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INFECTIOUS DISEASES

NEWCASTLE DISEASE

A multistrain disease with 3 domestic forms and an exotic form - viscerotropic velogenic newcastle disease (VVND).

Etiological agent - Paramyxovirus

Transmission - Aerosols from respiratory secretions, fecal contamination of food and water supplies, direct contact with infected birds or fomites.

Incubation period - 5 to 16 days in exotic birds.

VVND is highly contagious. Chickens liberate virus from the respiratory tract prior to showing clinical signs. Spread is rapid in a susceptible flock. VVND was eradicated from the U.S. at great expense. The current quarantine program was initiated in 1974 to prevent the re-entry of VVND into domestic poultry flocks. Birds are tested in quarantine for VVND by virus isolation from dead birds and from cloacal swabs of live birds.

Clinical signs - range from acute death to asymptomatic birds. Signs include: depression, sneezing, dyspnea, bright yellow green diarrhea, weight loss, and anorexia, ataxia, head wobbling, opistotonus. Neurological signs are seen in birds which survive the acute stage of the disease. Unilateral and bilateral wing and leg paralysis may be seen as well as ataxia, head wobbling and opistotonus.

Species susceptibility - Cockatoos and cockatiels are highly susceptible species. ND in these birds results in high morbidity, high mortality and many acute deaths. Species which are highly susceptible but have lower mortality include amazons and conures. In these birds neurological signs are more likely to be seen due to the more prolonged course of the disease. Species which are relatively resistant include macaws, lorys, african grey parrots, finches and canaries.

Gross post mortem lesions - Lesions in psittacines are not the same as those observed in chickens. Lesions commonly observed include: petechia on serosal surfaces of all viscera, petechia in the lumen of the trachea, hepatomegaly, splenomegaly and airsaculitis with petechia on airsacs.

Diagnosis cannot be made by histopathologic exam; it must be based on virus isolation by USDA approved lab.

Contact your local USDA office any time you suspect VVND.

Vaccination is prohibited in birds coming into the U.S. Vaccination does not eliminate the carrier state and would hinder the detection of virus during quarantine.

AVIAN HERPES VIRUSES

Many avian herpes viruses have been described in birds, including parrots, hawks, falcons, owls, pigeons and poultry.

Pachecos parrot disease

Transmission - fecal contamination of food and water, direct contact with infected birds, fomites.

Incubation period - 5 days to 2 weeks. Incubation period is dose and bird size related. Longer incubation periods have been reported but not verified.

Clinical signs - Acute death (some birds may be sick for a few days to a few hours prior to death), vomiting, bright yellow diarrhea, icterus in macaws; neurological signs may be seen terminally.

Gross lesions - Hepatomegaly, "fatty liver" appearance with streaks and petechia. Splenomegaly, friable spleen.

Histological lesions - Intranuclear eosinophilic inclusion bodies in liver or spleen. May also be found in intestines, lungs, bone marrow, kidneys.

Virus is shed from oral cavity and cloaca.

Carriers - Nanday conure, patagonian conure, many other species suspected.

Very susceptible - Macaws, amazons, parakeets, some conures, cockatoos, cockatiels. Macaws have been tested and observed extensively clinically and are not believed to be carriers.

Vaccination - Not available commercially. At least two serotypes have been identified and multistrain vaccines may be necessary.

Control in an outbreak - Spread out birds, careful disinfection of food and water bowls, cleanliness, chlorhexidine disinfectant (Nolvasan, Virosan) in drinking water. Bowls should hang high in the cage or be covered to prevent fecal contamination.

Duration of immunity after recovery is approximately 6 months.

Pigeon herpes

Adult birds are asymptomatic carriers and infect the squabs by feeding. Young birds are usually resistant after 16 weeks of age.

For more information on Avian Herpes viruses see Kirks Current Veterinary Therapy VIII.

CHLAMYDIOSIS

(Psittacosis - Ornithosis)

Etiological agent - Chlamydia psittaci

Chlamydia affect all species of birds as well as some mammals and man. Chlamydia have world wide distribution. Psittacines have long been incriminated in the spread of psittacosis to humans.

U.S. Public Health Service regulations require the use of Chlorotetracycline (CTC) in the feed of all imported psittacines during the 30 day quarantine period for VVND. This treatment is poorly enforced, due to a lack of interest and funding by the Public Health Service. Currently the USDA is considering enforcement of the feeding of CTC. Psittacosis is not limited to newly imported birds. It is also found in psittacine breeding collections, especially those of budgerigars and cockatiels.

