

Egg Drop Syndrome 1976

*Egg Drop Syndrome '76,
Egg Drop Syndrome,
Duck Adenovirus A Infection,
Duck Adenovirus 1 Infection,
Adenovirus 127 Infection*

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Importance

Egg drop syndrome '76 is a viral disease that can cause significant economic losses in poultry. The causative virus, duck atadenovirus A, is widespread among asymptomatic waterfowl. Outbreaks of respiratory disease, with elevated mortality, have been reported rarely in ducklings and goslings. However, the most significant impact of this virus is on the chicken industry. Egg drop syndrome '76 was first described in chickens in the 1970s. The initial outbreaks were linked to a contaminated Marek's disease vaccine, which infected breeding chicken flocks. The virus subsequently spread to other flocks through infected eggs. In chickens, duck atadenovirus A causes egg production to fall, and many eggs are thin-shelled, shell-less or otherwise abnormal. Although the initial outbreaks were controlled by eradicating this virus from commercial breeders, it became endemic in chickens in some parts of the world. Viruses carried in ducks and geese can also spread to chickens, either during direct contact or through sources such as contaminated water. A few outbreaks, with clinical signs similar to those in chickens, have been reported in quail and turkeys, and this syndrome might also occur in other birds.

Etiology

Egg drop syndrome '76 is caused by duck atadenovirus A (formerly duck adenovirus A), a member of the genus *Atadenovirus* and family Adenoviridae. This virus has also been called duck adenovirus 1 (DAdV-1) egg drop syndrome (EDS) virus, egg-drop-syndrome-76 (EDS-76) virus and adenovirus 127. Only one serotype has been found.

In the past, egg drop syndrome '76 was often called egg drop syndrome. The longer name is now recommended, to avoid confusion with duck egg drop syndrome, a recently identified disease caused by a flavivirus.

Species Affected

Waterfowl, including domesticated ducks and geese, appear to be the natural hosts for duck atadenovirus A. To date, this virus has only been isolated from domesticated birds. Antibodies to duck atadenovirus A also occur in various wild waterfowl, but attempts to recover the virus have been unsuccessful, possibly because birds are infected early in life and/or the period of virus shedding is brief. Respiratory illnesses have been documented in domesticated Pekin ducks, Muscovy ducks and geese. One study reported finding duck atadenovirus A in a flock of ducks with abnormal eggs and a drop in egg production, but a causative role was not demonstrated, and other agents (e.g., duck flaviviruses) might have been responsible.

Clinical cases affecting egg production have been reported in chickens, Japanese quail (*Coturnix coturnix japonica*) and turkeys. Experimentally infected turkeys, pheasants and guinea fowl remained asymptomatic, but one outbreak was diagnosed in turkeys in Croatia. This flock was also infected with avian pneumovirus; however, the clinical signs only developed after the birds became infected with duck atadenovirus A, and resolved after vaccination against egg drop syndrome '76. Some reports suggest that naturally infected guinea fowl are also affected.

Little is known about duck atadenovirus A in other birds; however, antibodies have been documented in cattle egrets (*Bubulcus ibis*), gulls and pigeons, and in two captive owls, a captive stork (*Ciconia* sp.) and a captive swan (*Cygnus cygnus*). Both owls and the stork had a history of having laid abnormal eggs one year, but any involvement of duck atadenovirus A is speculative. Egg-laying history for the swan was unavailable.

Zoonotic potential

There is no evidence that duck atadenovirus A infects humans.

Geographic Distribution

Duck atadenovirus A can be found worldwide in ducks and geese. Clinical cases are mainly reported from Europe, Asia, Africa and Latin America; however, an outbreak of respiratory disease occurred in two related flocks of ducks in Canada in 2007. In the U.S., the most recent reports of infections in domesticated birds were



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published in 1978 (a chicken) and 1979 (healthy ducks). Serological surveys have found duck atadenovirus (or serologically related viruses) in wild waterfowl in North America.

Transmission

Duck atadenovirus A can be transmitted vertically in eggs; both the interior and the exterior of the egg contain virus. Experimentally infected mature chickens primarily lay infected eggs 8 to 18 days after inoculation. Chicks hatched from infected eggs or infected when they are young may excrete the virus immediately. More often, the virus remains latent until birds become sexually mature. It is then excreted in both eggs and droppings. The viruses in droppings originate from replication in the oviduct rather than the intestinal tract.

