MYCOPLASMA MELEAGRIDIS INFECTION

Slide study set #13

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Mycoplasma meleagridis (MM) was first isolated in 1958 from turkey poults with air-sac lesions. Being antigenically distinct from *M. gallisepticum*, which was the mycoplasma of primary concern in turkeys at that time, the new serotype was named "N-strain." Since then, the organism has been classified into the H-serotype and given the present species name. The clinical syndrome of airsacculitis and/or associated skeletal abnormalities has been referred to as "day-old-type" airsacculitis and "turkey-syndrome-'65" (TS-65).

Distribution and incidence. MM has a worldwide distribution, being found wherever turkeys are raised. It is a specific pathogen of turkeys and causes a high incidence (20-65%) of airsacculitis in day-old poults.

Pathogenicity and associated disease conditions. The disease manifestations and economic losses due to MM are associated with embryo infection via egg transmission and include (a) late incubation (25-28 days) mortality of hatching eggs; (b) bone deformities, particularly during the first 3 to 6 weeks of life; (c) a decrease in livability and growth rate (related to item b above; and (d) condemnation at processing of fryer-roasters due to airsacculitis. Recent studies with experimental and naturally infected eggs suggest that MM causes hatchability losses of approximately 5%. The skeletal problems (TS-08) include bowing, twisting, and shortening of the tarsometatarsal bones, hock-joint swelling, and deformity of the cervical vertebrae. Stunting and poor feathering may accompany the disease. Affected flocks may show a 5 to 10% incidence of gross lesions. Mortality is due primarily to cannibalism.

Although the organism invades the upper (sinus and trachea) and lower respiratory tract, lesions are confined to the air sacs. Consequently, rales and sinusitis are not observed, and the newly hatched poult with extensive airsacculitis shows no clinical signs. Lesions, which first develop in the thoracic air sacs, progress to the abdominal air sacs by the third to fourth week of age. If uncomplicated, the lesion regresses in 15 to 16 weeks of age. MM interacts synergistically with *M. synoviae* to produce sinusitis and with *M. iowae* to produce a more severe airsacculitis.

Despite the high egg-transmission rate of MM in infected flocks (average of 25% over a season's lay), the skeletal deformities do not always accompany embryo infection, and condemnation at processing is not necessarily a consequence of MM airsacculitis. What determines whether an embryo will develop skeletal problems is not clear, but such factors as MM strains of varying pathogenicity and environmental stress during hatching or brooding may influence the disease picture. Secondary bacterial or viral infections may contribute to mixed air-sac infections, resulting in condemnation of fryer-roasters.

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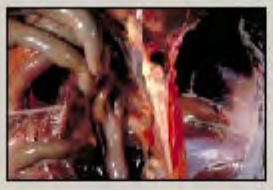

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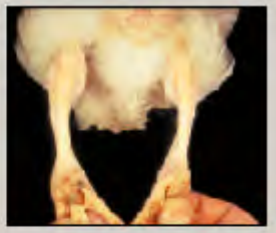

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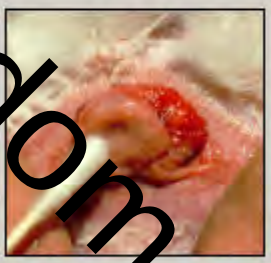

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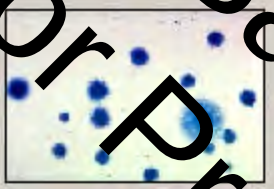

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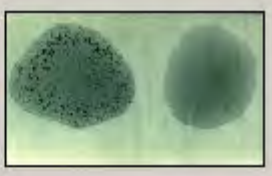

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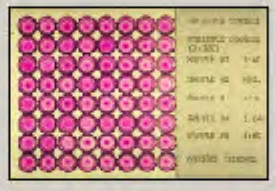

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