FOWL CHOLERA

Slide study set # 19

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This slide study set was created in 1995; some information may be outdated

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Fowl cholera is a septicemic disease caused by *Pasteurella multocida* which affects a variety of domesticated and wild birds. This highly contagious disease causes high morbidity and mortality resulting in great economic losses, especially in large industrial-type poultry complexes. It usually occurs as an acute disease, but chronic infections can also occur in some outbreaks.

In 1880, Louis Pasteur performed the fundamental experiments to attenuate these organisms for inducing active immunity. In his honor, "Pasteurella" was adopted to designate these bacteria in 1887. In the past, *P. multocida* has been called by many names: *P. septica*, *P. gallicida*, *P. avisepticida*, etc.

Etiology. *Pasteurella multocida*, is a small (.25 x .62 x 2.25 µm) gram-negative, nonmotile, non-spore-forming bacillus that can become pleomorphic or filamentous after prolonged cultivation *in vitro*. Gram staining reveals not only the size and shape of the bacteria, but also a special tinctorial quality that makes it bipolar. Bipolar characteristics of the organism, however, are best visualized by Wright or methylene-blue staining. This property is lost with continued cultivation on artificial media. *P. multocida* is a facultatively aerobic bacterium that grows best at 37° C. It grows on blood agar, but not on MacConkey agar. Three colony types have been described: mucoid with moderate-to-high mouse virulence, smooth with high-mouse virulence, and rough with low-mouse virulence. Mucoid colonies are not common. Virulence of *P. multocida* is highly variable, depending upon whether the strains are encapsulated. Encapsulated strains are generally virulent; strains that are not capsulated are usually of low virulence. *P. multocida* isolates have been typed into various serogroups based on serologic tests. Specific capsule serogroup antigens are recognized using passive hemagglutination tests. Five capsular serogroups, --A, B, D, E and F--, are currently recognized. *P. multocida* serogroup A is the usual cause of acute fowl cholera. Somatic serotyping has been done by tube agglutination tests and gel diffusion-precipitin tests. To date, 16 somatic serotypes have been described, all of which have been isolated from avian hosts. In the United States the gel diffusion test has been used routinely because of its simplicity.

P. multocida is usually destroyed by common disinfectants, sunlight, drying or heat. Unlike most gram-negative bacteria, *P. multocida* is usually susceptible to penicillin. It is also susceptible to tetracycline, polymyxin, chloramphenicol, novobiocin, neomycin, gentamicin and sulfonamides.

Epizootiologic characteristics. Fowl cholera is an enzootic disease with a remarkable trend to spread. The disease exists in most countries throughout the world, but it is more frequent in temperate and warm zones. In most European countries a sharp decline of fowl cholera occurred after 1930. However, sporadic outbreaks do appear from time to time. In the U.S., it is a fairly common disease of broiler breeders, but is a major economic problem for the turkey industry because of its rapid spread, and associated high morbidity and mortality.

Susceptibility. All domestic and wild species of birds are susceptible to fowl cholera. Most reported outbreaks involve chickens, turkeys and ducks, and occasionally species such as geese, pigeons, pheasant, quail, sparrows and finches. In turkeys, fowl cholera generally occurs between 10 to 13 weeks of age. It rarely occurs in birds less than 2 or 3 weeks of age.

Sources of infection. The primary source of *P. multocida* infection can be excretions from the nostrils, mouth and eyes of sick birds or chronic carriers. The secondary sources are contaminated feed, water, crates, equipment and shoes. Wild birds, including sparrows and pigeons and many mammals (especially pigs, cats, and wild rodents) can disseminate *P. multocida*. The organism can persist for years in the oral cavity of rodents and carnivores. Birds bitten by such animals can become infected and disseminate the disease within the flock. Cannibalism of sick or dead birds is also a significant method of dissemination.

Pathogenesis. *P. multocida* usually enters the host through the mucous membranes of the upper respiratory tract and probably the digestive tract also. The ability of pasteurella to resist phagocytosis after invading tissues allows these bacteria to multiply very quickly, causing septicemia and severe endotoxemia; death often ensues within 24 hours. The role of endotoxemia, however, in fowl cholera is not clear .The pathogenesis of clinical signs and lesions depends upon various factors such as virulence of the infecting pasteurella strain, the infecting dose, the age and immunologic competency of the host, the route of infection, and other predisposing factors including poor nutrition, environmental temperature and concurrent viral infections that may induce immunosuppression.

Clinical signs. The incubation period is generally short – about 1 to 4 days. Three forms of the disease can be distinguished: peracute, acute and chronic. In the peracute form, clinical signs may be absent; birds are found dead in their nests, near their feeders or sometimes with their beaks in the water.

The acute form of fowl cholera is the most common clinical form. Signs are fever, anorexia, ruffled feathers, mucus discharge from the mouth, diarrhea and increased

REFERENCES

1. Rhoades, K. R. and R. B. Rimler. Fowl Cholera. In Diseases of Poultry. Edited by B. W. Calnek, H. J. Barnes, C. W. Baird, W. M. Reid, H. W. Yoder Jr. 9th ed. Iowa State University Press, Ames, IA. pp. 145-162, 1991.

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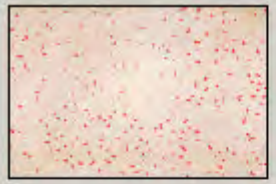
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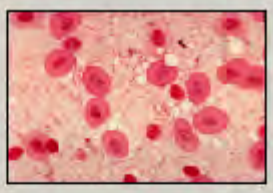
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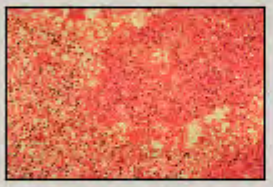
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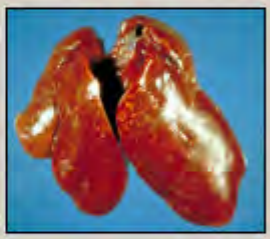
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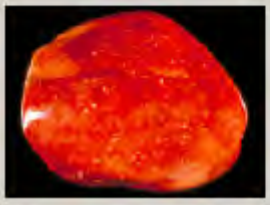
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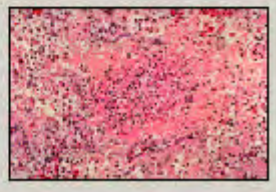
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