Transmission - The primary means of transmission is by inhalation of nasal exudates, expired aerosol droplets and fecal dust. Chlamydia concentrate in the lung, kidney and cloaca.

Incubation period - 5 to 100 days

Clinical signs are varied and non-specific.

Peracute form - sudden death of an apparently healthy bird. May be preceded by a profuse yellow green diarrhea.

Acute form - Begins with depression, profuse yellow green diarrhea and anorexia, followed by the development of respiratory disease. Ocular and nasal discharge, sinusitis, airsaculitis, pneumonia, severe weight loss and emaciation are seen as the disease progresses.

Chronic form - Chronic nasal discharge which will cake on the beak and often occlude the nostrils, sinusitis, airsaculitis, dyspnea, subnormal weight, depression, failure to thrive, diarrhea, and wasting.

An asymptomatic carrier state also exists.

Diagnosis - clinical signs, history of possible exposure, elevated liver enzymes, especially SGOT. Radiographic lesions include hepatomegaly, splenomegaly, airsaculitis.

Indirect Completement Fixation test - 1:16 is considered suspicious while 1:32 and above is considered positive. A rising titer will confirm active disease. Titers may remain elevated for an extended period of time after treatment.

Chlamydia may be isolated from the feces or from post mortem specimens. Isolation in eggs requires two weeks, while tissue culture is often faster. Chlamydia may also be cultivated by intracerebral innoculation into suckling mice. They are demonstrated by histopathologic examination.

Post mortem lesions - Hepatomegaly, splenomegaly, friable spleen, fibrous pericarditis, fibrous deposits on the surface of the liver, airsaculitis. Secondary bacterial infections are common and result in deposits of large masses of caseous pus in the airsacs. Impression smears of liver, spleen and airsacs may demonstrate elementary bodies. These bodies are intracellular colonies of chlamydia which can be seen when stained by Machieveilla, Giemsa or Jeminez staining techniques. Interpretation of the slides requires some expertise as the elementary bodies are very small.

Treatment - CTC is most commonly used due to proven efficacy and low cost. Birds should receive CTC in the food continuously for 30 to 45 days. If properly dosed, this treatment procedure has a high rate of success; however, some treatment failure may occur. This may be due to inadequate intake or absorption by some individuals. Some birds with severe lesions may not be cleared due to sequestration of organisms in fibrous deposits on the heart and liver.

Recovered birds are susceptible to reinfection after the end of treatment. Birds must be transfered to a clean environment or all dust and fecal matter must be cleaned from existing caging prior to discontinuation of CTC.

Vaccination is not practical due to the poor immunological response.

Treatment - Addition of CTC to water supplies should not be used as the sole source of tetracycline due to rapid breakdown. If CTC is used in water it should be changed three times daily.

Commercially available pelleted feeds are available for psittacines. These diets are balanced and supply a controlled level of CTC (1%) as well as calcium. Excess calcium in the diet interferes with the absorption of CTC.

CTC impregnated shelled millet seeds (0.5%) are available for the treatment of small psittacine birds. CTC capsules may be added to a nectar-food mix for lorys and lorikeets at the rate of 250 mg/pint of food mixture. SF66 (soybean meal base CTC) can be mixed with a cooked corn and rice mash at the rate of 1 lb SF66/20 lb mash.

Positive psittacosis cases should be reported to state public health officials in your state.

AVIAN POX VIRUS INFECTION

Etiology - Pox virus - a large DNA virus.
Five subgroups of avian pox virus: Canary, Fowl, Turkey, Pigeon and Parrot.

Transmission - Direct contact with infected birds or fomites; insects may act as mechanical vectors.

Canary pox - Two forms exist: (1) Acute viral septicemia or respiratory infection resulting in death within 1 to 3 days; (2) Chronic infection with proliferative skin lesions around the mouth, eyes,

nostrils, feet and inside mouth. Canary pox results in 90 - 100% mortality. Canary pox has been controlled experimentally by vaccination.

Pigeon pox - A very debilitating disease causing severe lesions around the eyes and beak. Some strains produce high mortality and survivors may be severely disfigured. Pox is common in feral pigeons as well as domestic pigeon lofts. Commercial vaccines are very effective.

Parrot pox - Common in some species of psittacines imported from Central and South America as well as lovebirds.

Two forms in psittacines: (1) Dry form which results in harmless dry scabs forming on facial areas; and (2) Wet pox which results in severe diptheritic oral lesions with concurrent systemic disease.