Duck atadenovirus A is also transmitted horizontally between birds. Birds are thought to become infected mainly by the oral route; however, respiratory disease in goslings was reproduced by intratracheal administration. Duck atadenovirus A can be spread on/in fomites including equipment and water. Some outbreaks have been attributed to contact with wild birds or water contaminated by wild bird feces. This virus can be transmitted iatrogenically via reused needles. Transmission by insects is theoretically possible, but unproven.

Disinfection

Adenoviruses are resistant to many commonly used disinfectants including lipid solvents, 50% alcohol and 2% phenol. Sodium hypochlorite, chlorine dioxide, iodophors, aldehydes and some other disinfectants have demonstrated efficacy against some adenoviruses, although certain agents may require prolonged contact times. Adenoviruses are tolerant of a wide pH range, from pH 3 to pH 10. They are also relatively tolerant of heat, but susceptible to autoclaving.

Incubation Period

The incubation period for egg drop syndrome '76 is highly variable, as birds infected in ovo or as chicks can remain asymptomatic until they begin laying eggs. Experimentally infected mature chickens produced abnormal eggs 10 to 24 days after inoculation, and experimentally infected Japanese quail began laying abnormal eggs after 9 days. Experimentally infected goslings and ducklings developed respiratory signs starting 2-4 days after inoculation.

Clinical Signs

Chickens infected with duck atadenovirus A lay abnormal eggs, with a variable decrease in egg production. In naive chicken flocks, the first sign is usually a loss of color in pigmented eggs, followed by the production of thin-shelled, soft-shelled and shell-less eggs. The shells may also be rough or chalky. Shell-less eggs are not always found, as they may be eaten by the birds. Chicken eggs that are suitable for hatching/setting remain fertile and hatch normally.

Affected birds do not usually appear sick, although some were reported to develop transient diarrhea (probably due to fluid and exudate from the oviduct) and dullness or inappetence before the changes in egg laying. Flocks with some pre-existing immunity usually experience a series of small disease episodes, with minimal effect on egg laying. Overall, there is a small drop in production or a failure to achieve predicted production targets. Naturally infected newborn chicks and growing young chickens seem to be unaffected, although one study described increased mortality during the first week if chicks were inoculated at one day of age. Similar effects on egg laying, without significant illness, have been reported in other species, including quail and turkeys.

Respiratory signs have been documented on a few occasions in young goslings and ducklings. In 2001, an outbreak of severe acute respiratory disease, with elevated mortality, affected 4-20 day-old goslings in Hungary. The clinical signs included anorexia, depression, sneezing, coughing, dyspnea and rales. Similar signs occurred in 1-week-old Muscovy ducklings in two related flocks in Canada in 2007, and in 9-day-old Pekin ducks in South Korea in 2011. This illness has been reproduced experimentally in 1-day-old goslings and 3-day-old ducklings. One study reported decreased egg production and abnormal eggs in a flock of infected ducks. Duck atadenovirus A was isolated from egg yolks and cloacal samples from these birds, but it was not proven to be the cause of the syndrome.

Post Mortem Lesions

In mature chickens and other gallinaceous birds, the lesions are minimal and confined to the female reproductive tract. There may be inactive ovaries, atrophy of the oviducts, and edema and white exudate in the uterus (shell gland). These signs were accompanied by enlargement and congestion of the spleen in some birds, such as experimentally infected quail.

In several reports of respiratory disease in goslings and ducklings, the most obvious lesion was a plug of gelatinous to firm, white, opaque material in the trachea. The trachea and lungs were edematous, with varying degrees of congestion in some birds. The lungs were sometimes consolidated. In some outbreaks, additional lesions included ecchymoses on the epicardium, mottling in the liver and slight enlargement of the spleen.

