VELOGENIC VISCEROTROPIC NEWCASTLE DISEASE

Newcastle disease (ND) is a highly contagious, multistrain viral disease affecting most species of birds. Four major types have been described in domestic poultry; they have varying degrees of pathogenicity and organ specificity. Velogenic viscerotropic Newcastle disease (VVND) or exotic Newcastle disease is the most virulent form, producing high mortality in chickens, psittacines, birds of prey, waterfowl, and other avian species. VVND has also been called fowl pest, avian distemper, and raniket.[15, 17, 28]

ETIOLOGY

The agent of ND is a paramyxovirus. It is unstable at high temperatures and is inactivated rapidly at a pH of 3.0 or by lipid solvents or ultraviolet light. Warm temperatures and sunlight facilitate the destruction of the virus by chemicals. The virus is protected by suspension in proteinaceous material such as feces or tissues, and freezing temperatures prevent its inactivation by chemicals. For proper disinfection, all objects must be physically cleaned, and supplemental heat must be provided in the winter. Phenolic compounds are recommended for disinfection.

EPIZOOTIOLOGY

Newcastle disease was first recognized in 1926 by Doyle in Newcastle, England; it was found simultaneously in Java and Korea. The disease disappeared from England shortly afterward but remained endemic in Southeast Asia. From Asia, ND began to spread around the world. As the various forms of the disease were recognized, the most virulent, Asiatic form became known as Doyle's form, exotic Newcastle disease, or VVND. Psittacine birds were thought responsible for the spread of the disease to Europe and to South America between the years 1926 and 1942. Newcastle disease was first reported in the United States in 1944. An epiornitic of a very virulent strain of VVND reached all continents between the years 1968 and 1972.[13, 15]

Pet birds, especially psittacines, are often thought to be the source of the virus that leads to outbreaks of VVND in poultry. Newcastle disease virus (NDV) that was introduced by imported psittacines and mynahs into an exotic bird aviary reportedly spread into neighboring chicken farms in 1971 and resulted in a widespread outbreak in southern California. At this time, many pet birds and ornamental poultry were examined for the presence of ND, and the virus was isolated from 1.01 percent of the 3780 exotic birds tested. Semidomestic birds, including ducks, quail, pheasants, peafowl, and pigeons, were examined and found to have a 0.76 percent infection rate (4367 birds). VVND was eradicated from southern California poultry farms by extensive testing, slaughter, and vaccination, and the outbreak was contained.[22, 23]

Because of a successful eradication program in the United States and intensive vaccination procedures in other countries, VVND was not a significant problem in most countries by 1976. The fear of re-entry of VVND into domestic poultry flocks resulted in the establishment of an avian quarantine program in 1974. All avian species are now required to undergo a 30-day quarantine and be free of VVND before entry into the United States is permitted. Since the disease is potentially explosive in nature, the USDA continually monitors for outbreaks of VVND in the United States. The decision to eradicate VVND and to prevent re-entry was costly to implement, but the cost of living with VVND would have been much higher. Estimates by the USDA indicate that if VVND were to become endemic in the United States, the cost to the poultry industry would be $230 million annually.[14, 16]

Shipments from Mexico, Central America and Southeast Asia are most commonly infected with VVND; however, the disease has been reported in almost every country from which exotic birds are imported. VVND appears to have a seasonal incidence in Mexico and Central America that corresponds to the rainy season (from March to June). Poultry flocks in these countries are vaccinated annually at this time. This is also the time of year when young or baby birds, which are highly prized as pets, are available. Smuggling of baby birds from Mexico and Central America is at its height in the spring. Young Amazon parrots are the most popular species, and a VVND-surveillance program to detect these birds was established in California in 1979 through cooperation with practicing veterinarians in the area. This effort of the State of California and the USDA found 34 cases of VVND within the state in fiscal year 1982.[2, 12, 18, 19, 24]

Cases of VVND have been noted in 11 families of birds in six orders since initiation of the quarantine program for imported birds in 1974. The disease has also been reported in 13 additional orders of birds, from hummingbirds to ostriches.[1, 3–5, 7–9, 11, 13, 17, 20, 25, 26, 29]

Reservoirs

Hanson identified two wildlife reservoirs of NDV: (1) A chicken-avirulent virus was associated with migratory waterfowl of the temperate zones, and (2) a chicken-virulent virus was associated with jungle birds of the Old and New World tropics.[14]

Several workers have isolated lentogenic strains of

Modified from Clubb, S. L.: Viscerotropic Velogenic Newcastle Disease in Pet Birds. *In* Kirk, R. W.: Current Veterinary Therapy VIII. Philadelphia, W. B. Saunders, 1983: pp. 628–630.

