

# VIRAL ARTHRITIS

Slide study set #1

*Prepared by:*

**Dr. N. O. OLSON**

West Virginia University

Morgantown, West Virginia

*Revised by:*

**Dr. GEORGE E. ONEY**

Central Veterinary Diagnostic Laboratory

Richmond, Virginia

**This slide study set was created in 1973 and revised in 1994; some information may be outdated.**

COPYRIGHT 1973, 1994

AMERICAN ASSOCIATION OF AVIAN PATHOLOGISTS, INC.



AAAP BUSINESS OFFICE  
NEW BOLTON CENTER  
382 WEST STREET RD.  
KENNETT SQUARE, PA 19348

*CD version produced in 2001 with the assistance of the AAAP Continuing Education and Electronic Information Committees*

## VIRAL ARTHRITIS

By: N. O. OLSON and G. E. ONET

---

Viral arthritis is an infectious disease of chickens and turkeys, affecting primarily the synovial membrane and tendon sheaths, caused by a reovirus. The occurrence is cyclical, causing heavy losses in broiler and broiler breeders. Morbidity can be as high as 100%, while mortality is generally less than 6%.

The disease was first recognized as a separate clinical entity in 1967 in the U. S. and England. It is probably world wide in distribution. Economic losses are caused not only by mortality but also by general lack of performance, expressed in diminished weight gain, poor feed conversion, and reduced marketability of affected birds.

Reoviruses have been associated with different disease conditions: enteritis, acute and chronic respiratory syndromes, growth retardation, myocarditis, pericarditis, hepatitis, early mortality in poults, bursal and thymic atrophy, and, most recently, the malabsorption syndrome.

---

**Etiology.** Viral arthritis is caused by an RNA nonenveloped virus with icosahedral symmetry and double capsid structure and is a member of the genus reovirus. The viral particle is approximately 75 nm in diameter. Like other reoviruses, this virus is resistant to heat (it withstands 60° C for 8-10 hours) and to pH 3.

Reoviruses can be readily differentiated from other viruses by their typical physicochemical characteristics and the presence of a group specific antigen, demonstrable with the agar gel precipitin test. Cultivation is possible in embryonated chicken eggs, primary chicken cell cultures, and some established cell lines.

The pathogenicity of selected reovirus isolates may be enhanced by coinfection with *Eimeria tenella* or *Eimeria maxima*. Also, exposure to infectious bursal disease or particular dietary regimes may increase the severity of viral arthritis clinical signs. Reoviruses may exacerbate disease conditions caused by other pathogens, including chicken anaemia agent, *E. coli*, and Newcastle disease virus.

**Epizootiology.** Reoviruses have been isolated from a variety of clinically diseased avian species: chickens, turkeys, pigeons, ducks, geese, and psittacines. However, a firm etiological relationship was not always established.

Reoviruses spread rapidly, in a period of about one week the whole flock may become infected. The disease seems to have a cyclical nature, in which parental immunity probably plays a part. The clinical stage may run its course in a single outbreak or in some cases continue for several months.

Chickens are most susceptible to pathogenic reoviruses at 1 day of age and then develop an age-related resistance beginning as early as 2 weeks of age.

Transmission of the infection can be horizontal or vertical. Because of virus resistance to inactivation, mechanical means may be an important factor in spreading the infection. The ability of the virus to spread laterally varies from strain to strain and is based primarily on the fact that it is excreted from both the intestinal and respiratory tracts of infected birds. The virus may persist for long periods of time in the cecal lymphoid points and patches. Carrier birds are potential sources of infection for penmates. Vertical transmission plays a major epidemiological role.

**Clinical signs.** The incubation period differs, depending upon virus pathotype, age of host, and route of infection. The incubation period in 2-week-old birds is 1-10 days. Often infections are inapparent and demonstrable only by serology or virus isolation. Many reoviruses cause microscopic inflammatory lesions in the digital flexor and metatarsal extensor tendons, without development of macroscopic lesions. Usually, outbreaks of viral arthritis occur when birds are 4-5 weeks of age. The disease is rare in older birds but has been described in birds 47 weeks of age.

