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Pasteurella Antipestifer Infections

Pasteurellosis is a name applied to infections caused by bacteria of the group Pasteurella, a group loosely related and widely distributed. Two of these infections, P. multocida and P. anatipestifer, cause serious disease of major importance in waterfowl.

AVIAN CHOLERA

(Fowl Cholera, Avian Pasteurellosis, Duck Cholera, Goose Septicemia)

Avian cholera is an infectious disease caused by the bacterium Pasteurella multocida. In waterfowl the infection is usually acute, with few signs observed before death. Losses may be high among both domestic and wild waterfowl, and there seems to be little relationship between the size of the flock and the number of birds that die. Exactly how the disease is spread is still unclear; however, the spread from sick, dead, or recovered carrier birds directly or indirectly by ingestion or by inhalation is likely. Pond water may remain infectious for days and even weeks, and decaying carcasses may remain infectious even longer.

The disease causing capability and harmfulness of this serious infection is complex and variable depending upon the strain of P. multocida, the susceptibility of birds, and the condition of contact between the two. Nevertheless, cholera should be the first disease suspected in waterfowl. The infection can cause localized abscesses in man.

Signs

Visibly sick waterfowl are few in numbers, and signs other than those of the death struggle are seldom seen. More often, birds in good condition are found dead at either the morning or evening feeding. Wild waterfowl are found on nests or nesting cover, some in remarkably uniform sitting position—ducks with heads in an upright position and geese with head and neck extended over the back. Carcasses may be found floating on the water or lying along the shore. Most of the remaining birds are observed in normal flight or loafing attitude.

Very few birds are seen sick an hour or two before they die. Those that are seen may show a variety of signs, few of which are striking. Individuals may move a little slower with uncertain gait and separate from the flock. They may burrow their head in the dirt, twisting it around, and they may have diarrhea and die in a day or two. Wild waterfowl may not try to escape, but instead simply huddle or waddle away. They may appear paralyzed but too weak to hold up their heads. Others have equilibrium difficulties, are unable to get off the water, swim in small circles, fly in peculiar patterns, tumble in flight, or fold their wings well above water and crash land.

Control*

Pasteurella multocida dies readily outside the host. The principles of control in their simplest terms are: depopulate, clean and disinfect the premises, and restock after three months. This is rarely practical; nevertheless, circumstantial evidence indicates value in taking certain measures.

Every means possible should be taken to limit transmission of the disease. Carcasses should be collected frequently. Feeders and waterers can be modified to discourage their use by free flying birds like crows and gulls.

Sanitation measures to eliminate or slow down the infective agent should be taken immediately. Remove litter and disinfect walls, roosts, nests, feeders, and waterers. Under some circumstances it is practical to drain ponds and let the natural purification of sun and air take effect. Contaminated yards and other land can be plowed or cultivated.

Treatment has met with varying success. Outbreaks of avian cholera usually occur in populations of stressed birds. Birds incubating eggs eat little and may be more susceptible at this time. Outbreaks among wild waterfowl suggest accumulated stresses. These include crowding, strong winds, rapid drop in outdoor temperature, low water levels, limited open water—factors that contribute to high densities (accompanied by contaminated soil, food, and water) and physiologically stressed birds. While not always curative, treatment can tide birds over these stressful times until recovery.

Chlortetracycline is approved at 200-400 g/ton of feed for no longer than 21 days in the control and treatment of avian cholera in ducks. The higher levels are indicated for treatment. Injectable oxytetracycline at 50 mg per goose has been used, as has tetracycline feeding at 500 g/ton of feed. Sulfonamides are used as a treatment; however, there is some concern for their possible toxic effects. Sulfamethazine at approximately 1:1000 dilution (3.75-4 g/gal. of drinking water) is used for two to three days, discontinued for two to three days, and then repeated; or it is used for six continuous days at a lower level. Sulfaquinoxaline (.025% to .05%) is also used in drinking water. Water soluble erythromycin (25 g erythromycin) at 1 lb to 50 gallons of drinking water for five days has been reported as effective. Penicillin is rarely useful in this disease.

Early treatment of the flock or preclinical treatment of susceptible birds may be a deterrent to the disease. Immunization may play a similar role.

Vaccination for the prevention of avian cholera has been practiced widely for many years. Although reasonably effective bacterins or vaccines (avirulent live culture, avian isolate) have been developed, there is considerable room for improvement. Autogenous bacterins are favored. Pasteurella multocida bacterins, avian isolates, types 1, 3, and 4 are available commercially. Types 1, 2, 3, 5, and 7 are associated with waterfowl. Revaccination may be indicated.

Post-mortem Findings

Evidence of septicemia is found, with hemorrhages possible on most serous surfaces and intestinal mucosa. Most notable are lesions of the heart, liver, and digestive tract. These appear as petechial hemorrhages in the epicardium and myocardium and coronary fat. Parenchymatous hepatitis and focal necrosis on the liver are described as pinpoint to pinhead white, gray, yellow specks. The digestive tract may be full of food in all stages of digestion. Hemorrhages on the gizzard and hemorrhage varying from slight to frank hemorrhagic enteritis may be seen. As the infection becomes more chronic, a catarrhal exudate may be found in

nasal passages and trachea as well as the occurrence of valvular endocarditis and otitis media.

Diagnosis

A presumptive diagnosis is based on the history, signs, and lesions. It can be partially confirmed by blood or liver impression smears that reveal bipolar cocco-bacilli. Isolation and identification of P. multocida are needed for a definitive diagnosis. Bone marrow and heart blood are preferred sources of samples.

