Pullorum Disease and Fowl Typhoid

A continuing education program prepared

by

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In cooperation with the
Continuing Education Committee
of
The American Association of Avian Pathologists
March 1996

AAAP Business Office
University of Pennsylvania
New Bolton Center
Kennet Square, PA 19348-1692

This study set is dedicated to the meory of Dr. G. H. Snoeyenbos. His encouragement and support contributed significantly to the preparation of this work.

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Slide Study Set #22

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INTRODUCTION: Pullorum disease (PD) and fowl typhoid (FT) are highly infectious and contagious diseases of poultry first described in 1900 and 1888, respectively. Losses from PD and FT were so severe that they once impaired the expansion of the poultry industry. The development of rapid serological tests and a voluntary national surveillance program have contributed significantly to the control of these two diseases in commercial poultry flocks in the United States. PD still occurs in backyard flocks, and occasionally in commercial flocks. For example, in 1990 and 1991 there were outbreaks of PD in integrated broiler/roaster operations in the Delmarva and Southeastern regions of the U.S. Currently, reports of FT in the U.S. are rare.

Because of the similarities between pullorum disease and fowl typhoid in regard to clinical signs, pathology, diagnosis and control, these two diseases will be described together. However, there are certain epizootiological and biochemical differences between the causitive agents of the two diseases which will be discussed where appropriate.

SUSCEPTIBLE SPECIES: PD and FT primarily affect chickens and turkeys and are rarely significant diseases in other avian species, although bobwhite quail are highly susceptible to PD.

Birds of all ages are susceptible, but the greatest mortality, sometimes approaching 100%, occurs in birds less than 4 weeks of age, especially in PD. In FT, the disease often continues for months and outbreaks can occur in some mature flocks with no history of an earlier onset. Acute infections of PD in mature fowl are rare.

ETIOLOGY: PD is caused by *Salmonella pullorum* and FT is caused by *S. gallinarum*. Both organisms are Gram negative, facultatively anaerobic, non-motile rod-shaped bacteria, and are members of Salmonella group D. The somatic (O) antigens of both organisms are similar, with the exception of form variations in antigen 12 of *S. pullorum*. Another important member of Salmonella group D is *S. enteritidis*, which also has similar somatic (O) antigens as *S. pullorum* and *S. gallinarum*. Antigenic and biochemical characteristics of the 3 species are shown in Table 1.

Table 1. Antigenic and biochemical differences among *Salmonella pullorum*, *S. gallinarum*, and *S. enteritidis*

	S. pullorum	***S. gallinarum***	***S. enteritidis***
O Antigens	1, 9, 12_1, 12_2, 12_3	1, 9 & 12	1, 9 & 12
H Antigens	-	-	g, m
Motility	-	-	+
Agglutination	+	+	+
Dextrose	+ gas	+ no gas	+ gas
Lactose	-	-	-
Sucrose	-	-	-
Mannitol	+ gas	+ no gas	+
Maltose	-	+ no gas	+
Dulcitol	-	+ no gas	+
Ornithine	+	-	+
Indole	-	-	-
Urea	-	-	-

TRANSMISSION: Both diseases may be transmitted vertically by the transovarian route. The organisms are transmitted via the yolk and survive the incubation process. Disease occurs in mature embryos and in newly hatched chicks. The diseases can also be transmitted horizontally by direct contact between birds. Adult carriers may also shed the organisms in their feces, resulting in lateral spread to other birds through contamination of feed, water, litter, etc. Chicks may also become infected at hatching via contaminated

eggshells. The organisms may be present on the surface of eggshells, and may also be present in the shell. This mode of transmission is probably of minor importance.

CLINICAL SIGNS:

Chicks and poults

Infected embryos may fail to hatch. Infected chicks and poults that do hatch appear lethargic, weak, anorectic, and will huddle near a source of heat. White diarrhea adherent to the vent is common, and was the basis for calling PD "bacillary white diarrhea". Morbidity and mortality typically increases at about the fourth or fifth day of age. Mortaility usually peaks during the second or third week post-hatching. Mortality can vary greatly, but is often very high and can approach 100%; it can be increased by environmental stresses and poor management. Chicks and poults that survive may be small, unthrifty and poorly feathered.

Adults

Often there are no clinical signs in adult birds infected with *S. pullorum*. Infected birds may appear unthrifty and clinical signs of PD include weight loss and decreased egg production. Semi-mature and mature birds are highly susceptible to FT. Clinical signs in birds infected with *S. gallinarum* include a general pallor of the combs and wattles, diarrhea, and decreased egg production; mortality can be substantial.

LESIONS:

Chicks and poults

There may be few or no lesions in young birds which die soon after an acute or peracute episode. Some birds may have pasty white feces adherent to the area around vents. At necropsy, the liver and spleen may be congested and enlarged, with white foci of necrosis. White to pale yellow nodules can occur in the heart, gizzard, lungs, and occasionally in the pancreas, liver, spleen, ceca and large intestine. The intestine may have white plaques on the mucosa, and the ceca may contain cores of white coagulum. Some birds may have swollen joints distended by viscous yellow fluid. Corneal opacity due to exudate in the anterior chamber of the eye may be observed occasionally. Also, peritonitis and distended ureters may be observed in some cases. Microscopically, these lesions are initially comprised of necrotic foci infiltrated by a mixed population of inflammatory cells, which progress to granulomatous inflammation with infiltration by macrophages, lymphocytes and plasma cells.

