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Paramyxoviridae

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This is a family of large, negative-strand RNA viruses. The two subfamilies comprise several genera with important veterinary and human pathogens, including canine distemper, rinderpest, and Newcastle disease. Respiratory syncytial virus is the major cause of croup in babies and of respiratory diseases in calves.

Viral Characteristics

- Viruses of Paramyxoviridae are enveloped - many are pleomorphic - and have a helical nucleocapsid (see Fig. 18.1).
- Genomes are single-stranded, negative-sense, non-segmented RNA.
- The envelope is covered with spikes whose glycoproteins are responsible for hemagglutinin, neuraminidase, and hemolytic activities.
- Replication takes place in the cytoplasm and envelopment at the surface (plasma membrane) of infected cells.
- The ssRNA (-) is used as a template for the production of mRNA (+) and progeny genomes.
- The viruses are generally sensitive to heat, drying, lipid solvents and many disinfectants.
- Most can be propagated in embryonated eggs and in cell cultures with the production of cytopathic effects including syncytia and cytoplasmic inclusions.

Figure 18-1. Paramyxoviridae. The virions are enveloped, with a helical nucleocapsid. The virions are highly pleomorphic; spherical and filamentous forms are observed. - To view this image in full size go to the IVIS website at www.ivis.org . -

Classification

This family contains two subfamilies, Paramyxovirinae and Pneumovirinae.
Paramyxovirinae is comprised of three genera, which with diseases, are as follows:

Respirovirus
Bovine parainfluenza 3

Morbillivirus
- Distemper
- Rinderpest
- Peste des petits ruminants
- Phocine distemper virus is the cause of a canine distemper-like disease in harbor seals.
- Dolphin distemper virus is the cause of a canine distemper-like disease in dolphins.

Henipavirus
- Hendravirus (formerly equine morbillivirus)
- Nipah virus

Rubulavirus
- Porcine rubulavirus infection
- Canine parainfluenza virus 2

Avulavirus
- Newcastle Disease virus
- Avian paramyxovirus

Pneumovirinae comprises two genera:

Pneumovirus
- Bovine respiratory syncytial virus infection

Metapneumovirus
- Turkey rhinotracheitis

Respirovirus

Bovine Parainfluenza-3

Cause

Bovine parainfluenza virus 3 (BPI-3).

Occurrence

Bovine parainfluenza 3 virus infections occur in cattle and sheep worldwide.

Transmission

Droplet infection, direct and indirect contact.

Clinical & Pathologic Features

The virus replicates in macrophages and alveolar epithelium; the mucociliary mechanism is adversely affected. Damage to alveolar macrophages lowers defense against bacteria.

BPI -3 virus is widely prevalent and most cattle have antibodies as a result of frequent exposure. Seroprevalence up to 90 - 95% is common in beef and dairy cattle. Initial exposure generally results in subclinical or mild respiratory infection. Environmental stresses, including those incidental to transportation with over-crowding and exposure to extremes of temperature, may lead to secondary bacterial infections with resulting pneumonia. The most important complicating bacteria are Mannheimia (formerly Pasteurella) haemolytica and Pasteurella multocida. This complex of a predisposing viral infection or other primary agent with subsequent bacterial infection is often referred to as "shipping fever" or bovine pneumonic pasteurellosis.

With secondary complications, a severe lobular pneumonia may develop with characteristic clinical signs. If untreated this complication is often fatal.

Diagnosis

- Clinical specimens: Nasal and tracheal swabs, transtracheal wash and lung; acute and convalescent sera.
- The virus can be isolated in cell cultures of bovine origin in which it produces cytopathic effects characterized by giant cell formation, cell rounding, syncytia and the production of both cytoplasmic and intranuclear inclusions.
- Examination of cryostat sections of lung tissue by immunofluorescence provides for a rapid diagnosis of BPI-3 infection in dead animals. This can also be done in slides prepared with cells obtained by scraping the nasal mucosa in live animals.
- A fourfold increase in antibody titer (virus neutralization, hemagglutination inhibition, ELISA, or indirect immunoflorescence) indicates that an infection has been sustained. Many cattle possess antibodies to BPI-3 virus; thus, paired sera are important.

Prevention

- Killed and live attenuated BPI-3 virus vaccines of cell culture origin are available, usually in combination with other viral and bacterial antigens.
- Efforts should be made to minimize stress associated with marketing by allowing calves to adjust to weaning and dehorning before they are subjected to the rigors of sale barns, stockyards, transportation and feedlots.

