only infect all species of birds but can also infect some domesticated mammals to cause the disease usually with localized lesions. Disseminated tuberculosis caused by *Mycobacterium avium* has also been reported in rabbits and swine (Fulton and Sanchez 2008). Unlike the other species mentioned previously cattle are highly resistant to the causative agent of avian tuberculosis and tuberculous lesions are detected in head lymph nodes or occasionally in liver lymph nodes only on meat inspection. *Mycobacterium avium avium* can be successfully isolated from tuberculous lesions in mesenteric lymph nodes from juvenile cattle. The isolation rate from cattle under 2 years of age was 34.4% in contrast to 13.0% from cattle over 2 years of age.

Infection from birds to human: All members of *M. avium* complex and *M. genavense* are capable of giving rise to a progressive disease in humans that is refractory to treatment especially in immunocompromised individuals (Tell et al. 2001). Moreover, *M. avium* infections have been commonly reported in patients with acquired immune deficiency syndrome (AIDS). In the United States *M. avium* serovars 1, 4 and 8 are isolated most frequently from AIDS patients and serovars 4, 8, 9, 16 and 19 are isolated most frequently from non-AIDS patients (Beamer et al. 2008). Recently, Mondal (2015) has observed that in humans *M. avium* infection is rare from birds, is extremely rare in immunocompetent individuals and most often occurs in immunocompromised patients such as AIDS/HIV patients.

Infection from human to birds: Although infections with *M. tuberculosis* in birds are rare infected birds could create a potential source of untreated tuberculosis and therefore have implications for public health in human-to-bird and possibly bird-to-human transmission of tuberculosis (Steinmetz et al. 2006). Psittacines are the most common avian species known to become infected with *Mycobacterium tuberculosis* presumably as a result of close contact with tuberculous owners.

Diagnosis: Office Internationale des Epizooties (2014) evaluated the Test methods available for the diagnosis of avian tuberculosis. A presumptive diagnosis of pathogenic avian mycobacteriosis in fowl usually can be made based on gross lesions. Demonstration of acid fast bacilli in smears or histologic sections of liver, spleen or other organs strengthens the diagnosis and is sufficient for most diagnostic cases. OIE (2014), states that when administered properly tuberculin test provides a satisfactory procedure for determining presence of avian tuberculosis in a flock. However, tuberculin testing in poultry may reveal a false-negative result twice during the course of infection once during early infection and again during late infection when there is immune system exhaustion or anergy. This test is also unreliable in some bird species. The technique involves intradermal injection of the wattle with 0.03–0.05 ml of a purified protein derivative tuberculin prepared from *M. avium*. The injection site then is monitored for a reaction.

Serologic tests available include agglutination, complement fixation (CF) and ELISA. These tests are highly species-specific and they are available for only a limited number of species (OIE 2014). A whole-blood agglutination test has been described for diagnosis of avian tuberculosis in fowl. The agglutination test has been more useful for detecting infected birds in a diseased flock however occurrence of false-positive agglutination reactions in healthy birds is a drawback. Zsivanovits et al. (2004) assessed the diagnostic accuracy of an enzyme-linked immunosorbent assay (ELISA) and evaluated it as a diagnostic screening aid for avian tuberculosis. The ELISA showed a sensitivity of 76.9% and a specificity of 55.6% using post-mortem findings as the 'gold standard'. Enzyme-linked immunosorbent assay (ELISA) has detected mycobacterial antibodies in sera of chickens experimentally inoculated with *M. avium* serovar 2 but false-positives may be common. ELISA is considered less specific than the tuberculin test (OIE 2014). *Mycobacterium a. avium* grows best on media such as Lowenstein–Jensen, Herrold's medium, Middlebrook 7H10 and 7H11 or Coletsos, with 1% sodium pyruvate added. Typically *M.a.a.* produces 'smooth' colonies within 2–4 weeks rough variants do occur. The DNA probe assay is a rapid method for identifying various species of *Mycobacterium* grown in culture. Although a very sensitive (95%) and specific (100%) test the currently available gene probe is not sensitive enough to directly detect acid-fast bacilli in specimens. Moreover, identification errors were reported due to the cross-reactivity which may have serious consequences.

Differential diagnosis: The most expedient way to diagnose the disease is by necropsy. Granulomas are rather characteristic but other conditions must be differentiated. These include coligranulomas (Hjarre's disease), pullorum disease, other *Salmonella* infections, *Staphylococcus* infection, fowl cholera, aspergillosis and neoplasia.

Prevention and control: OIE (2014) recommends the following procedures for establishing and maintaining avian tuberculosis free backyard flocks.

- Abandon old equipment and establish other facilities on new soil. Ordinarily it has been impractical to render an infected environment satisfactorily safe by disinfection. 2) Provide proper fencing or other measures to prevent unrestricted movement of chickens thus preventing exposure from previously infected premises.
- Eliminate the old flock burning carcasses of birds that show lesions of tuberculosis.
- Establish a new flock in the new environment from avian tuberculosis free stock. If chickens in a clean flock are prevented access to an infected environment and are protected against accidental exposure to an infected environment and accidental exposure to *M. avium*, it is reasonable to believe that they will remain free from avian tuberculosis.

