BIRNAVIRIDAE

Members of the virus family Birnaviridae possess two segments of linear double stranded RNA genome, an icosahedral capsid symmetry and are about 60nm in diameter. Five polypeptides designated VP1, VP2, VP3, VP4 and VP5 have been identified. The VP2 is the major capsid protein and contains epitopes which induce neutralizing antibodies. Birnaviruses replicate in the cytoplasm of the host cells and the process involves a virion-associated RNA-dependent RNA polymerase.

Classification

Family: Birnaviridae

Genera: 1. Avibirnavirus (infect chicken)

Virus: Infectious bursal disease virus

2: Aquabirnavirus (infect fish)

3. Entomobirnavirus (infect insects)

Birnaviruses are stable over a wide pH range and at temperature of 60^{O}C for one hour. They are resistant to ultraviolet irradiation and photodynamic inactivation. They are resistant to treatment with ether and chloroform. Infectious bursal disease virus is inactivated at pH 12.0 and by exposure to 1% phenol for 1 hour or 1% formalin for 1 hour. It is also inactivated by heat at 70^{O}C for 30 minutes. It does not haemagglutinate erythrocytes unlike Newcastle disease virus.

Two economically important diseases associated with birnaviruses are infectious bursal disease of chickens and infectious pancreatic necrosis of salmonids. These diseases occur worldwide and cause considerable losses in poultry units and in farmed salmons.

INFECTIOUS BURSAL DISEASE

Infectious bursal disease has been reported in most major poultry producing areas of the world. The greatest incidence is in chicks 3 to 8 weeks old. However, outbreaks have been reported in 9 days old chicks and in 20 weeks old layers. Infection which is usually acquired by the oral route occurs when maternally-derived antibody levels are waning at two to three weeks of age. Virus is shed in the faeces for up to two weeks after infection and can remain infectious in the environment of a poultry house for several months. Mortality is usually between 20 – 30% but could be as high as 37% while morbidity is usually high approaching 100%. The disease shows a spiking mortality curve. It is a self-limiting disese. Infection can spread to other poultry units via

formites. Vertical transmission and carrier state have not been demonstrated. Clinical infection occurs majorly in chicken but turkeys, ducks and guinea fowls may be infected.

Antigenic properties of IBDV:

Based on neutralization tests, isolates of IBDV are assigned to two serotypes:

Serotype 1: contains viruses that are pathogenic for chicken. There is considerable variation in the virulence of isolates in serotype 1. Very virulent (VV) strains of serotype 1 can cause disease even when maternally-derived antibody against the classical vaccine is present. These variants are classified as subtypes of serotype 1 because of their antigenic similarity. There are at least about six subtypes of serotype 1.

Serotype 2: these isolates infect chickens and turkey but these infections are of relatively low significance.

The two IBDV serotypes share group antigen detectable by IFT and AGID

Clinical signs of IBDV infection: the severity of clinical signs is influenced by the virulence of the virus, the age of chicks at the time of infection, the breed of the chicks as well as the level of maternally-derived antibody. An acute form of the disease develops following a short incubation period at about three to six weeks of age. Affected birds show signs of depression, loss of appetite, diarrhea and vent pecking due to irritation resulting from inflammation of the bursa of Fabricius. The disease is of very short course with surviving birds recovering within four days of onset of clinical signs.

Diagnosis:

CIRCOVIRIDAE

The virus family Circoviridae is a recently established virus family. Members cause diseases in vertebrate animals and plant. They are about 17 to 22nm in diameter. They are non-enveloped virus with icosahedral symmetry. Circoviruses are stable in the environment at pH 3 to 9 and are resistant to heating at 60 ^{O}C for 30 minutes. They possess a circular single stranded DNA genome.

There are three genera in the family circoviridae based on genomic composition:

1. Gyrovirus: chicken anaemia virus, type species of the family belongs to this genus
2. Circovirus: Porcine circovirus and beak and feather disease virus are members of this genus
3. Nanovirus: members of this genus have been removed from the family Circoviridae. They are plant viruses

Clinical Infections

Circoviruses are host specific and infect cells of the haemolymphatic system. Diseases of veterinary importance include:

i. Chicken anaemia virus infection
ii. Pig circovirus infection
iii. Beak and feather disease (a debilitating immunosuppressive disease of young pscittacine birds especially cockatoos)

Chicken anaemia virus infection

This disease can be transmitted by vertical and horizontal (faeco-oral) transmission. It causes aplastic anaemia and generalized lymphoid atrophy in young birds. Only chickens are susceptible.

Clinical signs: depression, anorexia, paleness, mortality of about 10% but may reach 50%. Other immunosuppressive diseases especially infectious bursal disease increase susceptibility.

