Pasteurellosis in backyard poultry and other birds

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Case 1

An investigation was requested to determine the cause of sudden mortalities in a group of 3 week old goslings bought from a local market. Within 4 days of purchase 2 of 12 birds were found very weak. Both died within 12 hours. One comatose and 2 dead goslings were submitted for necropsy examination. They were in good physical condition. Haematology results on blood collected from the live bird demonstrated a marked leucocytosis (37.3×10^9 cells/mL) due to a heterophilia (30.4×10^9 heterophils/mL) with a left shift.

Necropsy examination demonstrated multifocal abscess throughout the lungs, mild hepatomegaly and a mild thickening of the airsacs. Histological examination demonstrated a severe multifocal necrosuppurative airsacculitis and pneumonia with pulmonary abscessation. A heavy growth of *Pasteurella multocida* was cultured from the lungs and liver. Further cases did not occur in remaining birds which were treated with doxycycline (5 g/L drinking water).

Case 2

A backyard poultry flock of 29 Rhode Island red chickens consisting of 27 hens and 2 roosters all 1 year of age with a 3 month history of illness and deaths was investigated. Affected birds reportedly developed swollen eyes, lethargy, and inappetance and death within 2 days of the onset of clinical signs.

One affected hen was submitted for necropsy. Haematology results were normal. Necropsy demonstrated pale, swollen periorbital sinuses, nares and beak and a caseous exudate from the choana. The larynx, trachea, lungs, airsacs and other visceral organs appeared normal. A moderate burden of *Heterakis gallinarum* was present in the caecae.

Histological examination of the nasal cavities demonstrated a severe necrotising suppurative rhinitis and osteomyelitis.

Culture of the nasal cavities yielded a heavy growth of *Pasteurella* spp. and a light growth of *S epidermidis.* The *Pasteurella* spp were susceptible to penicillin, erythromycin, tetracycline, and trimethoprim-sulphonamide. Culture of the lungs also yielded a *Mycoplasma* spp, although the significance of this isolation was questionable since significant histological lesions of pulmonary mycoplasmosis were not present.

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Discussion

The family Pasteurellaceae contains Gram-negative, facultatively anaerobic and fermentative bacteria of the genera Pasteurella, Haemophilus, and Actinobacillus. Approximately 20 different species of the genus Pasteurella have been identified using phenotypic and genetic analyses and *Pasteurella multocida* is the type species (Confer 1993). Fowl cholera in chickens and turkeys is caused by various serotypes of *P. multocida* serogroup A (Diallo *et al* 1995) and is characterised by acute septicemia and pneumonia or chronic fibrinopurulent inflammation of various tissues. Fowl cholera has been studied for more than 200 years. The disease is characterised by high morbidity and mortality in the acute form and respiratory disease and malaise in the chronic form (Rhoades.& Rimler, 1991).

Other *Pasteurellacea* spp such as *P haemolytica*, *P gallinarum* and *P pneumotropica* can cause significant acute and chronic disease in birds and the diseases that these species cause should not be called cholera. The organism known as *P anatipestifer* has been reclassified as *Riemerella anatipestifer* and belongs to the family Flavobacteriaceae (Subramaniam *et al* 1997). This has been done no doubt by self conscious bacteriologists who feel the need to justify their existence in the scientific community.

Epidemiology

P. multocida is a member of the normal flora in the upper respiratory tract of many mammals and birds. It causes sporadic or epidemic diseases among different animal species but is particularly a pathogen of the respiratory tract. Although, outbreaks which are expressed mainly as primary skin or oviduct infections have been recognised. All avian species are susceptible, young adult poultry and breeders coming into lay, turkeys, chickens, ducks, geese are most commonly affected. Outbreaks have been reported in many different species of wild birds including waterfowl and penguins (Parmelee *et al* 1979; Hirsh *et al* 1990; de Lisle *et al* 1990; Wilson *et al* 1995). Scavenger birds such as crows and gulls are commonly affected. Deaths in raptors are not common and probably occur at a similar prevalence to other infectious diseases such as aspergillosis (Work & Hale 1996; Franson *et al* 1996; Morishita *et al* 1996).

