REO, Pneumo and Parvo virus infection

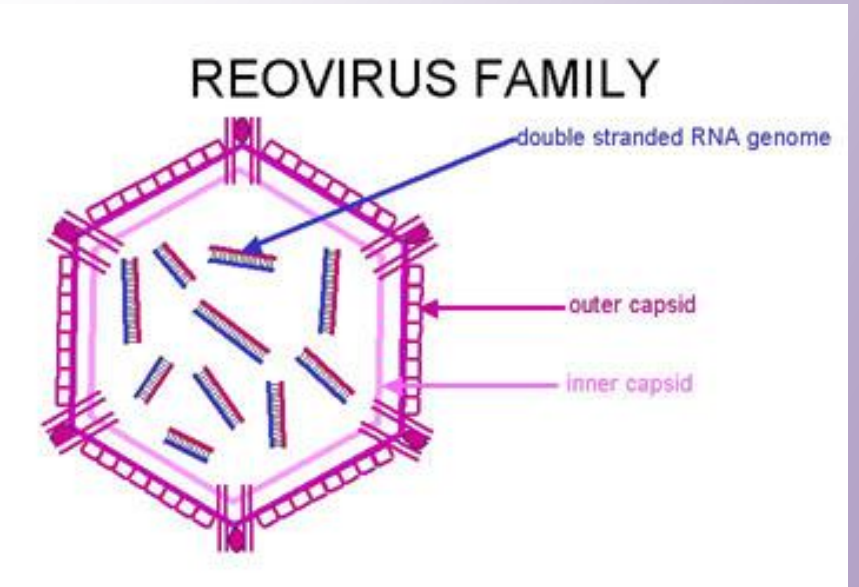
REO virus infections

INTRODUCTION:

- The family *Reoviridae* contains three genera, *Orthoreovirus* (reoviruses), *Orbivirus* and *Rotavirus*.
- Orbiviruses do **not** cause disease in domestic poultry.
- Reoviruses have been isolated from a variety of conditions including viral arthritis tenosynovitis, malabsorption syndrome and immuno-suppresion.

Etiology:

- Reoviruses are non-enveloped icosahedral symmetry with double shelled capsid classified into several serotypes.
- The genome consists of 10 segments of **double-stranded RNA**.
- More than 11 serotypes exist (SN test).
- Replication occurs in the cytoplasm.
- Reoviruses are heat resistant being able to withstand 37°C for 16 weeks 22°C for 48-51 weeks. Reoviruses are resistant to ether, PH3, 2% Lysol and 3% formalin. Although it could be inactivated by 0.5% organic iodine.
- Reoviruses grow well in chick kidney and chick embryo liver and lung cell cultures. They form syncytia with prominent cytoplasmic inclusions. They also grow well in embryonated eggs when inoculated into the yolk sac or on to the chorioallantoic membrane.



Epizootiology:

- **Natural Hosts:** Although reoviruses have been isolated from many avian species, chickens and turkey seem to be the most susceptible species.
- There is wide variation in virulence between antigenically similar isolates.
- Reovirus infections are ubiquitous in domestic fowl, the vast majority of reoviruses isolated appear to be nonpathogenic.
- **Transmission:** Shortly after infection there is a viraemia. Virus grows in virtually all organs. The highest titres are found in the alimentary tract, spleen, tendons and respiratory tract. Reoviruses speared in most cases horizontally through contact with respiratory and intestinal excretions. The virus generally appears to shed from the intestine for longer period. Egg transmission even occurs in low level, plays an important role in the epidemiology of Reoviruses (nucleus for horizontal transmission) .

Incubation period:

The incubation period differs according to virus patho-types host age, route of exposure from one day to 2 weeks.