In acute infections, chickens appear stunted and exhibit different degrees of lameness, depending on the severity of morphologic changes. Swelling of the tendon sheaths of the digital flexor tendons of the shank and of the metatarsal extensor tendons above the hock can be detected by gross inspection or palpation. Affected birds tend to sit on their hocks and are reluctant to move. Swelling of the footpad is less frequent. Rupture of the gastrocnemius tendon can occur occasionally and results in the bird's inability to hold the hock joint in an upright position and may lead to ankylosis and immobility. The typical uneven gait in bilateral rupture of the tendon results from the bird's inability to immobilize the metatarsus. A secondary consequence of tendon rupture is rupture of blood vessels. However, in spite of the marked tendon involvement, many birds appear in good condition. Erythrocyte, hematocrit, and total leukocyte determinations are frequently within normal range. In some cases, there is a rise of heterophil percentage and a decrease of lymphocyte percentage.

**Morphopathologic changes.** Macroscopic lesions in chickens naturally infected with viral arthritis consist of swelling of the digital flexor and metatarsal extensor tendons. The latter lesion can be observed just above the hock and may be readily observed when feathers are removed. The hock joint usually contains a small amount of straw-colored or blood-tinged exudate. In some cases, there is a considerable amount of purulent exudate resembling that seen with infectious synovitis. Early in the infection there is marked edema of the tarsal and metatarsal tendon sheaths. Petechial hemorrhages are frequent in the synovial membranes above the hock.

Internal organs are usually normal.

In chronic evolving cases, the inflammatory process leads to hardening and fusion of the tendon sheaths. Small-pitted erosions develop in the articular cartilage of the distal tibiotarsus. They enlarge, coalesce, and extend into underlying bone. Overgrowth of fibrocartilaginous pannus can develop on the articular surface. Condyles and epicondyles are frequently involved.

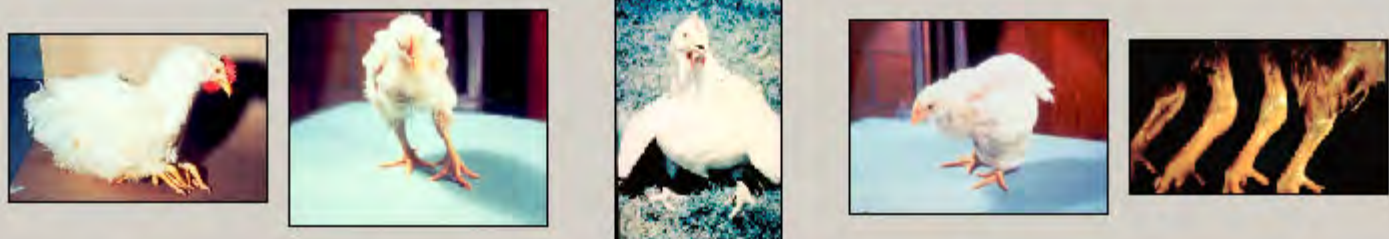
Histopathologically, edema, coagulative necrosis, heterophil accumulation, and perivascular infiltration can be seen. There are also hypertrophy and hyperplasia of the synovial cells, with infiltration by lymphocytes and macrophages, and proliferation of reticular cells. The latter changes cause parietal and visceral layers of tendon sheaths to become markedly thickened. The synovial cavity is filled with heterophils, macrophages, and sloughed synovial cells.

In chronic cases, the synovial membrane develops villous processes and lymphoid nodules. An increased amount of fibrous connective tissue and pronounced infiltration or proliferation of reticular cells, lymphocytes and plasma cells develops, appearing as chronic fibrosis of tendon sheaths, with fibrous tissues invading the tendons. Sometimes, tendons are completely replaced by irregular granulation tissue and large villi form on the synovial membrane. Development of sesamoid bones in the affected limb is inhibited. Linear growth of cartilage cells in the proximal tarsometatarsal bone becomes narrow and irregular. Osteoblasts become active and lay down a thickened layer of bone beneath the erosion. Osteoblastic activity is present in the condyles, epicondyles, and accessory tibia, producing osteogenesis and subsequent exostosis. There are transverse and vertical clefts in the femur growth plate, with necrosis of cartilage and fragmentation of the bone, but arthritis is not present.