Table 17–1. COMMERCIAL LOTS OF BIRDS ALLOWED ENTRY INTO THE UNITED STATES AND LOTS REFUSED ENTRY BECAUSE OF CONTAMINATION WITH VELOGENIC VISCEROTROPIC NEWCASTLE DISEASE (1974–1982).

Fiscal Year	Lots Allowed Entry	Number of Birds	Lots Refused Entry	Number of Birds	Percentage of Lots Refused
1974	13	27,696	6	18,969	31.6
1975	71	124,597	19	30,496	21.1
1976	179	222,922	24	47,943	11.8
1977	276	313,537	16	35,197	5.5
1978	409	520,725	12	28,770	2.9
1979	406	341,174	36	32,296	8.1
1980	428	591,375	15	6,810	3.4
1981	471	518,472	12	21,182	2.5
1982	394	554,321	10	19,145	2.5

NDV in migratory waterfowl that use the flyways over the United States. Serological reactors were found in six to 28 percent of the birds tested. This virus differs from the lentogenic strain found in chickens in that it is thermostabile. It was proposed that wild waterfowl neither receive their ND from nor pass the virus to poultry. The reservoir of the virus in nature is unknown.[1, 27]

Isolation of VVND has been reported on numerous occasions in exotic tropical birds. It has been proposed that the VVND virus became established in the tropical rain forests of South America, Asia, and Africa. Psittacines and other tropical birds become infected and yet are able to survive and act as "silent hosts" for the virus. Although the disease can cause high morbidity and mortality, many species have proved capable of carrying the virus for extended periods of time. Erickson and coworkers found that Amazon parrots could shed the virus for over one year after experimental exposure. Mynahs, conures, budgerigars, and nuns were shown to shed the virus for prolonged periods of time. Shedding of the virus may be intermittent and induced by stress.[9, 14]

Other unlikely avian reservoirs include (1) gamefowl or birds of prey, (2) pest birds such as sparrows and starlings that enter poultry yards, (3) pigeons, and (4) migratory songbirds. Outbreaks of VVND in gamefowl, pigeons, and birds of prey are usually associated with outbreaks in nearby poultry, but they have not been implicated as being the source. Sparrows require a large amount of virus for infection and the subsequent viral shedding is transient and in small amounts; therefore, they are probably not important in the spread of VVND. Attempts to isolate virus from songbirds have been infrequent, making it difficult to determine their role in the spread of infection. Erickson and coworkers found that pigeons were susceptible to infection with VVND when exposed to infected chickens and that they could transmit the disease back to chickens. Although pigeons were easily infected, 10,000 times more virus was required to kill them than was required to kill a chicken. Virus shedding was detected until 21 days after inoculation.[1, 10, 14, 27]

Mice living in poultry houses with infected birds can become naturally infected. Although mammals have been naturally and experimentally infected, and may bear the virus for a period of time, there is no evidence that the virus can perpetuate itself in mammals.[16, 17]

Transmission

Aerosols are the primary means of spread of NDV within a closely confined, susceptible flock of chickens. Approximately two days after exposure to the virus, the infected bird begins to liberate virus in expired air and continues to do so for several days. Coughing or sneezing is not required for production of these aerosols.[15]

Like any aerosol-borne disease, VVND is population-dependent in poultry, and is self-limiting if flocks are isolated. The high mortality would in most cases limit the spread. People are the primary means of spread of NDV from one poultry facility to another.[14, 15]

Other means of transmission that may be more prevalent in pet birds include direct contact with infected birds and contamination of food and water supplies with the virus excreted in feces. The spreading of virus among exotic bird facilities is primarily caused by the movement of infected birds among facilities during the incubation period.

CLINICAL SIGNS

Velogenic viscerotropic Newcastle disease is an extremely variable disease in exotic birds. No truly pathognomonic lesions or signs have been recognized. The overall pattern of clinical signs and gross postmortem lesions is much more significant than signs observed in individual birds.

The incubation period in psittacines is three to 16 days. In some cases, the only signs are acute death and high flock mortality after no response to antibiotics. This is especially true of young birds, which are more severely affected than mature birds. Severity of the disease also varies with the virulence of the virus, the species involved, and the viral dosage. Older or more resistant birds may initially show anorexia and depression. Bright yellow or bloody diarrhea may be present, and coughing, sneezing, and dyspnea may occur. In some birds the course of the disease is protracted. It is in these birds that neurological signs are seen; they include ataxia, in-

coordination, torticollis, hyperexcitability, and opisthotonos. Head tremors, nodding and jerking of the head, and unilateral or bilateral paralysis of the extremities are often observed. Neurological signs may persist in birds that recover from active infection. The neurological form may be manifested without any respiratory or visceral signs.[5, 7, 9, 11, 20]

Neurological signs of VVND in birds of prey are similar to those observed in chickens. Mortality rates are very high and postmortem lesions tend to be slight and nonspecific. Reported cases have corresponded to outbreaks in nearby poultry flocks.[6]

Strains of VVND also vary in virulence as they adapt to a particular host species. For example, some strains may be very virulent for psittacine birds but may cause a milder syndrome in chickens. Some strains will increase or decrease in virulence with each passage through birds. Clinical signs may be more severe at high ambient temperatures.

