A Review of Avian Pneumovirus

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Diseases caused by avian pneumoviruses include Avian Pneumovirus infection, which is now recognized in the United States (US), and Turkey Rhinotracheitis (TRT), which is recognized everywhere else in the world. Swollen Head Syndrome (SHS) of chickens is also recognized throughout the world, except in the US, and is also caused by an avian pneumovirus. The avian pneumovirus is a member of the Pneumovirane subfamily from the Paramyxoviridae family of viruses. The TRT and SHS viruses are classified as A and B subgroups. The US isolates are presently not completely characterized.

TRT affects chickens and turkeys of all ages and was first described in South Africa in 1970s; yet the causative agent was not identified until 1985. It has spread through Europe, South America, Japan and the Middle East. In February 1997 the US National Veterinary Services Laboratory (NVSL) isolated APV from turkeys in Colorado with respiratory disease, designated as APV/CO. Clinical disease was first recognized in May 1996. Colorado has been free of APV since February 1998. The disease was first recognized in Fall 1996 and subsequently APV was identified in Minnesota, South Dakota and North Dakota. APV in the US is distinct from TRT virus in other countries.

APV infection in turkeys causes a rapidly spreading respiratory disease of all ages. Turkeys appear depressed and act chilled/seek heat. Increased mortality (up to 30%) is associated with secondary bacterial infections. It has been characterized as "CRS" (cough, rhinitis and sinusitis). APV is associated with high condemnations due to airsacculitis. Mortality is more severe in young birds. Recovery takes 10-14 days in uncomplicated disease. Exceptions to these clinical signs do occur in the field. Upon necropsy, in uncomplicated cases, the birds show sinusitis, rhinitis, and mucoid tracheitis. In complicated cases, pneumonia, airsacculitis, pericarditis and perihepatitis is observed.

The rate of spread within a flock or between flocks may vary. Management factors, such as stocking densities and ventilation, affect severity of disease. Other concurrent infections, such as, E. coli, Bordetella avium, Pasteurella, Newcastle disease virus and Ornithobacterium rhinotracheale, also affect severity of disease. In breeders, APV disease can cause a drop in egg production.

In Minnesota, APV disease appears to be cyclical (occurring in "waves"). The disease is associated with high geographical density of turkeys. There have been a few reports of clinical disease not spreading on a multi-age farm, serologically positive flocks without clinical disease, serologically negative flocks but PCR positive with clinical disease, and flocks serologically

positive but PCR negative. In 1997, APV resulted in rapid spread, severe clinical disease, more treated flocks and high, acute mortality in younger flocks. APV infections in 1998 have resulted in slower spread, mild clinical disease, fewer treated flocks and low, chronic mortality in older flocks. These differences between 1997 and 1998 may be associated with variation in the virus and/or management changes.

APV is probably, but not yet proven, to be transmitted by the movement of contaminated birds, equipment, people and/or litter. Air-borne transmission is suspected (up to 1-2 miles). Egg transmission is not documented. Wild birds and reservoirs are also suspected in the spread of the virus. In the laboratory, the virus can survive for at least 2 weeks at room temperature, and at least 4 weeks refrigerated, and less than 72 hours at 37C (100F).

Clinical signs are not pathognomonic for a diagnosis of APV. A diagnosis may be made by either serology, PCR or virus isolation. Current serological tests include enzyme linked immunosorbent assay (ELISA), virus neutralization (VN), and immunofluorescence (FA). No commercial tests are available in the US. Laboratories in the US presently providing serological testing are NVSL and the University of Minnesota. ELISA is currently the preferred diagnostic. The European subgroup A and B serological tests detect APV/CO very poorly. The polymerase chain reaction (PCR) procedure detects virus nucleic acid and is used to sample respiratory tissues (such as, tracheal swabs and turbinates). Samples should be fresh, refrigerated, and not frozen. Presently the only US laboratory providing PCR is the University of Minnesota. For any diagnostic test, sample both affected and unaffected birds within a sick flock.