Commonly affected species - Amazons, pionus, lovebirds, australian parakeets, rosellas.
Relatively resistant species include - Eclectus parrots, cockatoos, cockatiels, african parrots and lorys.

Incubation period - Pharyngeal lesions appear at 7 - 10 days. Eye lesions appear at 10 - 14 days.

Clinical signs - Eye lesions begin with swelling of the lids, pasting, fluid accumulation with white flecks and white caseous masses. Scab formation follows and these lesions may progress to errosion of the cornea, corneal rupture, and scarring of the lids and cornea.

Pharyngeal lesions begin with thickening and paleness of the papillae on the edge of the choanal cleft and progress to encompass the edges of the cleft. Large white plaque-like lesions will also be found behind the choanal cleft, in the commisure of the beak, on the tongue, and on the glottis. Removal of these lesions will result in bleeding wounds. Caseous plugs may fill the trachea and bronchi. Pneumonia and airsaculitis are commonly found. In some species, scabs form on the cere and the feet. Secondary bacterial and fungal infections are common.

Death is usually due to: (1) Secondary bacterial septicemias; (2) Caseous plugs in the trachea or bronchi resulting in pneumonia or asphixia; (3) Wasting and malnutrition caused by reluctance to eat with painful oral lesions.

Recovery is slow and requires 3-8 weeks of nursing care.

Treatment - Vitamin A is of primary importance during the treatment period and if given to exposed birds prior to the development of clinical signs it will lessen the severity of the disease. Long term antibiotic therapy is necessary to decrease mortality from bacterial secondary invaders. Concurrent use of gentamycin and tylosin has proven very helpful. Eyes are washed daily with a mixture of 1/2 ounce of mercurochrome in 4 ounces of eye wash solution. Washing is followed by the application of chloromycetin ophthalmic ointment. Supportive care should include tubefeeding, warmth, and soft foods.

Residual effects - scarring of the eyes, choana, cere, and nostrils. Chronic respiratory disease may result. Aspergillosis is common following pox virus infection.

Prevention - Vitamin A, vector control, adequate quarantine of all new arrivals.

There is no evidence that a carrier state exists in psittacine birds.

Vaccination - Commercial pigeon and fowl pox vaccines are not effective in parrots.

TUBERCULOSIS

Etiological agent - Mycobacterium avium

Tuberculosis is a problem when it is endemic in an aviary or zoo. Otherwise it is very uncommon. The course is slow and insidious.

Transmission - Aerosols, direct contact and ingestion.

Diagnosis - Intradermal testing is unsatisfactory. Definitive diagnosis is based on histopathology. Culture techniques for mycobacterium are very difficult and take extended periods of time.

Treatment is neither effective nor recommended.

INFLUENZA

Etiological agent - Influenza virus (Orthomyxovirus)

USDA testing procedures for birds entering the U.S. are designed to diagnose influenza as well as Newcastle disease. Hemagluttinating viruses are often found in passerine birds, especially finches from Africa; however, they are uncommon in psittacines. Most of these viruses have been typed as turkey Paramyxo viruses or Yucapa viruses which are harmless to poultry. It has not been determined if these viruses are responsible for mortality in imported finches.

MAREKS DISEASE

Etiological agent - herpes virus

Although lymphoid leukosis has been diagnosed in pet birds, no link has been made to the herpes virus responsible for Mareks disease in chickens.

BUDGIE FLEDGLING DISEASE

Etiological agent - Papovavirus

Produces high mortality in young budgerigars at 1 to 3 weeks of age. Produces inclusion bodies in kidneys, preen gland, skin, feather follicles and other organs.

Clinical signs - diarrhea, dehydration, swollen abdomen and death in 1-2 days.

Diagnosis - Histopath, viral isolation, possibly serology.

An experimental vaccine is showing great promise in the control of the disease. No other species have been found to be affected.

SALMONELLOSIS

Etiological agents - many species of salmonella including: S. typhimurium, S. enteritidis, S. oranienburg, S. anatum, S. thomson and S. paratyphi.

Any species of bird can be affected. Salmonella are very common in poultry.

Transmission - Fecal contamination of food and water supplies by infected birds, rodents, or human carriers or by direct contact with infected birds. In some speices the young may become infected by fecal contamination on egg shells or by direct transmission through the egg from infected ovaries. Salmonella are very resistant in the environment and will live for an extended period of time on wooden surfaces or dirt aviary floors allowing infection by placing birds in contaminated cages.