PASTEURELLA ANATIPESTIFER INFECTIONS

(Infectious serositis, new duck disease, anatipestifer syndrome, duck septicemia, goose influenza)

Pasteurella anatipestifer infections have been reported in duck, goose, swan, turkey, and pheasant. The disease in ducklings is known as infectious serositis, a very serious disease with high death losses on commercial duck farms. A disease known as goose influenza caused by a Pasteurella organism that cannot be differentiated from P. anatipestifer on the basis of reported characteristics is probably the same disease. The remaining terms listed above have also been applied to conditions associated with colibacillosis.

The infection in waterfowl is characterized by fibrin deposits on serosal surfaces with pericarditis and perihepatitis the prominent pathologic changes. The young—ducklings, goslings, young adult wild ducks, and juvenile swans and geese—are apparently more susceptible than adult birds that may harbor the infectious organism while in good health.

Precisely how the disease is spread is not known. Experiments provide evidence that the infection may normally enter through wounds, scratches, feather follicules, or punctures of the skin. Contact exposure is not usual. Inhalation or mouth and eye instillation is almost certain to result in infection.

Signs

The reported signs in ducks are quite variable. Death may take place in the absence of clinical signs. More frequently mild coughing, sneezing, and depression are seen at the onset. Soiled vent feathers accompanied by diarrhea, described variously as yellow or white to whitish green to green; watery to mucus like discharges from the eyes and nose; and incoordination follow. Ducks may show signs of other nervous disorders. Slow tremor of the head, twisting of the neck, lying on the back, paddling convulsively with the legs may precede exhaustion and death. Survivors may have swollen hock joints, limp, and lose weight.

Wild ducks may show abnormal swimming posture, list to one side, and tend to swim in a circle. They may drown when the infection progresses beyond the stage where they can carry their flopping heads above water.

Goslings may not show clinical signs before death. The infection has been diagnosed in flocks where unexplained moderately higher than expected death losses were taking place. Others may show depressions, incoordination, and diarrhea before death. Wild goslings and juveniles generally found under unfavorable conditions may show signs of greater distress such as paralysis, nervous movements, twisted neck, and trembling. Death losses are greater in goslings, while juveniles may end up with swelling around the legs and feet.

Weakness has been noted in juvenile swans. Apparently sick birds have not been seen often. Diagnoses have been made on the basis of isolating the organism from well fleshed carcasses showing typical lesions of P. anatipestifer infection. Dead birds were found in crowded conditions during fall migration.

Control*

Until specific preventive measures are devised, the usual sanitary procedures must be relied upon. Some believe that P. anatipestifer is a secondary invader and the stress and concurrent diseases play an equally important part in precipitating the disease. It follows that the many hygienic practices that contribute to general health are extremely important. Recovered ducks are resistant to the disease; however, the infection tends to spread to younger and younger age groups on a farm, and the disease hangs on at a farm for the rest of the season. Treatment is employed as a stopgap until more extensive measures can be taken.

Several successful treatments have been reported: sulfaquinoxaline (.025% -.05%) in drinking water, sulfamethazine (.08% -0.1%) in drinking water. Adequate amounts of vitamin K should be provided when used at higher levels or for prolonged periods. Penicillin (12,000 units) and dihydrostreptomycin (15 mg) intramuscular reportedly give results superior to oxytetracycline (15 mg) given subcutaneously. Chlortetracycline has been given in all mash 500 g per 909 kg (ton) for seven days. Sulfachloropyrazine (.06%) has been given intermittently in feed. In wild waterfowl chloramphenicol in the drinking water at approximately 1.25 mg/oz has been used.

Experimentally when novobiocin was fed three days prior to challenge at adequate concentrations (.0193% - .0386%), death losses were markedly reduced. Lincomycin fed at levels of .011% - .022% was also very effective. A single dose of lincomycin-spectinomycin, penicillin-streptomycin, penicillin, oxytetracycline, and spectinomycin greatly reduced death losses when given parenterally five to six hours after infection. Drugs injected 24 hours after infection were not effective.

A trivalent bacterin containing an inactivated cell suspension of serotypes 1, 2, and 5 shows some promise when given to ducklings as early as day one. Multiple inoculations were indicated.

Post-mortem Findings

Post-mortem findings present a picture of generalized fibrinous inflammation of the pericardial, pleural, peritoneal, and meningeal serous surfaces. The inflammation shows little tendency to extend into the underlying parenchymatous organs. Lesions vary in extent and in type of inflammatory cells present.

Experimentally infected ducks may show no gross lesions in the acute phase. In the chronic phase the principal lesions are pericarditis, airsacculitis, and perihepatitis.

Field cases show more extensive involvement. Early inflammatory exudate may be seen as a clear thin film. In the chronic phase exudates tend to be drier, firmer, sometimes adherent, and organized into partial or complete membranes or casts. The picture is typically one of splenomegaly, airsacculitis, perihepatitis, salpingitis, and fibrinous cerebrospinal meningitis in birds showing signs of central nervous system involvement. Concurrent infection in field cases is not unusual, and other lesions have been noted. They include: tracheites, enteritis, edema, abscesses, myocarditis, necrosis, peritonitis, hemorrhages, pneumonitis, congestion, and sinusitis.

Diagnosis

A definitive diagnosis is based on clinical observations, post-mortem findings and isolation of P. anatipestifer. Heart blood, brain, air sacs, trachea, and liver are preferred sources of samples.

**Drugs or biologics listed represent favorable experiences reported by others and are not recommendations. The user is fully responsible for the consequences of their use when used other than indicated on the product label.*

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