Virus isolation, by propagation in cell culture and electronmicroscopy (EM) are other diagnostic tools. It is noted that Tracheal Ring Organ Culture (TROC) is not applicable for the APV/CO since this isolate is not ciliostatic (TROC relies on ciliostasis as a diagnostic criterion). Other procedures, such as Chick Embryo Yolk Sac and Chick Embryo Fibroblast cells, have been used successfully for initially isolating APV/CO. After the virus is isolated, it may be propagated in Vero Cells, BS-C-1, CEF or QT-35 systems. Sample respiratory tissues (especially the turbinates) early in the infection (before clinical signs). Multiple host systems (i.e., virus isolation procedures) should be used to maximize isolation of virus(es). Consult the laboratory for specific sample submission procedures.

APV can resemble other respiratory diseases found in turkeys. A differential diagnosis should include: MG (Mycoplasma gallisepticum), MS (Mycoplasma synoviae), AI (avian influenza virus), Fowl Cholera (Pasteurella multocida), NDV (Newcastle disease virus), BART (Bordetella avium rhinotracheitis), ORT (Ornithobacterium rhinotracheale) and all other respiratory diseases.

Researchers note that APV/CO is distinct from APV subgroups A and B that cause TRT in other countries. It differs in both molecular and antigenic characteristics. APV/CO is more closely related to subgroup A, comparing F protein and virus neutralization. APV/CO is distinct from A and B comparing M protein sequences. Further research is ongoing to more fully characterize the US isolates of APV.

Treatment of APV infections should focus on management practices ("tender loving care"). It is critical to adjust the barn temperature for bird comfort. Optimize air quality in the barn and be careful to avoid chilling birds. Suspend tilling of litter and suspend all vaccinations to avoid

stressing the flock. Antibiotics such as chlortetracyclines, sulfas and fluoroquinolones may be appropriate to control secondary bacterial infections associated with mortality. Results are highly variable. For best results, check antibiotic sensitivity patterns of bacterial isolates.

To control APV spread, biosecurity procedures must be a priority. Effective communication and cooperation among poultry growers coupled with integrated poultry company management is essential. If APV positive flocks are identified, isolate younger birds from older flocks. Wild bird control is important, as free-living birds are suspected in carrying the virus. Properly dispose of contaminated litter and of dead birds is a must, so as not to contaminate other farms. Proper loading and routing of live haul trucks, especially when moving infected flocks, is important. To insure compliance, audit all control measures. To minimize the risk of spreading disease to other flocks, avoid introduction of susceptible birds. It is suggested to delay multi-age farm placements. Depopulation may be necessary to minimize virus spread. Reduce the geographic density of turkeys through delayed placements or depopulation and coordinating with local farms. Proper cleaning, washing and disinfecting procedures (including the proper use of formaldehyde) have been parts of successful programs to minimize the spread of disease. Also minimize stresses (management) and other complicating diseases (NDV, ORT, BART). Another essential step is to avoid high stocking densities especially in brooder barns (less than approximately 1 square foot per bird). Optimizing barn temperature and ventilation is important in controlling an outbreak within a flock.

In Europe, vaccination is helpful for controlling TRT. There are vaccines that induce protective antibodies to A and B subgroups. No vaccines are available in the US. Laboratory experiments show that live subgroup A and B vaccines from Europe provide protection against APV/CO. In Europe there is cross-protection between A and B. Commercial birds are typically given a live vaccine, via spray administration, at the hatchery. Vaccination of commercial birds minimizes clinical disease and associated mortality, yet vaccination reactions can be a problem. In breeders, a live vaccine is given at 0, 6, and 10 weeks of age, followed by a killed vaccine at 18 and 26 weeks of age. Breeder vaccination minimizes egg drop, clinical disease and mortality.

The US industry has several needs relating to APV. The industry might consider eliminating the virus, since this disease appears limited to a specific geographic area. The industry must address biosecurity "failures", such as with contaminated employees and equipment moving between farms, inadequate barn clean out practices, inadequate downtime, improper dead bird disposal or wild bird control practices. The management of multi-age farms might also need to be re-evaluated.

More research is needed to better understand APV. Proper sampling protocol should be confirmed. There is much to be understood about the stability of the virus, of potential reservoirs, and how it is transmitted. Once the duration of virus shedding in the field is known, then other questions may be answered, such as: When is it safe to transport previously infected birds? When is it safe to put birds onto a previously infected farm? More information is needed to better characterize APV variation. This will be invaluable for monitoring infections and developing control programs. Vaccine research is needed with the US isolates. Finally, diagnostic tests must be rapid, reliable, inexpensive and easy-to-use.

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