**Immunity.** Contact with reoviruses results in specific antibody production, which can be detected by agar gel immunodiffusion, serum neutralization, or chicken embryo assays. Neutralizing antibodies are detectable 7-10 days following infection and precipitating antibodies after approximately 2 weeks. The former seem to persist longer than the latter (over 300 days). The importance of antibodies in establishing protection is not clearly understood, since birds may become persistently infected in the presence of

## REFERENCES

1. Dalton P. J., Henry R., 1967, Tenosynovitis in poultry, *Vet. Rec.*, 80, 638
2. Guneratne J. R. M., Jones R. C., Giorgiou K., 1982, Some observations on isolation and cultivation of avian reoviruses, *Avian. Pathol.*, 11, 453-462
3. Ide P. E., 1980, Avian reovirus antibody assay by indirect immuno-fluorescence using plastic microculture plates, *Can. J. Med.* 46, 39-42
4. Jones R. C., Giorgiou K., 1966, Reovirus-induced tenosynovitis in chicken: The influence of age at infection, *Avian Pathol.*, 13, 441-457
5. Olson N. O., Kerr K. M., 1966, Some characteristics of an avian arthritis viral agent, *Avian Dis.*, 10, 470-476
6. Olson N. O., Solomon D. P., 1968, A natural outbreak of synovitis caused by a viral arthritis agent, *Avian Dis.*, 12, 511-516
7. Onet E., 1985, Arthritis virala, In: *viruses and Viral Diseases of Animals*, Vol. II, Ed. Dacia, Cluj-Napoca, 339-341
8. Page R. K., Fletcher O. J., Rowland G. N., Gaudry D., Villegas P., 1982, Malabsorption syndrome in chickens, *Avian Dis.*, 26, 618-624
9. Page R. K., Fletcher O. J., Villegas P., 1982, Infectious tenosynovitis in young turkeys, *Avian Dis.*, 26, 924-927
10. Robertson M. D., Wilcox G. E., 1986, Avian Reovirus, *Vet. Bull.* 36, 155-174
11. Rosenberger J. K., Olson N. O., 1991, Reovirus infectious, In: *Disease of Poultry*, Ninth Edition, Iowa State University Press, 639-647
12. Stuart B. P., Cole R. J., Waller E. R., Vesonder R. F., 1986, Proventricular hyperplasia (malabsorption syndrome) in broiler chickens, *J. Environ. Pathol. Toxicol. Oncol.*, 6, 369-385
13. Van Der Heide L., 1977, Viral arthritis/tenosynovitis: A review, *Avian Pathol.*, 6, 271-284
14. Van Der Heide L., 1983, Development of an attenuated pathogenic reovirus vaccine against viral arthritis, *Avian Dis.*, 27, 698-706
15. Wood G. V. V., Nicholas R. A. J., Herbert C. N., Thornton D. H., 1980, serological comparisons of avian reoviruses, *J. Comp. Path* 90, 29-38