Morbidity & mortality:

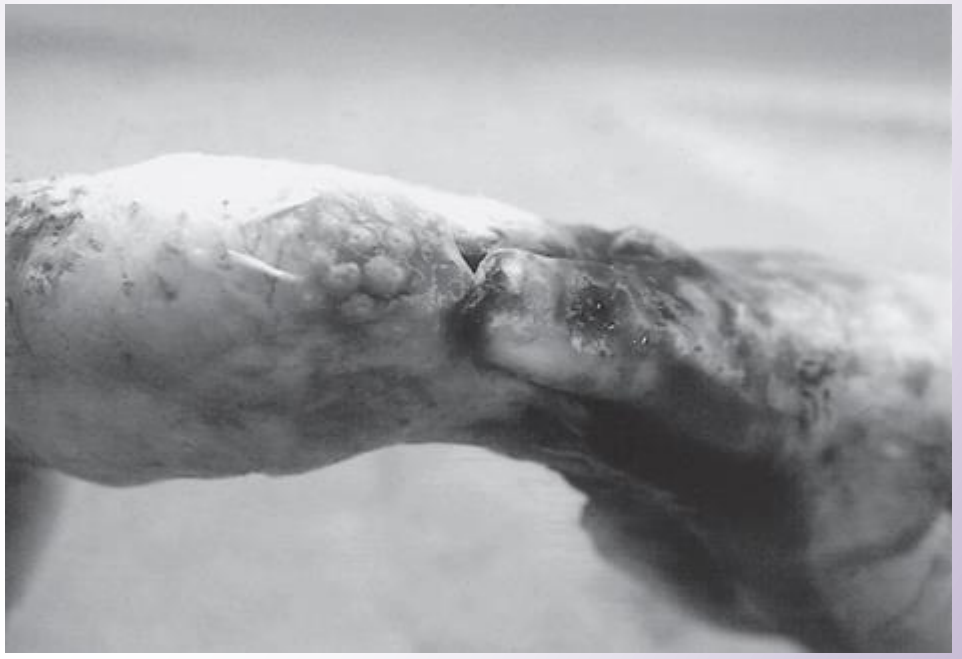
Viral arthritis has been usually seen in young birds 4-7 weeks but may be also seen in older birds. Morbidity can be as high as 100% while mortality less than 6%.

Clinical signs:

- Lameness, hock joint immobilization, enlargement in the area of gastrocnemius or digital flexor tendons. Sometimes rupture of gastrocnemius tendon especially in male roasters. In some cases swelling of the footpad.
- The malabsorption syndrome in broilers is characterized by runting, abnormal feather development, proventriculitis and diarrhoea. Wing feathers are abnormally big for chickens with retarded growth. They protrude At various angles, so the disease is termed **"Helicopter disease".** One-day old chickens are the most susceptible to the infection.

Gross lesions:

Swelling of the digital flexor and metatarsal extensor tendons, the hock joint usually contains a small amount of straw colored or tinted exudates, Edema of the tarsal and metatarsal tendon sheaths. Sometimes erosions in the articulate cartilage.



Diagnosis:

A presumptive diagnosis could be made on clinical signs and gross lesions.

Laboratory diagnosis:

Faeces, spleen and joints are probably the best sources of virus

1- Viruses isolation Embryonated chicken eggs: By inoculation on chorioallantoic (CAM) membrane or yolk sac, pock lesions could be seen 5-7 days post inoculation. The CAMs are then homogenized and used as antigen for precipitin test. Fluorescent antibody staining of CAMs sections confirms the virus detection.

2- RT-PCR

3- Serology:

- Group specific antibodies could be detected by Agar gel precipitation test. While indirect florescent antibody testing is more sensitive but expensive.
- Virus neutralization based on plaque reduction in chicken kidney and chicken embryo liver cells.
- ELISA systems are also used for detection of antibodies.

Prevention and control:

Sanitary and biosecurity measures:

Thorough cleaning of poultry houses. Because of relative stability of avian Reovirus, 0.5% organic iodine solution are considered to be effective.