Diagnostic Tests

Duck atadenovirus A, its nucleic acids and antigens occur in the reproductive tract, especially the uterus (shell gland), of affected hens. This virus may also be detected sometimes in cloacal swabs, and it is occasionally found in other internal organs. The oviduct and kidneys are reported to be frequently positive in PCR tests. The period of virus shedding is relatively short. If the virus is not found in affected hens, abnormal eggs may be fed to naive hens, and their shell gland examined for evidence of infection when they produce abnormal eggs. In goslings and ducklings with

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respiratory disease, duck adenovirus A has mainly been detected in the respiratory tract (i.e., lungs and trachea). It was also found in the liver and intestines of some goslings.

Duck adenovirus A can be isolated in embryonated duck or goose eggs, and in cell cultures. Susceptible cell lines include duck and chick embryo liver, duck kidney, and duck embryo fibroblast cells. Viral nucleic acids can be detected by PCR, and antigens by antigen-capture ELISAs or immunofluorescence. Histology of the reproductive tract can help support the diagnosis,

Serology is also used for diagnosis in unvaccinated flocks. Serum samples should be collected from hens that have laid abnormal eggs, as they are most likely to have produced antibodies. A rise in titer is needed for a definitive diagnosis. Serological tests include hemagglutination inhibition (using fowl red blood cells), ELISAs and serum neutralization. An immunofiltration (flow-through) assay has been published, and may be useful as a preliminary field test. The double immunodiffusion test was employed in the past. Chickens infected before sexual maturity do not usually develop antibodies until they mature. In breeding flocks certified as duck adenovirus '76 free, serological testing is generally done around 32-35 weeks of age.

Treatment

No specific treatment is available. Supportive care, including treatment for any secondary bacterial infections, is likely to be helpful in young waterfowl.

Control

Disease reporting

Veterinarians who encounter or suspect duck adenovirus A infection should follow their national and/or local guidelines for disease reporting. In the U.S., state or federal veterinary authorities should be informed immediately.

Prevention

Duck adenovirus A can enter a poultry flock in infected eggs or inapparently infected replacement birds. It is difficult to detect this virus in infected chickens before they become sexually mature. Some countries have eradicated duck adenovirus A from breeding chickens or have flocks certified to be virus-free. Uninfected flocks should also be kept from contact with domesticated and wild birds that may be infected, especially waterfowl and their environments.

Biosecurity measures, including cleaning and disinfection of any shared equipment, can reduce the risk from fomites. In many cases, this virus is thought to enter a flock on contaminated egg trays. Dedicated farm equipment and egg trays help mitigate this risk. Shared egg trays should be cleaned and disinfected before use. Potentially contaminated water should be chlorinated.

Inactivated vaccines can prevent clinical signs and also decrease virus shedding, although they do not prevent birds from becoming infected. Sentinel birds, tested periodically

for antibodies, can be used to detect virus circulation in a vaccinated flock.

Quarantine and depopulation of infected farms, followed by cleaning and disinfection, may be employed during some outbreaks. Composting infected chicken carcasses for 20 days has been reported to inactivate the virus.

Morbidity and Mortality

Egg drop syndrome '76 is reported most often in chickens, and occasionally in quail and other gallinaceous birds. However, one study suggested that experimentally infected quail are as susceptible to this illness as chickens. Outbreaks in chickens usually last 4 to 10 weeks. A 10-40% drop in egg production can be expected in naive chicken flocks. In flocks with some immunity, this decrease may be as little as 2-4%. All breeds of chickens are susceptible. In quail, egg production decreased 10% during one outbreak, and 50% in another. Deaths have not been reported in affected chickens, quail or turkeys.

Respiratory disease outbreaks have only been seen in young goslings and ducklings. In the outbreaks to date, affected geese ranged from 4 to 20 days of age, while duckling flocks contained 7-9 day old birds. Mortality rates were approximately 6-7% in goslings, and 2-5% in ducklings. Respiratory disease is mainly thought to occur in flocks where the young birds are not protected by maternal antibodies. Very few outbreaks have been documented; their rarity may be explained by high exposure rates in waterfowl. However, some authors suggest that more virulent strains of duck adenovirus A might be able to break through the protection from maternal antibodies.

Internet Resources

[International Veterinary Information Service \(IVIS\)](#)

[The Merck Veterinary Manual](#)

[World Organization for Animal Health \(WOAH\)](#)

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* Link defunct