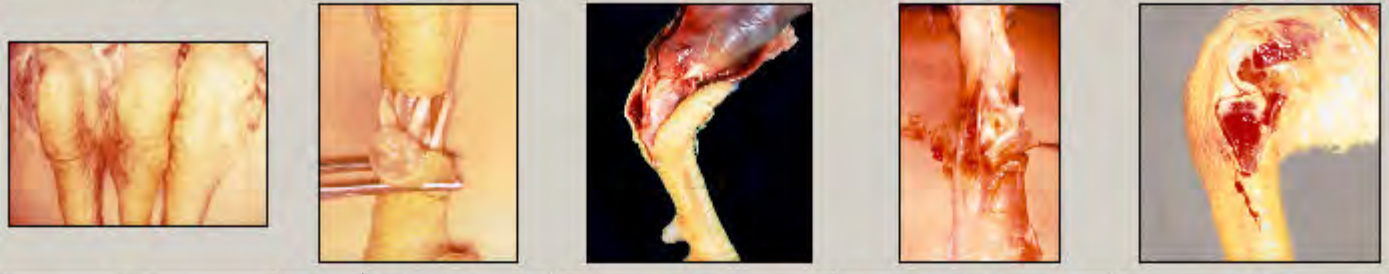
1.01.jpg

1.02.jpg

1.03.jpg

1.04.jpg

1.05.jpg



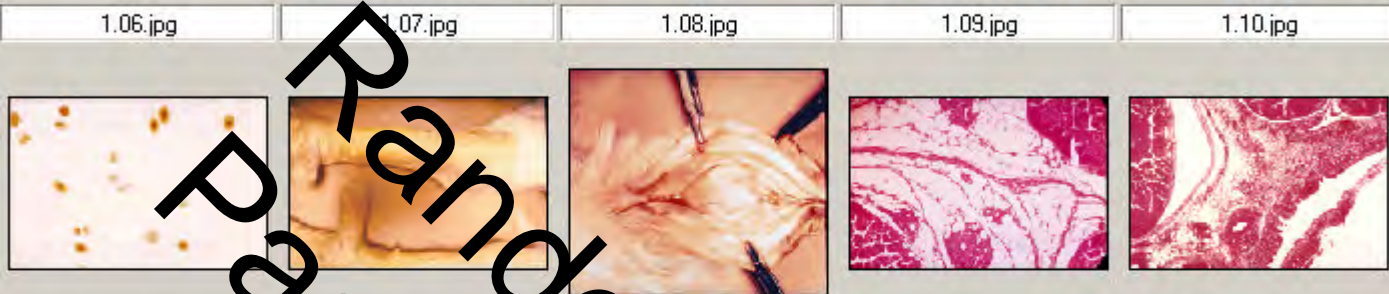
1.06.jpg

1.07.jpg

1.08.jpg

1.09.jpg

1.10.jpg



1.11.jpg

1.12.jpg

1.13.jpg

1.14.jpg

1.15.jpg



1.16.jpg

1.17.jpg

1.18.jpg

1.19.jpg

1.20.jpg



1.21.jpg

1.22.jpg

1.23.jpg

1.24.jpg

1.25.jpg



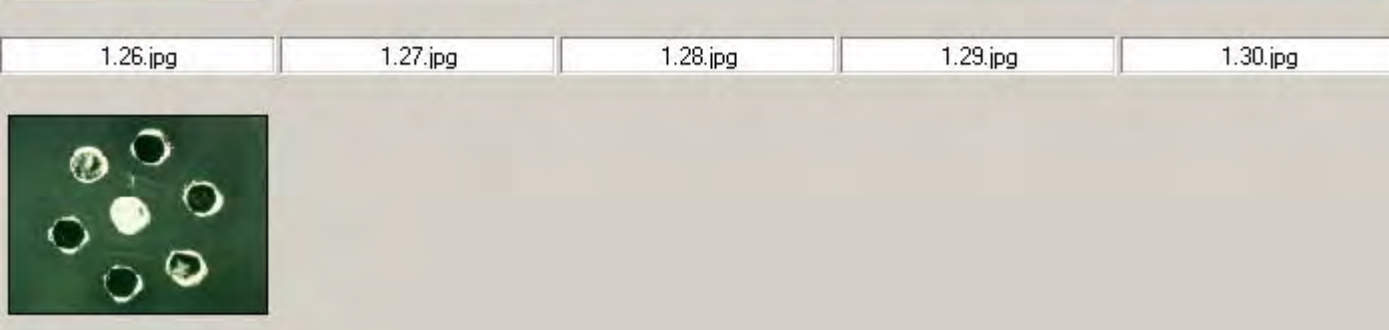
1.26.jpg

1.27.jpg

1.28.jpg

1.29.jpg

1.30.jpg



1.31.jpg

Random Sample Pages for Preview