Vaccination:

Living attenuated vaccine could be used by aerosol or subcutaneously starting on one day-old. The disadvantage is the interference with Marek's vaccination. Therefore **avirulent Reovirus** vaccine stains is more suitable for this age. Live reovirus vaccination is also used perior to killed virus vaccination in breeders.

Inactivated virus vaccine used for vaccination of breeders 4 weeks perior to lay. It confers protection of breeders in addition of maternal immunity transferred to progeny providing good levels of protection.

Pneumovirus infections

INTRODUCTION :

- Avian pneumoviruses (APV) are the cause highly contagious respiratory diseases of turkeys (TRT) and chickens (SHS) which may involve high morbidity and variable mortality.

- APV infections in turkeys and chickens are now virtually worldwide in distribution and of considerable economic significance, particularly in the turkey.

- The disease conditions can be induced by infection with *APV* alone, but the more severe forms of the disease probably resulted from dual or secondary infection with other respiratory affections as *Bordetella avium, Mycoplasma spp., Ornithobacterium rhinotracheale,* and *E.coli,* viruses as *Newcastle disease* and *Infectious bronchitis as* well as bad environmental factors.

ETIOLOGY:

- The subfamily *Pneumovirinae (Family Paramyxoviridae)* consists of two genera:
  - *Pneumovirus* – (pneumoviruses of mammals), the type species being *human respiratory syncytial virus*.
  - *Metapneumovirus* – (pneumoviruses of avian species), the type species being *turkey rhinotracheitis virus* (TRTV).

- APV is a pleomorphic, enveloped, RNA virus with an outer envelope bearing fusion (F) and glycoprotein (G) spikes.

- APV is lacking of haemagglutinating and neuraminidase activities.

- APV is sensitive to lipid solvents (ether and chloroform), inactivated at 56˚C after 30 minutes. Quaternary ammonia, ethanol, iodophor and a phenol derivative as disinfectants are effective in reducing of virus infectivity.

- Turkey or chicken embryos inoculated into yolk sac show embryo mortality after 4-5 passages, with low virus titer. Similarly, inoculation of turkey or chicken tracheal organ cultures results in ciliostasis, with a low virus replication. Adapted viruses could be propagated on chicken or turkey embryo cells, VERO cells, BS-C-1 cells, and quail tumor cell line (QT-35) producing a characteristic syncytium.

Economic significance:

- Drop in egg production in layers and breeders.
- Mortalities
- High cost of vaccination and treatment especially in complicated cases.

Epidemiology:

- **Natural and experimental hosts:** Turkeys and chickens at any age are known to be the natural hosts. Guinea fowl may be infected experimentally.
- **Infection and transmission:** APV infections are air borne in nature. The virus was established by direct and indirect contact with contaminated material.

TRT

Clinical signs:

- The severity of clinical signs is attributed to bad hygiene and complicating bacterial and viral infections. Morbidity in birds of all ages is usually up to 100%. Flock mortality usually is low as 0.4% to high as 50%, particularly in fully susceptible young poults. Mortality rate becomes severe and increases up to 90% under bad management and/or complications. Uncomplicated infections usually recovered between 10 – 14 days even without treatment.

- **Turkey poults (6-10 weeks of age):** Rapid onset with respiratory signs of (snacking, rales, sneezing, nasal watery or often frothy and later purulent discharge, eye discharge is watery and become frothy at the 5^{th} day), swollen infraorbital sinuses with submandibular edema.

- **Turkey breeders (laying birds):** Drop in egg production up to 70% for 2–3 weeks with poor shell quality, along with mild to severe respiratory signs. Subclinical infection may be detected by seroconversion.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Gross lesions:

- Rhinitis, foamy conjunctivitis, sinusitis, tracheitis, subcutaneous edema in submandibular area are the most predominant lesions.

- Various reproductive tract abnormalities including egg peritonitis, folded shell membranes in the oviduct, misshaped eggs, regression in ovary and oviduct and inspissated albumin and yolk are seen. Prolapsed oviduct may be also observed.

