Lecture 1 / Dr.Abtisam Jwaad Ali/2022-2023

Avian Mycoplasmosis

History

-The first possible isolation of Mollicutes was in 1898 by Nocards and Roux from a cattle viewing signs of contagious bovine pleuropneumonia (CBPP), from which the‘’Pleuro Pneumonia like Organisms (PPLO)’’ avian Mycoplasma was by Dodd in 1905 in England (sinusitis in turkeys)

”,Delaplane and Stuart (1943) referred to the etiologic agent of CRD to the pathogen responsible for the infectious sinusitis of turkeys were It was then considered as a member of the PPLO group and later named as *M.gallisepticum* ,Infectious synovitis caused by *Mycoplasma synoviae* was isolated from wild turkeys in Colorado, Georgia and California in 1980,In January 1994, the first house finches with symptoms of *M. gallisepticum* were observed in the Washington DC area,

Avian Mycoplasmosis

pathogenic mycoplasmas including *M.gallisepticum*, *Mycoplasma synoviae* ,*Mycoplasma meleagridis* and *Mycoplasma iowae* .

Mycoplasma are very small prokaryotes ,devoid of cell walls, bounded by a plasma membrane only . “fried egg” type of colony morphology, resistance to antibiotics that affects cell wall synthesis, and complex nutritional requirements.

phylogenetically classified in the

kingdom: Bacteria,

phylum :Tenericutes (soft skin),

class :Mollicutes,

Order :Mycoplasmatales.

Genus :*Mycoplasma,*

Characterization of avian mycoplasma

1-Require a protein-rich medium containing 10—15% animal serum. yeast-derived. *M. synoviae* requires the addition of nicotinamide adenine dinucleotide (NAD).

2-Mycoplasma organisms tend to grow rather slowly, usually prefer an environment of 37—38°C, Resistant to thallium acetate and penicillin, which are frequently employed in media to retard growth of contaminant bacteria and fungi.

3-Typical colonies are small (0.1—1.0 mm), smooth, circular, and somewhat flat with a more dense central elevation.

4- One useful characteristic of *M. gallisepticum, M. meleagridis,* and *M. synoviae* is hemagglutination of erythrocytes from chickens or turkeys. Hemagglutinating antigens are used for hemagglutination-inhibition serologic tests

***Mycoplasma gallisepticum* (MG)**

Mycoplasma gallisepticum (MG) infections are commonly known as chronic respiratory disease (CRD) of chickens and infectious sinusitis of turkeys.

M. gallisepticum diseases are characterized by respiratory rales, coughing, nasal discharge, and conjunctivitis, and frequently in turkeys, infraorbital sinusitis. Clinical manifestations are usually slow to develop, and the infection or disease may have a long course.

“Air sac disease” describes a severe airsacculitis that is the result of MG infection complicated by a respiratory virus infection (e.g., infectious bronchitis or Newcastle disease) and usually *Escherichia coli*. *M. gallisepticum* stains well with Giemsa .By

light microscopy, organisms generally appear coccoid, approximately 0.25—0.5 m in size.

By electron microscopy, organisms show a filamentous or flask-shaped polarity of the cell body. the presence of well-organized terminal organelles (blebs or tip structuresIt has been theorized that such structures govern motility or host-pathogen interactions (e.g., cytadhesion) pathogenicity

Economic Significance

Mycoplasma gallisepticum is the most pathogenic and economically significant of poultry.

Airsacculitis in chickens or turkeys resulting from MG infections, with or without complicating pathogens,causes increased condemnations at processing.
, reduced egg production and feed efficiency,and drops in hatchability

Prevention and control programs,which may include surveillance, medication, vaccinationand elimination of infected breeding stock, account foradditional costs.

Pathogenesis of the Infectious Process

The MG enter the respiratory system by inhalation, aerosol or via the conjunctiva MG mainly grow in the respiratory tract, but other organs such as reproductive tract, brain and eyes are also under attack (indicates that transient systemic infections occur, resulting in acute and chronic diseases at multiple sites the respiratory tract and lungs are frequent sites of infection The critical event for *Mycoplasma gallisepticum* pathogenesis is attachment and colonization of host respiratory epithelium and leading to further pathological changes in the host ,*M.gallisepticum*, which attaches firmly to mucosal cells by its

well organized **terminal organelles or blebs** that govern motility, host-pathogen interactions, and finally pathogenesis

Transmission

The main route of leave of *Mycoplasma gallisepticum* (MG) from the host is via the respiratory tract while infection of the genital tract may result excretion through egg or semen. MG transmitted vertically within some eggs (transovarian) from infected breeders to progeny, and horizontally via infectious aerosols and through contamination of feed, water, and the environment, and by human activity on fomites (shoes, equipment, etc). Flock-to-flock transmission occurs readily by direct or indirect contact from the movement of birds, people, or fomites from infected to susceptible flocks.

