Fowl Cholera

A continuing education program prepared

by

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Introduction

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 this bacteria in 1887. In the past, *P. multocida* has been called by many names *P. septica*, *P. avicida*, *P. aviseptica*, 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's 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 tetracyclines, polymyxin, chloramphenicol, novobiocin, neomycin, gentamicin and sulfanomides.

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 is 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 this 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 respiratory rate. Cyanosis often occurs immediately prior to death and is most evident in unfeathered areas of the head such as comb and wattles. Diarrhea most common in duck is whitish in color but later becomes greenish and contains mucus.

The chronic form of fowl cholera is most common in chickens and may follow an acute stage of the disease or result from infection with organisms of low virulence. The birds may exhibit anemia, diarrhea, progressive weakness, weight loss and death. Signs are generally related to localized infections such as swollen wattles, sinuses, foot pad or tendon sheaths. Conjunctivitis, pharyngeal abscesses, and torticollis due to involvement of the middle ear, ear canal, or the bones of the cranium may also be observed. The chronic form may last 2 to 3 weeks or longer.

Lesions

Lesions can vary in type and severity, depending upon the stage of the disease. In peracute deaths, there may be no lesions or the liver may be swollen or friable with petechiation on the abdominal viscera. In the acute form, petechial and ecchymotic hemorrhages are frequently found in organs such as epicardium, lungs, abdominal fat, intestinal mucosa and ovarian follicles. The liver may be enlarged and friable and may contain white foci, scattered throughout. Histologically, these foci are composed of multifocal, acute coagulative necrosis of hepatocytes, with infiltration of heterophils mixed with fibrin and few mononuclear inflammatory cells. Lungs of turkeys are more severely affected than those of chickens, with pneumonia being a common sequelae. Histologically, pneumonia appears as fibrinosuppurative inflammation either involving the lung parenchyma or the pleura with an extension into the lung. There may be large numbers of bacteria within this exudate. Exudation into the pericardial sac and the peritoneum can occur. The intestines may have mucosal congestion with increased mucus, which is common in ducks. Sometimes, extensive cellulitis of the head and neck region can be seen, especially in turkeys, biting or pecking may be the mode of transmission in such cases. There may be regression of mature ovarian follicles and rupture of the yolk follicles, resulting in peritonitis in mature birds.

In the chronic form of fowl cholera, the lesions are usually localized, suppurative and may be distributed widely. Lesions often involve joints, tendon sheaths, wattle, conjunctival sac, infraorbital sinus, nasal turbinates, the middle ear or cranial bones and the base of the skull. Pneumonia is common lesion found in turkeys. Consolidated lungs eventually become sequestered and necrotic. It is not unusual for only one lung to be affected.

Turkeys can have fowl cholera as a complication of airsacculitis caused by *Mycoplasma gallisepticum*. In these cases, a marked pericarditis, perihepatitis, airsacculitis and extensive fibrinous pneumonia usually occurs. Occasionally, peritonitis, meningitis, and salpingitis also can be seen.

Diagnosis

History, clinical signs and lesions may strongly suggest fowl cholera. At necropsy, gram-stained impression smears of the livers and heart blood from septicemic cases can reveal bipolar, gram-negative rods suggestive of *P. multocida*. Use of Wright's stain or methylene blue readily demonstrate the bipolar morphology of the organism.

P. multocida can usually be isolated from lesions of chronic cases and isolated readily from viscera of birds that die of acute fowl cholera. Bone marrow, heart blood, liver, meninges or localized lesions are preferred for culturing. Rabbits, hamsters, or mice can be inoculated with a small quantity of the exudate or minced tissue from the infected bird. These animals usually die within 48 hours and the organism can be isolated in pure culture from heart blood or liver. Biochemical tests should be done to identify *P. multocida* positively. *P. multocida* grows readily on blood agar without hemolysis but does not grow on MacConkey's agar. Isolates of *P. multocida* should be tested for antibiotic sensitivity because of its resistance; it should be typed, especially when treatment and control procedures appear ineffective. Currently, an ELISA test is available commercially and is used widely. Serology is used primarily to evaluate the efficacy of vaccination rather than for diagnosis of a disease outbreak.

Fowl cholera must be differentiated carefully from erysipelas, acute colibacillosis, and complicated *Mycoplasma gallisepticum* infection in turkeys and other birds that may have both diseases. Related organisms that can cause cholera-like diseases or complications include *P. gallinarum*, *P. haemolytica*, *P. anatipestifer* and *Yersinia pseudotuberculosis*.

Prevention

Preventing the introduction of fowl cholera in disease-free flocks is essential to prevention. New birds that may possibly be carriers should not be introduced into the flock. Disease-free birds, raised in disease-free premises away from all possible carriers will prevent fowl cholera.