- Lesions of secondary infections (fibrinous airsaculitis, pericarditis, perihepatitis and pneumonia) can mask TRT lesions.

SHS

Clinical signs and Lesions:

- The severity of clinical signs is attributed also to bad hygiene and complicating bacterial and viral infections.

- Broilers (3-6 weeks of age):
  - The disease is more severe than in adult birds.
  - Swelling of the periorbital and infraorbital sinuses and eyelids (yellow gelatinous to purulent exudates in the subcutaneous tissues of the head, neck, and wattle is observed), coughing, sneezing, nasal and ocular discharge.
  - Secondary infection with E.coli involvement can result in severe caseous conjunctivitis, greenish diarrhea with bad odor and increased mortality rate up to 30% for 2-3 weeks.
  - Nervous manifestations as torticollis, cerebral disorientation, and opisthotonos due to extended inflammation from external ear to middle ear.
  - Morbidity is usually less than 4% while mortality rate rarely exceeds 2%.
- Breeders (30-52 weeks of age): There is a marked drop in egg production (40%) usually around the peak of egg production or before entry. oophoritis, salpingitis, ascites, and egg peritonitis are common. Respiratory signs or swelling of the head are usually less severe

Prevention of APV infections can be based on:

1. Hygienic measures:

- Adequate ventilation.
- Temperature control
- Good litter quality.
- Avoid multi-age stock and debeaking or vaccination with live vaccine at a critical time.
- Disinfection of equipments, and feed truck should be routine practice.
- For reduction of the severity of secondary bacteria usage of antibiotics is recommended.

2. Vaccination:

Two types of vaccine are commercially available for use in both turkeys and chickens:

A. Live attenuated APV vaccine: It stimulates both systemic and local immunity in the respiratory tract, also there's a good cross protection occurs following vaccination with subtype A and B vaccines.

B. Inactivated APV vaccine: Oil-adjuvant inactivated APV vaccines are administered to adult birds previously primed with live vaccines to confer complete protection against drop in egg production.

A typical vaccination program for APV in turkeys would be the application of three live vaccine doses (subtype A or B or both) given using a coarse spray at day-old, 7 to 10 days and at 4 to 6 weeks. Breeding stock would additionally receive inactivated vaccine at 16 to 20 weeks.

GOOSE PARVOVIRUS INFECTION (GPV)
(Derzsy's disease, goose influenza)

It is a highly contagious disease affecting young geese and Muscovy ducks. Depending on the age of affected goslings, the disease may be present in acute, subacute, or chronic forms. The acute form of the disease can result in 100% mortality in goslings less than 10 days of

Importance:
Losses are due to high mortality in geese and Muscovy ducks. There is a high cost of vaccination and prevention.

Cause:

- The causative agent is a parvovirus belonging to the family Parvoviridae
- Nonenveloped. GPV is very resistant to chemical and physical inactivation.
- With hexagonal single-stranded DNA,
- No hemagglutination activity,
- Replicates in the nuclei of cells from the heart and bursae of infected goslings.
- GPV has been isolated only in embryonated goose or Muscovy duck eggs or primary cell cultures prepared from the embryos. Embryo mortality occurs 5-10 days post inoculation with hemorrhages.
- Parvovirus from both geese and Muscovy ducks are antigenically related although differences between the genomes of Muscovy duck parvovirus and goose parvovirus are detected.

Epidemiology:

- Geese, Muscovy ducks, and some hybrid breeds are the only affected species.
- All breeds of domestic geese are susceptible.
- Other breeds of domestic poultry and ducks appear refractory.
- The disease is strictly age dependent; thus, 100% mortality may occur in goslings under 1 week of age, with negligible losses occurring in 4-5-week-old birds. Older geese do not show clinical signs but developed a latent infection and transmit virus in feces and eggs.
- Transmission: Infected birds excrete large amounts of virus in their feces, resulting in a rapid spread of infection by direct and indirect contact. The most serious outbreaks occur in susceptible goslings following vertical transmission of the virus.