Diagnosis:

- Clinical signs and gross postmortem lesions are suggestive.
- Laboratory confirmation is by viral antigen detection using immunocytochemical technique.
- Viral DNA can be detected in bone marrow and thymus samples using in situ hybridization, dot-blot hybridization or by polymerase chain reaction
- Serological tests include virus neutralization, indirect immunofluorescence and ELISA

Pig circovirus infection

This disease was first described as a picornavirus-like contaminant of the continuous pig kidney cell line (PK115). The virus is of doubtful pathogenicity. Another antigenically and genomically distinct circovirus, porcine circovirus 2 (PCV 2) has been isolated from piglets with wasting disease. Porcine circovirus 2 affects piglets of about six weeks of age causing post-weaning multisystemic wasting syndrome (PMWS), a progressive wasting condition with lesions in several organ systems.

Diagnosis:

- Clinical signs and pathological findings
- Antibodies detection by using indirect immunofluorescence or ELISA
- Virus isolation in pig cell line
- Demonstration of PCV 2 antigen antigen by immunocytochemistry or nucleic acid by molecular techniques

CALCIVIRIDAE

- Latin *calyx = cup*
- They have cup shape depression, demonstrated by electron microscopy on the surface of virion

- Virion are 27 – 40nm is diameter
- They have icosahedral symmetry and are non-enveloped
- Genome consist of a single molecule of linear, positive sense single stranded RNA
- Replication takes place in the cytoplasm and virion are released by cell lysis
- Many calciviruses have not yet been cultured
- They are resistant to heat but are sensitive to acid pH values
- Calicivirus are closely related to piconaviruses and were formerly grouped within the picornaviridae
- Genera: (4) two named, two unnamed (human)

1. Vesivirus: vesicular exanthema of swine virus

   Type species of the family

- San Miguel sea tion virus
- Feline calcivirus

2. Lagovirus

- Rabbit haemorrhagic disease virus
- European brown hare syndrome virus

3. Norwalk-like viruses: (referred to as small, round, structural virus group 1 and 2). They have fuzzy appearance and lack surface detail at electron microscopy. Cause gastroenteritis in human
4. Sapporo-like viruses: cause gastroenteritis in human

Vesicular Exanthema of Swine

- First reported in the USA (Southern California) in 1932
- Widely spread in the USA in the 1950s and eradicated in 1959
- A highly contagious, acute disease similar to foot-and-mouth disease
- Caused by VESV
- Reservoir of virus exists in marine animals
- Probably occur due to feeding sea lion and seal meat contaminated with SMSV to pigs
- Strains of SMSV produce VES in pigs experimentally inoculated
- Antigenic heterogeneity: 13 serotypes of VESV and 17 of SMSV

C.S

- Incubation period about 72 hours
- Course of disease is about two weeks
- Vesicles develop on the tongue, lips, snout, interdigital spaces and coronary ban
- Fever, lameness
- Weight loss
- Neonatal mortality
- High mobidity, low mortality
- Differentials: FMD, vesicular stomatitis, swine vesicular disease

DX

- Samples: vesicular fluid, epithelial layer/scrapings
- Isolation of virus in pig kidney cell lines
- Identification of isolates by ELISA, CFT and immunoelectronmicroscopy

Feline Calcivirus Infection

- Aetiology Feline calcivirus
- Account for about 40% of upper respiratory tract inflammatory disease in cats worldwide
- All felidae susceptible, natural infection more in domestic cats and captive cheetahs
- Incubation period: up to 5 days
- Clinical signs confined to upper respiratory tract and the conjunctivae
- Fever, oculonasal discharges, conjunctivitis, vesicles on the tongue and oral mucosa
- Lameness and stiff gart at acute phase
- High morbidity and mortality

DX

- C.S, upper respiratory signs, ulcer on the oral mucosa
- Differential: feline herpesvirus 1 infection (most severe infection)
- Sample: oropharyngeal swat, lung tissue
- Isolation of feline cell line
- Isolation may not connote active disease because of wide carrier state in cats

- Demonstration of a rising antibody titre in paired serum samples necessary for laboratory confirmation

CX

- management practices to reduce exposure to virus
- vaccination
    - inactivated vaccine for parenteral administration
    - modified live vaccine for parenteral and intranasal administration
- vaccination does not prevent subclinical infection and carrier state but protects against clinical disease
- live vaccine may cause clinical infection (if given by other routs apart from injection)

Rabbit Haemorrhagic Disease

- highly contagious, acute often fatal diseas of European rabbits (*Oryctolagus cuniculus)*
- first reported in china (1984) and has since been encountered in many parts of the world
- occur in rabbits above two months of age
- RHDV is considered a mutant of a non-pathogenic virus, rabbit calcivirus known to be endemic in commercial and wild rabbits in Europe for may years
- RHDV has been used as a biological control for rabbit populations in Australia and New Zealand
- Transmission: faeco-oral, inhalation, through the conjuctiva. Mechanical transmission by mosquitoes and fleas and other insects has been demonstrated. Indirect transmission through formites and foodstuff. Close contact required

C.S

- Incubation period is up to three days
- Characterised by high morbidity and high mortality
- The course in short, death occur within 36 hours of infection
- Virus target cells of the mononuclear, phagocyte lineage
- Rabbits under two months are not susceptibl

- There is severe hepatic necrosis and there may be disseminated intravascular coagulation
- Pyrexia
- Depression
- Increased respiratory rate
- Serosanquinous nasal discharge
- Haematuria
- Neurological signs including convulsion
- Some animals may survive for a few weeks with jaundice, weight loss and lethargy