Morbidity rate

M. gallisepticum infection usually affects nearly all chickens in a flock but is variable in severity and duration. It tends to be more severe and of longer duration in the cold months and affects younger birds more severely than mature birds

Newcastle disease (ND) or infectious bronchitis (IB) may precipitate outbreaks of MG infection, *Escherichia coli* has been found to be a frequent complicating organism.

Mortality rate In broilers the mortality may range from low in uncomplicated disease to as much as 30% in complicated outbreaks, especially during the colder months.

Clinical Signs

Chickens

Incubation Period in experimental infections of chickens varies from 6—21 days. the most characteristic signs are tracheal rales, nasal discharge, and coughing. Feed consumption is reduced,

and birds lose weight. In laying flocks, egg production declines ,male birds frequently have the most pronounced signs, and the disease is often more severe during winter. In **broiler flocks**, most outbreaks occur between 4—8 weeks of age. Severe outbreaks with high morbidity and mortality observed in broilers are frequently due to **concurrent infections .**Cases of keratoconjunctivitis caused by MG in commercial layer chickens, showed swelling of the facial skin and the eyelids, increased lacrimation, congestion of conjunctival vessels, and respiratory rales.

Turkeys.

Incubation Period within 6—10 days ,in experimental infections of Turkeys are more susceptible to MG than chickens, more severe clinical signs including sinusitis, respiratory distress, depression, decreased feed intake, and weight loss. swelling of the paranasal (infraorbital) sinuses. As the disease progresses, affected birds become thin. Tracheal rales, coughing, and labored breathing may become evident if tracheitis or airsacculitis is present. in 12—16-week-old commercial meat turkeys displaying torticollis aencephalitic form of MG .

Gross

catarrhal exudate in nasal and paranasal passages, trachea, bronchi, and air sacs. Sinusitis in turkeys , chickens and other affected avian hosts pneumonia may be observed. In severe cases of typical air sac disease in chickens or turkeys, there is the triad of airsacculitis, fibrinous or fibrinopurulent perihepatitis, and adhesive pericarditis resulting in high mortality and extensive condemnations at processing. Commercial layer chickens with MG-associated keratoconjunctivitis had marked edema in the facial subcutis and eyelids, with occasional corneal

opacity Conjunctivitis in house finches and other songbirds salpingitis

Microscopic.

marked thickening of the mucous membranes of affected tissues from infiltration with mononuclear cells and hyperplasia of the mucous glands Focal areas of lymphoid hyperplasia found in the submucosa In trachea, destruction of cilia. In the lungs, lymphofollicular changes and granulomatous lesions are also found.

DIAGNOSIS

Isolation and Identification of Causative Agent

The gold standard for MG diagnosis is isolation and/or identification of the organism. For MG culture, suspensions of tracheal or air sac exudates, turbinates, lungs, or fluid sinus exudate should be inoculated directly to mycoplasma broth or agar medium PPLO ,Swabs can also be taken from the trachea or choanal cleft (palatine fissure) for MG culture *M. gallisepticum* may also be present in oviducts and rooster semen ,to optimize the possibility of isolation, flocks should be sampled for MG culture prior to initiation of antimicrobial therapy).

Control

1-A crucial step in the control of the disease was maintaining breeding flocks free of *Mycoplasma gallisepticum* is biosecurity should be based on the avoidance of *Mycoplasma gallisepticum* enterance to the farm.

2-The “all in-all out”system may allow eradication of *M.gallisepticum*).

3- Using of antibiotic in the prevention of egg transmission by Dipping or Injection of eggs with antibiotics helped to limit

vertical transmission (Heating eggs in a forced air incubator (12-14) hours to reach an internal temperature of 46.1°C -,.

4-Medication is the main objectives of antibiotic medication are to reduce clinical signs, lesions.

5- Monitoring is performed using serology test ELISA and HI, with confirmation of mycoplasma by PCR.

6- Vaccination,in activated, oil emulsion bacterins, live vaccines or, recombinant live vaccine. three live MG vaccines; F, 6/85 and ts-11 which originated from field isolates which induce protection against MG These alive vaccines may be apply by spray, eye drop and drinking water.

Treatment

tetracyclines ,macrolides (erythromycin, tylosin, spiramycin, lincomycin, and kitasamycin), quinolones (imequil, norfloxacin, enrofloxacin and danofloxacin) or tiamulin.Drugs that accumulate in high concentrations in the mucosal membranes of the respiratory and genitourinary tracts, such as tiamulin and enrofloxacin are frequently chosen .

