Tikrit University
College of Veterinary Medicine

Lect. 7-Virology

Subject name: RNA Viruses families Birnaviridae and Rhabdoviridae
Subject year:2024-2023
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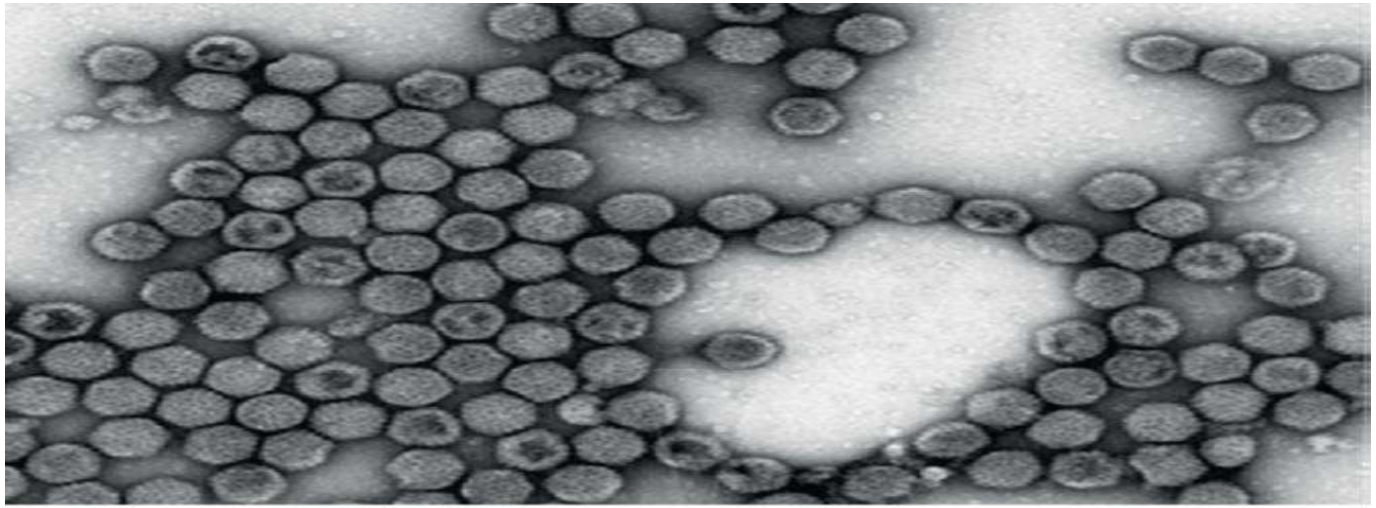
Birnaviridae

Viruses in the families Birnaviridae and Picobirnaviridae have nonenveloped, single-shelled virions that include a genome of two linear segments of double-stranded RNA.

Two members of the family Birnaviridae are economically significant pathogens, specifically, the etiologic agents of infectious bursal disease of chickens and infectious pancreatic necrosis of fish.

Properties of Birnaviruses
Virions of birnaviruses are nonenveloped, hexagonal in outline, approximately 65 nm in diameter, with a single shell having icosahedral symmetry; picobirnaviruses are smaller (35 nm) viruses with similar appearance
Genome consists of two molecules of linear double-stranded RNA, designated A and B, approximately 6 kbp in overall size for birnaviruses
A single major capsid protein, and one or more nonstructural proteins (RNA polymerase (transcriptase))
Cytoplasmic replication
Survives at 60°C for 60 min; stable at pH 3 to pH 9
Birnaviruses occur in chickens (infectious bursal disease virus), salmonid fish (infectious pancreatic necrosis virus), and many species of marine fish and shellfish (aquatic birnaviruses) as well as reptiles, crustaceans, and insects.



The viral mRNA is transcribed by a virion associated RNA-dependent RNA polymerase (transcriptase- VP1)

Genera of Birnaviridae:

1. Avibirna virus: - infectious bursal disease (Gumboro).
2. Aquabirna virus: - infectious pancreatic necrosis in salmonids (fish).
3. Entomobirna virus: - infect insects.

Infectious bursal disease (Gumboro).