Lesions seen at necropsy in exotic birds are also extremely variable. The most consistently observed lesions in psittacine birds include petechial hemorrhages on serosal surfaces of the intestines, proventriculus, pericardium, and air sacs and petechiae in the tracheal lumen. Air sacculitis, hepatomegaly, and splenomegaly may be found. The lungs, proventriculus, and trachea may be congested. Petechiae may be found on the mucosal surface of the proventriculus and under the lining of the ventriculus. The visceral lesions of VVND that are commonly observed in chickens are rarely seen in psittacine birds.

Microscopic lesions that may be observed in the cerebellum include gliosis, neuronal degeneration, neuronophagia, mononuclear perivascular cuffing, endothelial hyperplasia in the molecular layer, and loss of Purkinje cells. Satellitosis, neuronal degeneration, perivascular cuffing, and endothelial hypertrophy may be present in brain-stem nuclei. Reticuloendothelial hyperplasia may be found in the liver and spleen.

DIAGNOSTIC PROCEDURES

Diagnosis must be based on the isolation of the virus by a USDA-approved laboratory. The virus may be isolated from cloacal or pharyngeal swabs of live birds. Samples of brain, lung, colon, spleen, and trachea should be submitted from dead birds. Swabs and tissues should be placed in brain-heart infusion broth, frozen, and shipped to the lab. Newcastle disease virus is isolated in the allantoic chamber of embryonated chicken eggs. Hemagglutination and hemagglutinin inhibition tests are performed on all eggs that die between two and five days after inoculation. If both tests are positive, the virus is inoculated into specific pathogen-free chickens for typing.

The local USDA office will designate a laboratory to process samples from the state involved. Most samples are processed by the National Veterinary Services Laboratory at the National Animal Disease Center in Ames, Iowa. Some state labs also perform NDV isolation tests.

An oligonucleotide "fingerprinting" technique has been developed for accurate identification of strains of NDV that cannot be differentiated by previous *in vitro* and *in vivo* typing methods. Although very expensive, this procedure allows accurate epidemiological studies.[21]

VACCINATION

Vaccination has made the control of VVND possible in poultry. The antigenic similarity of strains of NDV has simplified the vaccination of poultry and allowed worldwide use of vaccines that will protect against a variety of strains. Effective vaccination of exotic birds has also been reported, but federal regulations strictly prohibit the vaccination of exotic birds intended for import. The use of vaccines would produce an immune flock and thereby mask the presence of asymptomatic carriers. In quarantines of large numbers of birds, testing each individual bird is impossible. However, the shedding of virus in a closely confined, susceptible population would result in the rapid spread of infection and the easy detection of the virus. Vaccination does not eliminate the carrier state and fails to prevent infection with NDV; therefore, it contributes to the development of immune carriers.

MANAGEMENT OF SUSPECTED CASES

Velogenic viscerotropic Newcastle disease must be reported immediately, when suspected, to the local office of the USDA in your area. The policy of the USDA for birds in commercial trade is that once a bird is exposed, it is considered infected and must be given up for diagnostic evaluation. The USDA will pay indemnity on all birds they destroy. In the case of suspect, isolated pet birds, breeding collections, and zoo birds, arrangements may sometimes be made for isolation and testing.

All suspect birds must be quarantined until the results of viral isolation and typing are complete. This usually requires 10 to 20 days after specimens are received by the lab. If the isolate is of a domestic strain of NDV, the quarantine will be lifted. In the event of an isolation of VVND, the birds will be destroyed and the premises will be cleaned and required to remain empty for 30 days. A complete epidemiological trace of all infected birds will be undertaken by the USDA. Federal regulations for the handling of suspect birds change rapidly. Any local USDA office will be able to provide the most up-to-date regulations.

In view of the threat to zoo collections of purchasing an infected bird or a bird from an infected facility, the following recommendations are made for the handling of new avian acquisitions.

1. A quarantine of 30 to 60 days is recommended. The quanrantine facility should be physically isolated from the main bird collection and should be serviced by separate keepers.

2. Birds should be purchased from reliable sources. Donations should be considerd high-risk animals and should possibly be swabbed for isolation of viruses.

3. During an epidemic or outbreak, movement of birds should be severely restricted or stopped.

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