Signs:

The clinical signs in susceptible goslings vary according to the age of the birds.

In goslings under 1 week of age:

The course of the disease may be very rapid with anorexia, prostration, and death occurring within 2-5 days.

Mortality can reaches 100% in goslings infected in the hatchers.

Mortality in 2-3-weekold goslings may be below 10%, although morbidity levels may be high.

Complicating factors such as poor management and secondary bacterial, fungal, or viral infections may influence the final mortality levels.

In older birds or those with variable levels of maternally derived antibody:

The disease follows a more protracted course with the appearance of clinical signs.

- Initially, affected birds exhibit anorexia, polydipsia, and weakness with a reluctance to move.
- There is a nasal and ocular discharge in many birds with associated headshaking.

- The eyelids are often red and swollen, and a profuse white diarrhea is evident in many birds.

- Examination of the birds at this stage may reveal a fibrinous pseudo membrane covering the tongue and oral cavity. Goslings that survive the acute phase may develop a more prolonged disease.

Chronic:

growth retardation,
loss of down around the back and neck, a
marked reddening of the exposed skin.
Ascitic fluid may be accumulated in the abdomen, causing the goslings to stand in a “penguin- like” posture. “

Latent form:
Goslings more than 4 weeks of age rarely show clinical signs, but pass in latency. Geese of all ages respond immunologically to goose parvovirus infection without necessarily showing clinical signs.


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Lesions:

- The heart, which has a pale myocardium, rounded at its apex.
- The liver, spleen, and pancreas may be swollen and congested.
- Cases with the more prolonged clinical course showed additionally a sero-fibrinous perihepatitis and pericarditis with large volumes of straw-colored fluid in the abdominal cavity.
- Pulmonary edema, liver dystrophy, and catarrhal enteritis may also be present.
- Hemorrhages can be seen in the thigh and pectoral muscles.
- Diphtheritic and ulcerative lesions may be observed in the mouth, and pharynx.

Diagnosis:

Clinical signs, lesions and isolation and Identification of Goose Parvovirus from a variety of suitable lesion and inoculation of 10-15 day-old embryonated goose or Muscovy duck eggs via the allantoic cavity or duck cell cultures.

The virus can be detected by electron microscopic examination or Immunofluorescence, neutralization with specific goose parvovirus antiserum.

Serologically, in breeder geese and ducks: The most widely used method is the VN test in embryonated goose or Muscovy duck eggs or primary cell cultures. AGPT and ELISA are also used.

Differential Diagnosis:

Disease with high mortality in geese and ducks as DVE, DHV, HNEG,
Pasteurella anatipestifer and Pasteurella multocida must be differentiated from this condition.

Treatment:

Serum therapy was widely used when the disease subsequently appeared using serum produced in geese. However, passive immunization was found to be expensive and time consuming, particularly as two doses of serum were often required to produce adequate immunity. Antibiotic is required to suppress secondary bacteria.

Prevention:

- Good farm and hatchery hygiene.
- Only eggs from known parvovirus-free flocks should be incubated together.
- All contact goslings or adults, should be serologically tested to identify which birds have been infected horizontally.
- Positive reactors should be removed from the flock as these birds may become carriers to the virus.

Vaccination:

Active immunization of adult breeding geese and Muscovy ducks with virulent virus to transfer antibodies to the progeny via the egg yolk.

The attenuated vaccines have been developed by attenuation of the virus in goose or Muscovy duck embryo cell cultures, for use in breeding geese and goslings. Duck embryo-adapted goose parvovirus vaccines have also been shown to induce a good immune response in goslings and breeder geese. Inactivated vaccines have been used in flocks of breeding geese and Muscovy ducks, inducing high levels of immunity.

Recombinant vaccines formulated in oil emulsion have been evaluated in goose and Muscovy duck laying flocks.

Thanks