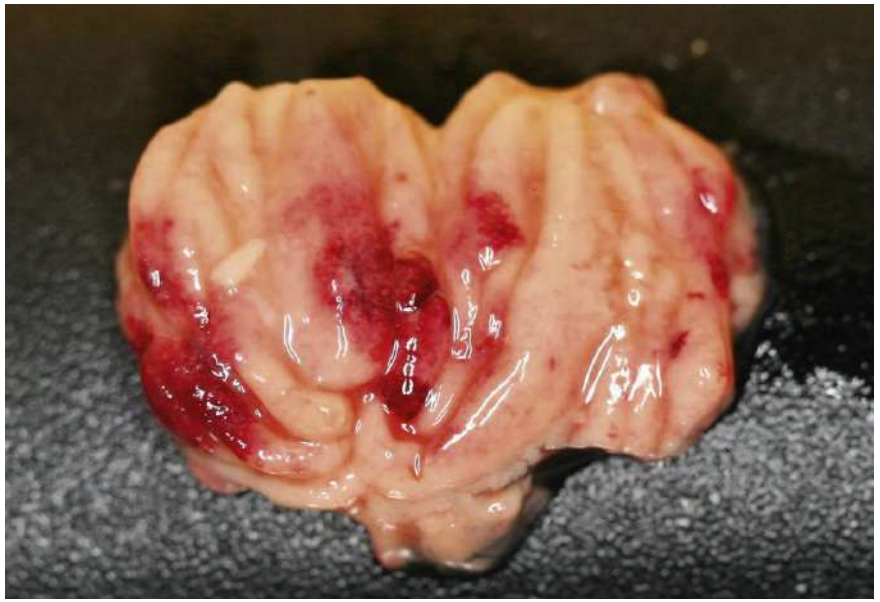
Infectious bursal disease (IBD) is a viral disease infected domestic chickens. Characterized by present clinical or subclinical disease, but immune suppression and related secondary infections are typically seen. Severity of the immune suppression depends on the virulence of the infecting virus and age of the host.

Etiology

IBD is caused by avibirnavirus that is most readily isolated from the bursa of Fabricius but may be isolated from other organs.

Two serotypes of IBDV have been identified.

1. **Serotype 1 viruses cause disease in chickens and, within them, antigenic variation can exist between strains.** Antigenic drift is largely responsible for this antigenic variation, but antigenic differences can also occur through genome homologous recombination.
2. **Serotype 2 strains of the virus infect chickens and turkeys but have not caused clinical disease or immunosuppression in these hosts.**



Infectious bursal disease:

Infectious bursal disease. Swollen, edematous(swollen with an excessive accumulation of fluid), and hemorrhagic cloacal bursa from an infected chicken, with superficial hemorrhage

Transmission

1-feces . 2-transferred from house to house by fomites(objects or materials which are likely to carry infection, such as clothes, utensils, and furniture) transmission occurs directly through contact and oral uptake

Clinical Findings:

IBD is highly contagious; results of infection depend on age and breed of chicken and virulence of the virus.

Infections may be subclinical or clinical.

Chickens are most susceptible to clinical disease at 3–6 weeks of age when immature B cells populate the bursa and maternal immunity has decreased.

1. **In clinical infections, beginning of the disease occurs after an incubation of 3–4 days. Chickens may exhibit severe prostration, incoordination, watery diarrhea, and inflammation of the cloaca.**
2. **Subclinical infections before 3 weeks of age are usually subclinical. subclinical disease has no or minimally recognizable clinical findings.**
3. **Severe infections have occurred in Leghorn chickens up to 18 weeks of age.They cause severe, long-lasting immunosuppression due to destruction of immature lymphocytes in the bursa of Fabricius, thymus, and spleen. The humoral (B cell) immune response is most severely affected; Immunosuppression is a reduction of the activation or efficacy of the immune system.**

Diagnosis:

1) clinical signs. 2) Viral isolation. 3) PCR to identify the viral genome in bursa tissue. 4) Titration of the virus and virus-neutralization assays.

Control : Live vaccines of chicken embryo can be administered by eye drop, drinking water, or SC routes at 1–21 days of age 2-oil-adjuvanted, inactivated vaccine.