***Mycoplasma synoviae* Infection**

Mycoplasma synoviae (MS) infection most frequently occurs as a subclinical upper respiratory infection. It may cause air sac lesions when combined with Newcastle disease (ND), infectious bronchitis (IB), or both. At other times, MS becomes systemic and results in infectious synovitis, an acute to chronic infectious disease of chickens and turkeys, involving primarily the synovial membranes of joints and tendon sheaths producing an exudative synovitis, tenovaginitis, or bursitis.requirement for nicotinamide adenine dinucleotide (NAD).Infectious synovitis was observed primarily in growing birds 4—12 weeks of age in

broiler-growing .Chickens, turkeys, and guinea fowl are the natural hosts of *M. synoviae.* Ducks Lateral transmission. And Vertical transmission occurs in naturally and artificially infected chickens

Incubation11—21 days.

Clinical Signs

Chickens. The first observable signs in a flock affected with infectious synovitis are pale comb, lameness, and retarded growth. As the disease progresses, feathers become ruffled and the comb shrinks. Swellings usually occur around joints, and breast blisters are common. Hock joints and foot pads are principally involved, Acute signs described previously are followed by slow recovery; however, synovitis may persist for the life of the flock. Chondrodystrophy was noted in the opposite leg of chickens inoculated via the foot pad. Air sac infection resulting from *M. synoviae* infection occur in winter. Progeny of MS infected breeders may have increased air sac condemnations, reduced weight gains, and reduced feed efficiency.

Turkeys. *M. synoviae* generally causes the same type of signs in turkeys as in chickens. Lameness is the most prominent sign..

Differential Diagnosis

Bacteria as causes of synovitis or arthritis. *Staphylococcus aureus, Escherichia coli,* pasteurellae, and salmonellae may also be present as primary causes of synovitis. *M. gallisepticum* may also be a cause of breast blisters and joint lesions ,

Viral arthritis Fibrosis of metatarsal extensor or digital flexor tendons and lymphocytic infiltration of the myocardium associated with the viral arthritis agent help to differentiate it

from *M. synoviae,* Serum from viral tenosynovitis infected chickens does not agglutinate MS antigen

Mycoplasma meleagridis (MM)

Mycoplasma meleagridis (MM) is a specific pathogen of turkeys. It is the cause of an egg transmitted disease in which the primary lesion is an airsacculitis in the progeny,decreased hatchability, skeletal abnormalities, and poor growth performance. Infection of the female reproductive tract occurs as an endogenous infection during embryonic development , as an ascending infection from foci in the cloaca or by insemination of hens with MM-containing semen Infection rates of 19—57% .

Mycoplasma iowae Infection

Mycoplasma iowae has been associated with reduced hatchability and embryo mortality in turkeys. It has also been shown experimentally to induce mortality in turkey and chicken embryos and mild to moderate airsacculitis and leg abnormalities in chickens and turkeys.[2]Poult hatchability can be reduced by 2—5%. The natural host appears to be the turkey, but isolation of *M. iowae* from chickens is not uncommon and it has also been reported in geese

MYCOPLASMA IMITANS

Mycoplasma imitans is of interest because of its close relationship to *M. gallisepticum. M. imitans* strains share many phenotypic properties with *M. gallisepticum,* including biochemical reactions, hemadsorption, hemagglutination, and the presence of an attachment organelle. It reproduced respiratory disease similar to but somewhat milder than *M. gallisepticum* in turkeys reproduced a respiratory disease that was more severe when it was present in a dual infection with

rhinotracheitis virus,*M. imitans* did not produce signs or lesions when inoculated into chickens, but in a dual infection with infectious bronchitis virus, a synergistic effect was seen.

MYCOPLASMA GALLINARUM

M. gallinarum has not been considered to be one of the pathogenic avian mycoplasma species, but there is one report of consistent isolation from air sacs and tracheas from a series of broiler flocks that were having higher than normal condemnations due to airsacculitis. One of those isolates had the ability to induce airsacculitis when given in conjunction with Newcastle disease–infectious bronchitis vaccine.*M. gallinarum* ordinarily is isolated primarily from chickens, but it may also been found in turkeys It has been isolated from jungle fowl ducks and pigeons .

MYCOPLASMA PULLORUM

M. pullorum was classified as avian serotype C and was later named *Mycoplasma pullorum* . It has been isolated from chickens, quail, partridge, pheasants, and turkeys . *M. pullorum* has been isolated from turkey embryos from flocks in France that were experiencing low hatchability and was shown to be pathogenic for chicken and turkey embryos.