Transmission of infection is predominantly by bird to bird contact and ingestion of contaminated food or water. Wild birds and rodents can enhance transmission. Infection can come from newly introduced birds, free-flying birds, infected premises, predators, or rodents. In an infected flock birds become infected by cannibalising dead or weakened birds and by ingesting oculo-nasal discharges, or contaminated food and water. Spread from flock to flock can be attributed to movement of personnel, contaminated equipment, wild birds and predators. Artificial insemination in turkeys can introduce the infection.

Recovered birds can remain carriers. The organism persists for many months in the carcasses of dead birds (Beveridge & Hart, 1985). The capsule of *P. multocida* along with expression of bacterial toxins (dermonecrotic toxin, hyaluronidase, neuraminidase and proteases) are probably responsible for virulence.

P. multocida infections in psittacine birds

Several case reports have documented the pathogenicity of *P. multocida* for psittacine birds. However, the prevalence of infection of psittacine birds has not been investigated in detail. In

a recent study by Morishita *et al* (1996) *P. multocida* was not was not isolated from the pharynx, choana, or cloaca of 328 psittacine birds (253 clinically healthy and 75 clinically ill). However, *P. multocida* was isolated from five dead birds submitted for necropsy and some of these were septicaemias due to cat bight wounds. All isolates were susceptible to penicillin G, sulfasoxazole, gentamicin, erythromycin, tetracycline, and trimethoprim-sulfamethoxazole but resistant to streptomycin.

P. multocida infections in humans

P. multocida is responsible for the majority of human Pasteurella infections (Avril & Donnio 1995). The most common human infection with *P. multocida* is a local cellulitis following dog or cat bites and scratches. Serious local complications are sometimes responsible for prolonged disability. The respiratory tract is the second human source of *P. multocida* isolates. The frequency of recovery of *P. multocida* from the oropharynx of apparently healthy pig breeders suggests that respiratory pasteurellosis may be an occupational disease. There are no confirmed cases of human infection with avian strains of *P. multocida*. Although, a single case of valvular endocarditis due to *P gallinarum* infection has been reported.

Clinical presentation:

As in the first case described above affected flocks may experience an acute outbreak of sudden deaths with few clinical signs or gross lesions. In sub-acute forms of the disease, affected birds may appear depressed and have a serous oculo-nasal discharge. They lose weight, become lame and develop a "rattle" due to exudates in the trachea. In the chronic form the disease may be localised to the wattles and comb and there may be involvement of the middle ear.

In the peracute disease there is usually splenomegaly and hepatomegaly, with widespread petechiation in the mucosa of the intestine and serosal surfaces of the heart and abdominal organs. There may be focal hepatic necrosis. In subacute disease there is often a fibrinous pericarditis, airsacculitis and caseous plugs in the lungs, airsacs, sinuses and wattles.

Severe infections localised to the facial skin have also been documented in turkeys (Jeffrey *et al* 1993). Affected turkeys appeared lethargic, anorexic or died suddenly. The heads of affected birds appeared cyanotic and swollen. Affected flocks ranged in age from 6 to 18 weeks and included both toms and hens. Histologically, the facial cellulitis was characterized by extensive fibrinonecrotic inflammation of the deep dermis with heterophilic perivasculitis and thrombosis.

Wild birds and waterfowl that die acutely often have substantial amounts of fat and the upper GIT usually contains recently digested food. In ducks these signs are similar to those seen in duck plague (duck virus enteritis) which is an exotic disease. Therefore a laboratory diagnosis is necessary. The lower GIT can contain a tenacious yellow fluid that is heavily laden with *Pasteurella* organisms.