Recommendations for control of avian tuberculosis in exotic birds include the following

- Prevent contact with tuberculous birds premises and housing previously used by them are to be avoided.
- Quarantine additions to the aviary for 60 days and retest with avian tuberculin.

Vaccination: No vaccines are available for use in birds. There are only rare reports of vaccination against mycobacteriosis in birds. The bacille Calmette-Guérin (BCG) vaccine a human product directed against *Mycobacterium tuberculosis* was tried in poultry but was found to be of little benefit. A vaccine against *Mycobacterium avium* also has been given to poultry and more recently captive waterfowl in Britain. Recently, vaccination of chickens using various fractions of a homologous strain of *M. avium* for vaccine production and challenge revealed that the number of lesions and bacilli per gram of liver were decreased it did not however prevent infection.

Conclusion

Avian mycobacteriosis represent veterinary and economic risks in birds as well as mammals. In India the intermittent loss of breeder stock with decrease production in layers could plausibly be linked to sub clinical or chronic infection by *Mycobacterium avium avium.* Due to long latency periods, difficulties in diagnosis, lack of effective treatment and a pronounced tenacity of *Mycobacterium avium* in the environment, avian TB can easily spread undetected and represents a significant threat to both captive and wild populations in contact with infected birds. The significance of an outbreak of avian TB in aviary is considerable due to the effects on breeding programmes. Diagnosis of the disease is notoriously difficult and thus the disease remains uninvestigated in many parts of our country.

It is emphasized that concentrated efforts are required, to assess the prevalence of potentially pathogenic mycobacteria in meat and the risk of human consumption of contaminated meat. The presence of avian TB in local chicken in the society who has low awareness about zoonotic TB poses a serious threat to human health. To conclude, the importance of Avian Mycobacteriosis cannot and should not be undermined if the tuberculosis prevention programmes implemented by our Government are to have a potential effect.

REFERENCES

Beamer, G.L., Flaherty, D.K., Assogba, B.D., Stromberg, P., Gonzalez-Juarrero, M. et al. 2008. Interleukin-10 promotes *Mycobacterium tuberculosis* disease progression in CBA/J mice. *J. Imm*. 181: 5545-5550.

Dhama, K., Mahendran, M., Tiwari, R., Singh, S.D., Kumar, D. et al. 2011. Tuberculosis in birds: Insights into the *mycobacterium avium* infections. *Vet Med Int*, pp 14.

Fulton, R.M. and Sanchez, S. 2008. Tuberculosis In*:* Saif YM, Fadly AM, Glisson JR, McDougald LR, Nolan LK, Swayne DE Eds. Diseases of Poultry, (12th edn), Blackwell Publishing, Oxford, UK, pp 940-951.

Kearns, K.S. and Loudis, B. 2003. Avian mycobacteriosis. Recent Advances in Avian Infectious Diseases, NY, Recuperado.

Lennox, A.M. 2007. Mycobacteriosis in Companion Psittacine Birds: A Review. *J Avi Med Surg*, 21(3): 181-187.

Marco, I., Domin, M. and Lavin, S. 2000. *Mycobacterium* infection in a captive-reared capercaillie (*Tetrao urogallus*) *Avian Diseases*, 44: 227-230.

Mondal, D. 2015. Zoonotic implication of duck and chicken diseases on public health concern. *S M Uni Med J,* 2(1): 317-329.

OIE 2014. Avian tuberculosis. Manual of Diagnostic Tests and Vaccines for Terrestrial Animals Chapter 2.3.6 pp 497-508.

Shitaye, J.E., Matlova, L., Horvathova, A., Moravkova, M., Dvorska-Bartosova, L., et al. 2007. *Mycobacterium avium* subsp. *avium* distribution studied in a naturally infected hen flock and in the environment by culture serotyping and IS901 RFLP methods. *Vet Microbiol,* 127(1-2): 155.

Steinmetz, H.W., Rutz, C., Hoop, R.K., Grest, P., Bley, C.R. et al. 2006. Possible human-avian transmission of *Mycobacterium tuberculosis* in a green-winged macaw (*Ara chloroptera)*. *Avian Diseases*, 50(4): 641-645.

VanDerHeyden, N. 1986. Avian tuberculosis: diagnosis and attempted treatment In*:* Proceedings of the Annual Conference of the Association of Avian Veterinarians, pp 203-214.

Zsivanovits, H.P., Neumann, U., Brown, M.J. and Cromie, R.L. 2004. Use of an enzyme linked immunosorbent assay to diagnose avian tuberculosis in a captive collection of wildfowl. *Avian Pathol.*, 33(6): 571-575.

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