Incubation period - deaths usually begin within a few days to two weeks after ingestion of the bacterium.

Clinical signs - Vary in severity from asymptomatic carriers to acute death, depending on the virulence of the strain and the avian species involved. Mortality may reach 90 to 100%. Spread through a susceptible flock is rapid. Other clinical signs include depression, anorexia, shivering, rapid weight loss, watery yellow green droppings which may in some cases by bloody. Neurological signs may be seen terminally. Affected pigeons may develop arthritis. Chronic enteric disease may be seen in some birds producing an intermittent diarrhea. In breeding flocks, asymptomatic carrier adults may infect young birds and eggs causing dead in shell or weak fading chicks.

Diagnosis - Culture of feces. Serological tests which are widely utilized in poultry are not useful in pet birds due to the specificity of the test for the serotype of salmonella involved.

Post Mortem lesions - Hepatomegaly, splenomegaly, enteritis. Salmonella may be cultured from the liver, heart, blood or intestinal tract. Salmonella may be found in almost any body tissue.

Treatment and control - It is difficult if not impossible to ever eliminate salmonella from a bird once it has become infected. In the carrier state the bacterium is not shed in the feces; however,

stress may bring on the recurrence of enteric disease or acute death due to septicemia at which time the bird will be infectious.

Antibiotics are effective in slowing the spread of salmonella in a susceptible flock and decreasing mortality and morbidity. Antibiotic susceptibility testing is essential due to increasing drug resistance. Chloromycetin is the drug of choice due to its excellent properties of tissue penetration. Other antibiotics which are helpful include nitrofurazone, sulfonamines (sulfadimidine and sulfadiazine), tetracyclines, trimethoprim - sulfa, spectimomycin and gentamycin.

Thorough cleaning of the environment is essential to control. The numbers of infective organisms in soil may be reduced by acidification. All wood perches and nest boxes should be destroyed. Wooden surfaces which cannot be removed should be whitewashed. Care must be taken to avoid contamination of feedstuffs and water supplies.

ENTERIC BACTERIA

Escherichia coli, Klebsiella, Enterobacter and related organisms are the object of continual debate as to their importance in avian disease. Gram negative bacteria should not be found as a component of the intestinal flora of seed eating birds; however, it is frequently found in fecal cultures of healthy birds.

Coliform bacteria are usually considered secondary invaders or opportunists but some virulent strains may act as primary pathogens. In the most severe infections death may be acute, due to bacterial septicemia from an intestinal infection. Enteric bacteria can be found in almost any tissue or organ system causing a variety of clinical signs. Included are: Pneumonia, airsaculitis, salpingitis with resulting egg binding or infertility, septic arthritis, peritonitis, sinusitis and enteritis. In cases of chronic enteric infections an intermittent foul smelling diarrhea is common.

Diagnosis is based on culture of feces, pharyngeal swabs and any available body fluid. Blood culture would be ineffective except in cases of septicemia.

Antibiotic susceptibility testing is vital when treating enteric bacteria, especially E. coli, due to the rapid development of antibiotic resistance.

PASTEURELLOSIS

Pasturella are often incriminated in septicemic deaths in poultry and water fowl. The incidence in pet birds however appears to be very low. Both an acute and chronic form exists in pet birds.

Diagnosis - Culture of blood or tissues of a dead bird.

Sulphonamides are the most effective drugs for therapy.

STREPTOCOCCUS INFECTIONS

Streptococcus is relatively unimportant in birds. Some problems which may be associated with strep include wound infections (especially bumblefoot) and occasionally septicemias.

STAPHYLOCOCCUS INFECTION

Like strep, staph is of relatively minor importance in pet birds. It is commonly found in and contributory to bumblefoot and may at times be associated with bacterial arthritis.

PSEUDOMONAS

Pseudomonas acts much as the enteric bacteria in birds. They are usually secondary invaders or opportunists. Pseudomonas can cause acute deaths especially when associated with pneumonias. They commonly invade the respiratory system causing chronic sinusitis, rhinitis, and airsaculitis. Enteric infections are less frequently encountered and more responsive to therapy than respiratory infections. Flock outbreaks are not frequently encountered with pseudomonas infections, but can occur.

Both P. aeruginosa and members of the fluorescent group of pseudomonas are found in birds.

Treatment - Combined therapy with gentamycin and carbenicillin has proven most helpful in treating pseudomonas infections. I have had good luck in treating upper respiratory infections by giving one drug IM while administering the other by intratracheal injection or nasal flush. Amakacin may be used in infections which are resistant to gentamycin. Antibiotic susceptibility testing should be used to confirm the choice of drugs; however, therapy should not be delayed in cases of serious infection.