Birds with chronic Pasteurellosis, as in the second case presented above, may have a severe necrosuppurative rhinitis, sinusitis and glossitis. Similar suppurative foci in the lungs, pericardium and airsacs may be present in the lower respiratory.

Diagnosis

Cytological examination of nasal exudates can be achieved using choanal swabs and/or nasal flushes using warm saline. The presence of a large number of relatively small bipolar Gram-negative coccobacilli in Gram stained smears of nasal exudates is suggestive of Pasteurellosis.

Nasal flushes or swabs of the choana are appropriate samples for bacteriological culture providing culture results are interpreted in relation to the cytology findings.

In the acute forms of Pasteurellosis, as in the first case presented above, haematology results may indicate a severe heterophilia with a left shift. Giemsa and or Gram-stained blood smears may also demonstrate a septicaemia. However, chronic cases may have only mild elevations in heterophil, monocyte and lymphocyte cell counts or the haematology results be within normal limits.

From necropsies whole lung, liver or heart submitted as whole sections and not as swabs are the most appropriate samples for culture. Culture of *Pasteurella* spp usually takes a minimum of 3-5 days. The organism can be fastidious, most *Pasteurella* spp are sensitive to cooling and therefore, where possible, swabs of the respiratory cavities should not be chilled below 10˚C. The organism may survive better in swabs transported at room temperature. Avian strains of most *Pasteurella* spp can be isolated on blood agar plates at 37-41˚C. Some strains may prefer an atmosphere enriched with CO_2.

In backyard poultry flocks other differential diagnosis which should be included are coryza (*Haemophilus paragallinarum*), colibacillosis, aspergillosis, mycoplasmosis, chlamydiosis, erysipelas and turkey rhinotracheitis. Avian influenza and Newcastle disease should also be ruled out.

Treatment and control

Aggressive antimicrobial therapy is important for individual affected birds and flocks that are affected with the acute disease. Tetracycline 400-600 ppm in feed or sulphonamides (250 ppm in feed) are probably the most appropriate medications for controlling the disease in poultry but sulphonamides can cause a serious drop in egg hatchability. Waltham and Horne (1993) have demonstrated resistance of *P. multocida* isolates to sulphonamide and resistance to streptomycin, trimethoprim, lincomycin was demonstrated in one study of 45 strains of *P. multocida* isolated from Australian chickens (Diallo *et al* 1995). For backyard poultry, individual bird treatment with oxytetracycline (Terramycin® LA) at a dose rate of 20 mg/kg can be effective. Penicillin along with sulphonamide are probably the drugs of choice in caged birds.

Penicillin is considered to be the drug of choice for Pasteurella infections in humans (Avril & Donnio 1995). Tetracycline is efficient for bites but has no bactericidal effect. Oxacillin, first-generation cephalosporins, macrolides and aminoglycosides have poor activities. In the case of β-lactamase producing strains a bactericidal effect could be achieved with fluoroquinolones or third generation cephalosporins.

Control

Elimination of rodents, and improved hygeine and quarantine measures are important for preventing and controlling outbreaks of Pasteurellosis. Most disinfectants are effective. Wild-

bird proof cages and effective quarantine procedures should be implemented. Decreased overcrowding and rotational grazing of paddocks for geese and ducks and improved drainage of waterfowl housing and sanitation of feeding and watering utensils should also be considered. All-in all out rearing of waterfowl is also warranted.

In 1880 Louis Pasteur used *P. multocida* in his classic experiments to demonstrate attenuation of bacteria for producing immunity. However, in-field use of attenuated *P. multocida* vaccines did not (and have not) proven practical because uniform attenuation could not be obtained and heavy losses sometimes occurred in vaccinated flocks (Rhoades & Rimler 1991). Killed vaccines on the other hand do not suffer from this risk. A bacterin vaccination (Fowl Cholera Vaccine, Arthur Webster Pty. Ltd.), which is given by subcutaneous injection at 12 weeks of age followed by a booster 4 weeks later, is currently available in Australia.

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