Rhabdoviridae:- Rod

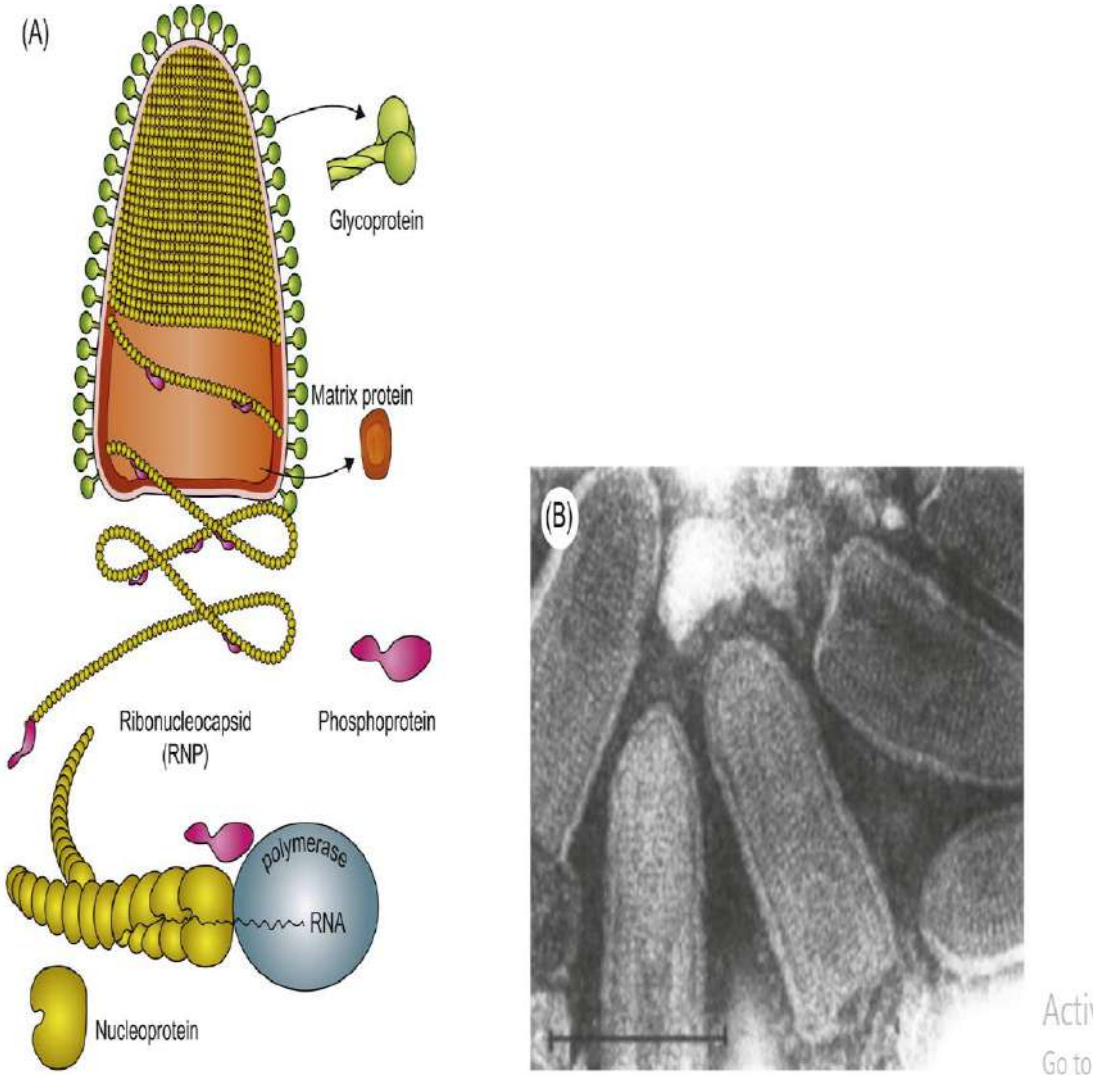
- The family Rhabdoviridae is ecologically diverse and includes viruses that infect a broad range of hosts, including mammals, birds, fish, insects, and plants.
- Some rhabdoviruses are transmitted by arthropod vectors.
- The family Rhabdoviridae contains important pathogens of mammals, notably rabies, vesicular stomatitis, and bovine ephemeral fever viruses(*Bovine ephemeral fever virus* (BEFV) is an arthropod-borne rhabdovirus that is classified as the type species of the genus *Ephemerovirus)*, and several economically important viruses of fish.
- There are also a large number of rhabdoviruses of uncertain pathogenic significance or importance that infect cattle, pigs, marine mammals, birds, bats, and reptiles.

Genera of Rhabdoviridae:

1. Lyssavirus:Rabies virus .
2. Ephemero virus:Bovine ephemeral fever virus.
3. Vesiculovirus: vesicular stomatitis virus.