MYCOPLASMA

Mycoplasma have been associated with chronic respiratory disease in poultry for many years and are probably involved in many cases of chronic respiratory disease in pet birds. However, due to their very specialized growth requirements, they are seldom isolated. Mycoplasma have been confirmed as the causative agent in a respiratory disease in a flock of macaws and in a flock of amazons. These organisms, however, were closely related to poultry pathogens with which poultry diagnostic laboratories have had experience. Many more species may exist which have more specialized growth requirements.

Transmission - Direct contact, aerosols and possibly through the egg.

Clinical signs - the disease is essentially an inflammation of the mucous membranes resulting in congestion, swelling and discharges

which may at first be watery and later become mucoid or caseous. Morbidity is usually high while mortality is low. Frequently observed signs include nasal discharge, ocular discharge, depression, raspy respiratory sounds, airsac clicking, partial or complete closure of one or both eyes. The disease will often become endemic in a population of birds.

Treatment - Tylosin, erythromycin, lincomycin-spectinomycin. Prolonged therapy may be required. Tylan powder may be mixed 1:10 with water and sprayed into the eyes to treat conjunctivitis.

HAEMOPHILIS

Haemophilus gallinarum, the causative agent of coryza in chickens, is another pathogen which is probably more common than it is reported. This is due to the difficulty of growing the organism.

ASPERGILLOSIS

Etiological agent - Aspergillus fumigatus is one of the common green bread molds which is ubiquitous in the environment.

Transmission is by inhalation or ingestion of spores.

Aspergillosis is a chronic debilitating disease which is common in raptors and penguins and occasionally found in parrots and mynahs. Infection can usually be related to excessive stress, concurrent or previous serious illness or immunodeficiency. The incidence of the disease is also related to the number of inspired or ingested spores. Very dusty or damp environments or exposure to moldy foods or bedding may increase the incidence of disease.

Clinical signs - Aspergillosis most commonly affects the lungs and airsacs producing plaques which are often covered with hyphae and spores. Mycotic granulomas are often found in the lungs and if these are opened they will often contain spores. The nares and sinuses and under the eyelids may become filled with caseous material. The main signs encountered are gasping, dyspnea and wheezing. Although mycotic septicemia may cause acute death the disease is most often chronic, progressing for weeks or months.

Diagnosis - Endoscopy, radiography, culture of tracheal swabs, trans tracheal wash , or airsac wash . Serological diagnosis is not reliable.

Treatment - IV amphoteracin B may be used in combination with oral flucytosine. Nebulization or intratracheal injections may be helpful but are of questionable efficacy. One researcher opens airsacs on raptors and swabs out plaques with amphoteracin. Systemic therapy is used in conjunction. Amphoteracin may be given interperitoneally or intravenously. Levamisole may be helpful in returning a depressed immune system to normal. Treatment in most cases is unrewarding due to the advanced state of the infection at the time of diagnosis.

Prevention - cleanliness, good management.

CANDIDIASIS - MONILIASIS

Etiological agent - Candida albicans (some other species may also be involved).

Candidiasis most frequently affects the crop but may also invade the gut mucosa causing a malabsorption syndrome. The eyes and the lungs may also be affected. Young birds are most susceptible but adults of some species (especially lovebirds) are also frequently affected. Prolonged antibiotic therapy, vitamin A deficiency and the feeding of sour foods may predispose birds to candida but are not necessary for infection. Candida are found in low numbers as part of the normal flora in psittacine birds; disease is due to overgrowth. Candida in combination with bacteria is a common cause of crop impactions and death in baby psittacines.

Clinical signs - unthriftyness, subnormal weight, thickened crop, regurgitation, slow food passage in baby birds, increased appetite with concurrent weight loss in adult birds. Raised white patches of epithelium may be found in the mouth.

Diagnosis - Gram stain or culture of the feces, crop swabs or oral lesions.

Treatment - Nystatin, chlorohexidine, ketoconizole, amphoteracin, iodine swabbing of lesions, methylene blue, supplemental vitamin A, levamisole.

FAVUS

Etiological agent - Trichophyton sp.

Causes a chronic skin infection which is most common on the head. Lesions may appear crusty or pearly. Transmission is slow from bird to bird and incidence of the disease is low.

Diagnosis is by culture.

Treatment - No effective treatment has been found.