DX

- High mortality and with characteristic gross lesions (hepatic necrosis, congestion of liver, lung, spleen)
- Virus culture is different and usually unsuccessful
- Confirmation is by electron microscopy, demonstration of antigen by ELISA, IF or haemmagglutination using human erythrocytes
- Demonstration of virus-specific antibodies by ELISA and haemagglutination inhibition test. Reverse transcriptase PCR for detection of RHDV nucleic acid

CX

- Vaccination in endemic regions
- Inactivation and adjuvanted given at 10 weeks of age
- New vaccine based on recombinant eyxoma virus expressing RDHV capsid protein and virus-like particles from capsid protein produced in baculovirus expression system are being developed

TOGAVIRIDAE

Latine Toga=cloak

Togavirus are enveloped RNA viruses approximately 70nm in diameter with icosahedral symmetry. The envelope contains glycoprotein spikes and is closely bound to the capsid. They agglutinate goose and chick erythrocytes

There are two genera in the family Togaviridae:

1. Alphavirus: virus of veterinary importance
2. Rubivirus: only one member; rubella virus which causes German measles in children and young adults

Alphavirus are divided into a number of groups based on their genomic composition. These groups are:

1. Venezuelan equine encephalitis virus (VEEV) complex
2. Eastern equine encephalitis virus (EEEV) complex
3. Semliki forest virus complex
4. western equine encephalitis (WEEV) complex

western equine encephalitis virus has been shown to have originated by a recombination between EEEV and Sindbislive viruses

Alphaviruses have positive sense, single-stranded RNA genome and replicate in the cytoplasm. The nucleo capsids are assembled in the cytosol. They are released from infected swat cell by cytolysis. The viral envelope is composed of virus-derived glycoprotein spikes expressed on the host cell plasma membrane.

Viral infection of invertebrate cell is usually non-cytolytic and is persistent. In this case, virus assembly occurs in association with intracellular membranes rather thatn through the plasma membrane.

Alphavirus are sensitive to pH changes, heat, detergents and disinfectants. They are not stable in the environment.

Alphaviruses and certain members of the flaviviridae, Reoviridae, Rhabdoviridae and Bungaviridae are arthropod-borne and are thus termed “arboviruses”.

Domestic animals and humans are usually considered to be ‘dead-end’ hosts of alphaviruses because they do not develop a significantly high titre of circulating virus to act as reservoir hosts.

A number of important equine diseases are caused by the alphaviruses. The three equine encephalitis viruses (Venezuelan, Eastern and Western) are confined to the western hemisphere and are transmitted by mosquitoes. Getah virus occurs mainly in south-east asia and Australia. The three equine encephalitis viruses produce similar clinical signs but infections by Western equine encephalitis virus tend to be milder.

Eastern equine encephalitis virus: mosquito (*Culliseta melanura*) and Aedes species

VEEV: mosquito (culex species)

WEEV: mosquito (cules tarsalis and other culex species, Aedes species)

Getah virus: mosquito

Clinical signs of Equine Encephalitis

The clinical manifestatui if the three equine enceohalitis viruses are similar. Incubation period may be up to nine days and clinical signs may last from four to nine days. Clinical signs range from mild fever and depression to fatal febrile encephalomyelitis. Some of the neurological signs commonly observed include photophobia, blindness, head pressing, circling, ataxia and difficult deglutition. Terminally, animals become recumbent and semi-comatose with convulsion prior to death. The case fatality rate is 90% for EEE, 50% - 80% for VEE and 20% -40% for WEE.

Diagnosis

- clinical signs and history of previous cases of equine encephalitis in the same region may be suggestive
- laboratory confirmation:
    - virus isolation carried ut in cell culture or in suckling mice whole blood or serum collected during the pyrexic phase of the disease is suitable for virus isolation. Brain and/or cerebrospinal fluid collected at PM are also suitable for virus isolation. In cases of VEE, isolates should be typed to distinguish virulent from non-virulentsubtype
    - EEEV antigen in fixed brain section can be detected by an immuno histochemical staining technique
    - WEE and EEE are usually diagnosed by serological assays. A rising antibody tire is demonstrated in paired serum samples. ELISA, phage reduction neutralization assay, haemagglutination inhibition and compliment fixation tests are usually employed for serology.

An IgM capture ELISA has been used to provide evidence of infection in single serum samples. The vaccination status of an animal must be considered in interpreting the result of serological tests. The interpretation of serological results for VEEV is complicated by the presence of antibodies produced in response to inapparent infection with non-virulent subtypes.

Treatment and Control

- Suppurative palliative treatment may be beneficial but prognosis is poor
- Effort should be made to control mosquito population
- Horses should be housed in netted stables
- Monovalent, bivalent and trivalent vaccines are available. Vaccines for EEE and WEE are inactivated. A live attenuated TC-83 VEEV vaccine provides affective protection and has been used successfully to prevent epizoonotics of VEE
- It should be noted that the alphaviruses produce zoonotic infections.