Properties of Rhabdoviruses

Virions are enveloped, bullet shaped, 45–100 nm in diameter and 100–430 nm long (although some are longer, some shorter), and consist of an envelope with large spikes within which is a helically coiled cylindrical nucleocapsid
The genome is a single molecule of linear, negative-sense, single-stranded RNA, 11–15 kb in size
Cytoplasmic replication
Viral RNA-dependent RNA polymerase (transcriptase) transcribes five subgenomic mRNAs, which are translated into five proteins: (1) L, the RNA-dependent RNA polymerase (transcriptase); (2) G, the glycoprotein that forms the envelope spikes; (3) N, the nucleoprotein, the protein that associates with RNA to form the viral nucleocapsid; (4) P, a phosphoprotein that mediates binding of L protein to the nucleocapsid; (5) M, which associates with the viral nucleocapsid and lipid envelope
Maturation is by budding through the plasma membrane
Some viruses, such as the vesicular stomatitis viruses, cause rapid cytopathology, whereas others, such as unadapted rabies virus, are noncytopathogenic



Family Rhabdoviridae. (A) Diagram illustrating a rhabdovirus virion and the nucleocapsid structure. (B) Vesicular stomatitis Indiana virus showing characteristic bullet-shaped virions

Rhabdoviridae properties

- Rhabdoviruses are relatively stable in the environment, especially in cool moist environments and when the pH is alkaline—vesicular stomatitis virus can contaminate water troughs for many days for example—but the viruses are thermolabile and sensitive to the ultraviolet irradiation of sunlight.

- Rabies and vesicular stomatitis viruses are inactivated readily by detergent-based disinfectants, and iodine-containing preparations are commonly applied as disinfectants for reducing or eliminating fish rhabdoviruses such as those that occur on the surface of fish eggs.

Rabies virus

Rabies virus is a neurotropic virus that causes rabies in humans and animals. Most animals, the virus will spread through the nerves of the bitten animal towards the brain. Virus reaches the limbic system of the brain, where it replicates extensively,leading to the behavioral change. rabies virus spreads centrifugally from the central nervous system through peripheral nerves to a variety of organs, including the adrenal cortex, pancreas, and, most importantly, the salivary glands.

Transmission

1. **Usually the bite or scratch of an infected animal, which introduces the virus through the skin or mucous membrane. .**
2. **Aerosol transmission from an infected animal, usually a bat.**
3. **Tissue transplants (such as corneas) from infected humans.**

Clinical signs:

The virus is relatively slow moving and the average time of incubation from exposure to brain involvement is between 3 to 8 weeks in dogs, 2 to 6 weeks in cats, and 3 to 6 weeks in people.

However, incubation periods as long as 6 months in dogs and 12 months in people have been reported. After the virus reaches the brain it then will move to the salivary glands where it can be spread through a bite.

Diagnosis:

- Clinical signs.
- Microscopic examination : the presence of eosinophilic intracytoplasmic inclusions (Negri bodies) in neurons is characteristic and diagnostic, These spherical structures having a diameter of a few micrometers (2 to 10 μm) are found in the cytoplasm of some infected nerve cells. By electron microscopy observations, the Negri bodies were found to be composed by a matrix of granular or filamentous material consisting of viral nucleoprotein.
- Blood samples

Treatment,Vaccination and prevention

There is no treatment. Vaccination is the best way to prevent infection

Rabies virus proteins are highly immunogenic, and numerous different types of efficacious vaccines have been developed to protect humans and animals from rabies virus induced disease. Inactivated, live-attenuated and recombinant vaccines have been developed for the parenteral immunization of animals and humans against rabies.Successful oral vaccination against rabies has been achieved.

After the virus reaches the brain the animal will show one, two, or all of the three different phases