Destroy all sick or dead birds before cannibalizing can occur. Dispose of carcasses properly by burying or burning. Clean and disinfect crates, cages and other equipment used in poultry production after each use.

Oil-emulsion bacterins are used to immunize breeders prior to production, but they can cause a drop in egg production if given to laying birds. Bacterins can be given when the birds are 8 and 12 weeks of age. However, bacterins do not provide adequate cross-protection between various serotypes.

Live oral vaccines for chickens and turkeys are used in many countries. In the United States, live vaccines such as the Clemson University (CU) strain of *P. multocida* and a slow growing mutant (M-9) vaccines have been used. Live oral vaccines frequently are administered

via drinking water to turkeys at 2 to 4 week intervals beginning at 6 to 7 weeks of age. Layers and breeders can be inoculated with fowl cholera vaccine by wing web stick or subcutaneously in conjunction with fowl pox vaccine. Problems can occur due to use of the live vaccines in the field if there are concurrent diseases or other diseases that induce immunosuppression. Parenteral administration of vaccines has been known to cause localized lesions, including severe arthritis. Live vaccines confer better protection against most serotypes than killed bacterins.

Depopulation of the entire flock should also be considered during an outbreak since many surviving birds can become carriers of the disease. After depopulation, the premises and equipment should be thoroughly cleaned and disinfected. The house should be kept free of poultry for several weeks after cleaning and disinfection. Most importantly, strict control measures for rodents, cats, predators, scavengers and wild birds should be adopted. Use of certain antibiotics such as tetracyclines in the feed may help prevent or reduce the mortality rate due to fowl cholera. However, this can be an expensive preventive measure compared to the use of vaccines.

Treatment

Many antibiotics and sulfa drugs are effective in treating fowl cholera. Antibiotics lower mortality when used, but the mortality may resume when the treatment is discontinued. Most of these medications are given in the feed or water. Care should be taken to use only products approved by the Food and Drug Administration for the class of poultry being treated. Be aware of side effects such as drop in egg production. Sulfa drugs and antibiotics used in treating fowl cholera are sulfadimethoxin, sulfaquinoxilin, sulfamethazine, tetracyclines, erythromycin and

penicillin. Consider marketing the flock early if fowl cholera cannot be controlled adequately. Comply with regulations relating to medication withdrawal should be followed.

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DESCRIPTION OF SLIDES

1. Gram-negative rods of *P. multocida* (courtesy Dr. R. B. Rimler, NADC).
2. Bipolar morphology of *P. multocida*, liver impression, Wright's stain (courtesy Dr. R. B. Rimler).
3. Typical colonies of *P. multocida* on a blood agar plate.
4. Turkey with clinical signs of acute fowl cholera showing severe depression.
5. Severe fibrinous pericarditis, perihepatitis, and airsacculitis, turkey.
6. Acute fibrinous pericarditis, turkey.
7. Acute fibrinous pleuritis and pneumonia, turkey. Notice the unilateral involvement of the lung.
8. Cut surface of the lung from a turkey with acute fowl cholera. Notice fibrinous exudate in the parabronchioles as well as fibrinous pleuritis.
9. Histopathology of lung with acute fowl cholera. Fibrinosuppurative exudate in the parabronchiole extending into the adjacent parenchyma.
10,11. Livers from chicken (slide 9) and turkey (slide 10) with acute fowl cholera. White foci of necrosis.
12,13. Histopathology of liver in a turkey with acute fowl cholera. Acute coagulative necrosis of hepatocytes with infiltration of few degranulating heterophils mixed with fibrin and bacterial colonies (slide 12, H and E 15X). Acute coagulative necrosis of hepatocytes and infiltration of degranulating heterophils, fibrin exudation, noticeable especially in the sinusoids (Slide 13, H and E 150X).
14. Enlarged and mottled spleen in acute fowl cholera turkey.

15. Histopathology of spleen with fibrinosuppurative inflammation (H and E 75X).

16. Ovary of a hen with acute fowl cholera. Severe congestion of the follicular membranes.

17. Facial cellulitis, acute fowl cholera, turkey.

18,19. Histopathology of the subcutis demonstrating fibrinosuppurative cellulitis with bacterial colonies (H and E 125X and 375X).

20. Acute fibrinous synovitis, hock joint, broiler breeder.

21. Head tilt (torticollis) due to otitis, broiler breeder.

22. Subacute to chronic pneumonia in a turkey. Again, notice the unilateral involvement of the lung.

23. Chronic fowl cholera in a broiler breeder. Severe swelling of the wattles.

24. Subacute to chronic oophoritis. Discolored nodular ovarian follicles. Differential diagnosis: pullorum disease.

25. Subacute to chronic fowl cholera in a broiler breeder with severe synovitis, cellulitis and tendonitis.