Adults

Lesions of acute FT in older birds may include enlarged livers (with or without necrotic foci), enlarged spleens and kidneys, and enteritis of the proximal small intestine, often with ulcerations. The cadaver may look pale. Often, there may not be any lesions due to PD in adult birds. Occasionally, there may be nodular myocarditis, pericarditis and peritonitis.

Lesions in the gonads due to PD and FT are similar. The ovaries may have atrophic, discolored or grayish nodular follicles ranging in number from few to many. Microscopically, the ovarian lesions consist of granulomatous inflammation characterized by the coagulation of yolk material, which in turn is surrounded by multinucleated giant cells, a mixed population of inflammatory cells, and fibrosis. Occasionally the oviduct is impacted with caseous exudate. Affected testes may be atrophied.

DIAGNOSIS:

In young chicks and poults, a typical history of high mortality, clinical signs and lesions, especially white or yellow nodules in the heart, gizzard and other organs are most suggestive of PD and FT. Positive serological test results using sera from live birds and Salmonella group D antigen will aid in the diagnosis, but is not conclusive. Other *Salmonella spp.* in serogroup D, including *S. enteriditis*, may be the cause of positive serological reactions. Also, antibodies to other bacteria may result in false-positive reactions in the plate agglutination test.

CONTROL:

Because transovarian transmission has a dominant role in the spread of these diseases, it is mandatory that only eggs from flocks known to be free of PD and FT be used for hatching. Non-infected eggs from clean flocks should be properly fumigated, hatched, and raised on PD and FT-free premises.

Birds which test positive for antibody to group D Salmonella (reactors) can be removed from the flock and tested for PD and FT by culturing organs for the presence of *S. pullorum* and *S. gallinarum.*

The control of insects, rodents and wild birds should be rigorously practiced. Proper disposal of dead birds is essential. Detailed regulations for the control of PD and FT have been developed by the National Poultry Improvement Plan.

TREATMENT:

Because of the serious nature of these two diseases, treatment is not recommended. Antibiotics such as sulfonamides are available for treatment of noncommercial breeding flocks. However, antibiotic treatment may result in poor growth and feed conversion, and recovered birds may become carriers of the disease. Hence, eradication is recommended. Both diseases are reportable to the state veterinarian as soon as the diagnosis is confirmed.

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DESCRIPTION OF SLIDES:

1. Gram negative rods of *S. pullorum*.

2. 18-day-old chick: Liver is enlarged and congested and heart has early stages of myocarditis (white nodules).

3. 18-day-old chick: Liver is enlarged with white foci of necrosis and inflammation.

4. Adult chicken: Liver with multifocal necrosis and inflammation. (Courtesy Dr. M. Peckham)

5. & 6. Histopathology of liver: Focal hepatic necrosis with fibrin exudation (sinusoidal congestion) (slide 5), hepatic necrosis, severe heterophilic infiltration and mild biliary hyperplasia (slide 6).

7. 18-day-old chick: Moderate to severely enlarged spleen and liver with congestion. Notice white cast in the cecum.

8. 5-week-old chick: Three white nodules resembling tumors in the myocardium. Notice also serous atrophy of fat on the heart. Liver is enlarged and mottled red and tan.

9. 6-week-old chick: The heart is misshapen due to the presence of many yellow nodules in the myocardium. Notice also the thickened pericardium. Liver is discolored tan-brown and mottled, due to chronic passive congestion.

10. & 11. Histopathology of heart: Diffuse infiltration of lymphocytes (slide 10) and histiocytes (slide 11).

12. 6-week-old chick: Gizzard with numerous yellow nodules of various sizes in the wall.

13. Histopathology of gizzard: Necrosis of muscular layer with infiltration of heterophils, lymphocytes and few macrophages.

14. 2 to 3 week old chick: Lungs with white to grey nodules due to *S. pullorum*. (Courtesy Dr. M. Peckham)

15. Tibiotarsometatarsal (hock) joint (chick): The joint is enlarged because of exudate which also extends along the tendon sheath (Courtesy Dr. M. Peckham)

16. Chick: White nodules in the pancreas due to *S. pullorum.* (Courtesy Dr. M. Peckham)

17. Adult chicken: Enlarged and mottled white spleen. (Courtesy Dr. M. Peckham)

18. Adult chicken: Opened body cavity exhibiting severe fibrinosuppurative peritonitis.

19. Adult chicken: Ovary with many misshapen nodular gray to yellow follicles. Notice distention of oviduct due to exudate and also exudate on the serosa of the oviduct.

20. Adult chicken: Exposed abdomen with numerous yellow nodular follicles infected with *S. pullorum.*

21. Histopathology of ovule: Granulomatous inflammation surrounding cellular debris and yolk material.

22. Rapid whole blood agglutination test. Antigen only (blue) in one well, positive agglutination in the center well, and negative agglutination (dark red) in another well.

23. Rapid whole serum agglutination test showing positive and negative tests.

24. Tube agglutination test showing positive and negative tests. Notice floccules in the positive tube.

25. Microagglutination test for pullorum disease. This is a 96 well microagglutination test plate. Note positive and negative controls on 10th and 11th columns, respectively. Compare these to the 9 samples at various titers. Samples 2, 5 and 8 are negative. Samples 1, 3, 4, 6, and 7 are positive at 1:320, 1:40, 1:20, 1:80 and 1:20, respectively. Sample 9 is suspicious, and needs to be re-tested.

Note: Because of the similarities of antigens among *S. pullorum*, *S. gallinarum* and *S. enteritidis*, there will be cross-reaction among these three species.