I. Prodromal phase

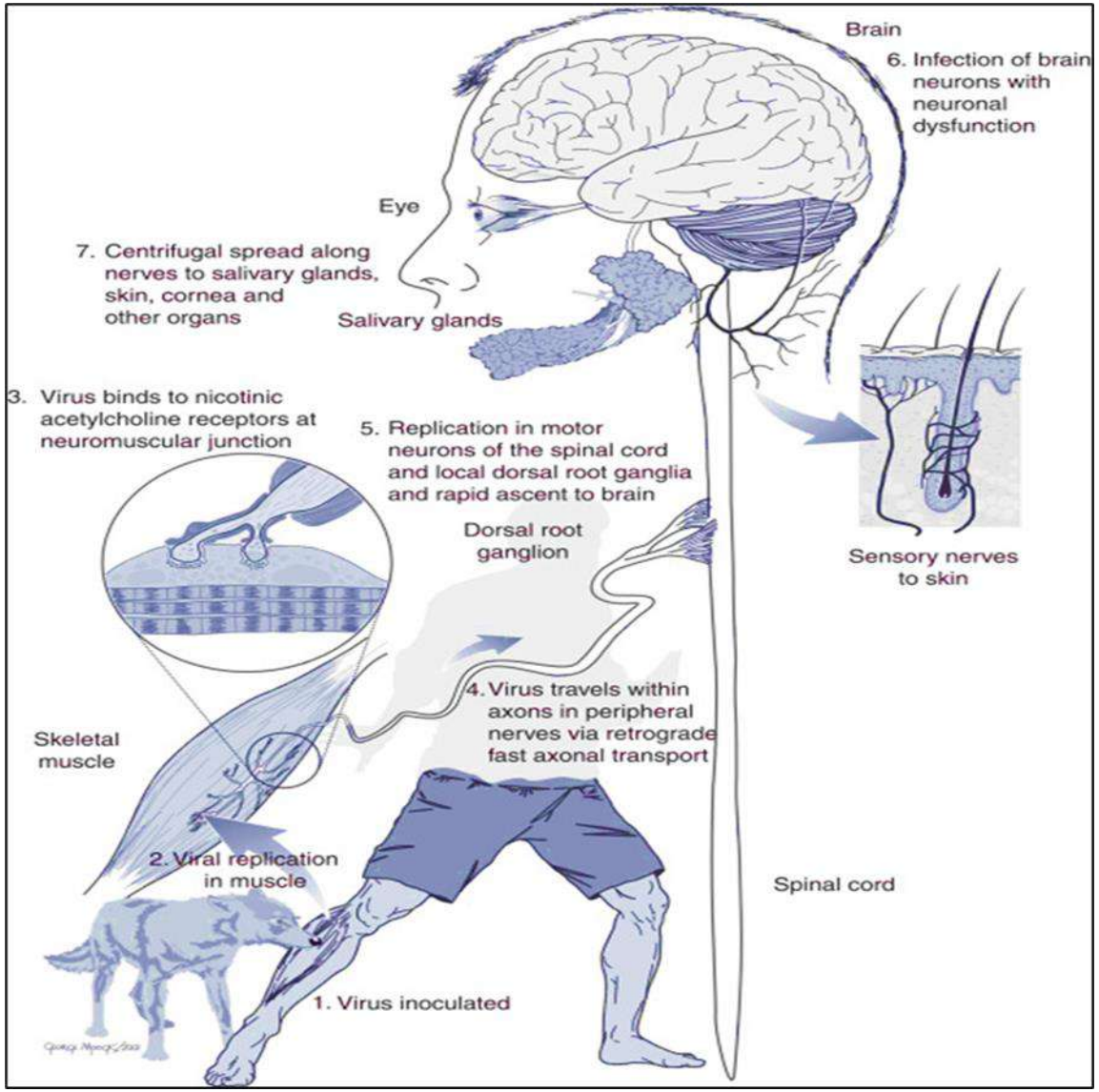
The first phase is the prodromal phase and usually lasts for 2-3 days in dogs. Apprehension(fear,worry) , nervousness, anxiety, solitude (isolation) the state or situation of being alone.
, and a fever may be noted. Friendly animals may become shy or irritable and may snap, whereas, aggressive animals may become affectionate(kind) and docile

II. Furious phase

From the prodromal phase, animals may enter the furious stage; cats are particularly prone to developing this phase. The furious stage (angry) of the disease in dogs usually lasts for 1 to 7 days. Animals become restless (troubled, nervous)and irritable(and are hyperresponsive to auditory and visual stimuli. As they become more restless, they begin to roam and become more irritable and vicious(malignant, malicious, evil). When caged, dogs may bite and attack their enclosures. Animals progress to become disoriented and then have seizures and eventually die .

III.Aralytic (dumb) phase

Animals may develop the paralytic phase either after the prodromal or furious stage. The paralytic phase usually develops within 2 to 4 days after the first signs are noted. Nerves affecting the head and throat are the first to be involved and animals may begin to salivate as a result of their inability to swallow. Deep labored breathing and a dropped jaw may result as the diaphragm and facial muscles become increasingly paralyzed. Animals may make a choking sound and many owners think that there is something lodged in the dog's throat. The animal will get weaker and eventually go into *respiratory* failure and die.



Schematic diagram showing the sequential steps in the pathogenesis of rabies after an animal bite/peripheral inoculation of rabies virus.