- Antimicrobial drugs are used to control secondary bacterial infections.

Morbillivirus

Canine Distemper

Cause

Canine distemper virus. It is antigenically closely related to the rinderpest and measles viruses.

Occurrence

Canine distemper occurs frequently worldwide. In addition to dogs, wolves, foxes, coyotes, raccoons, ferrets, mink, weasels, dingoes, and skunks are susceptible. Some additional susceptible wild and exotic animals are listed in Table 18.1. Mild influenza-like infections occur in humans.

Table 18-1. Some Exotic and Wild Animals Affected by Both Canine Distemper and Feline Panleukemia Virus and/or Its Host Range Variants*

Canidae	Procyonidae	Mustelidae
Coyote	Bassariscus	Ferret
Dingo	Coati	Fisher
Domestic	Kinkajou	Grison
Fox	Lesser panda	Marten
Jackal	Racoon	Mink
Wolf		Otter
Cape hunting dog		Sable
Raccoon dog		Wolverine
		Badger
		Skunk

*Host range variants of feline panleukemia virus include canine parvovirus, mink enteritis, and raccoon parvovirus.

Transmission

Spread is by direct and indirect contact and the mode of infection is by ingestion or inhalation (droplets). Food, water, litter, etc., are readily contaminated with infectious discharges and secretions.

Pathogenesis

The virus replicates in the upper respiratory tract, tonsils and bronchial lymph nodes. A macrophage-associated viremia follows, infecting general lymphoid tissue. In the absence of an adequate immune response the virus infects the major systems including the CNS. Virus replication can damage immune cells resulting in immunosuppression.

Clinical & Pathologic Features

Canine distemper is usually an acute, febrile disease, especially of young dogs, although older unprotected dogs are also susceptible. The first clinical manifestation of distemper is a diphasic febrile response. The first response may be overlooked, but the second generally occurs 2 - 3 days later in conjunction with other clinical signs, which initially include congested conjunctiva and nasal mucosa with subsequent serous to mucopurulent discharges. Pneumonia, depression, anorexia, vomiting, and diarrhea usually follow. Neurologic disturbances, such as neuromuscular tics, "chewing gum" seizures, and paresis are frequent sequelae in dogs that recover from acute disease.

Hyperkeratosis of the nose and digital pads ("hard pad") develops in some cases. Pustular dermatitis may be seen affecting the abdomen of puppies.

Gross necropsy lesions characteristic of pneumonia and enteritis may be present. Thymic atrophy may be noted in young dogs. Microscopic lesions are widespread in visceral organs and the brain and characteristic viral inclusion bodies are commonly found in brain, lung, stomach, and urinary bladder.

Dogs that recover may years later develop what has been called "old dog encephalitis" as a result of a persistent infection. This manifestation is usually recurrent, with a few to several episodes of neurological manifestations within weeks to

months that usually end with death of the dog.

Diagnosis
- Clinical specimens: Conjunctival scrapings, blood (buffy coat) smears, lung, urinary bladder, stomach, and brain.
- A laboratory diagnosis may not be feasible. A presumptive diagnosis is frequently made on the basis of clinical signs in a young unvaccinated dog. Nonetheless, the vaccinated status does not assure protection since many cases of distemper have been reported in well vaccinated dogs.
- A reliable way to diagnose canine distemper is the demonstration of viral infected cells by immunofluorescence. Examination of conjunctival scrapings and blood smears is useful during early stages of the illness, but false negative results are likely to occur as the disease progresses. Tests are accurate when performed on appropriate necropsy tissues.
- Microscopic lesions of demyelination in the cerebellum and characteristic inclusion bodies in various tissues are diagnostically significant. The inclusions are primarily intranuclear in the brain and intracytoplasmic in other tissues.
- The prognosis is poor for dogs with CNS involvement.

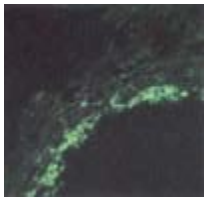

Figure 18-2. Canine distemper virus immunofluorescence in frozen section of canine lung. *Courtesy of A. Wayne Roberts.* - To view this image in full size go to the IVIS website at www.ivis.org . -

Treatment
- Supportive treatment, and antimicrobial therapy to cope with secondary bacterial respiratory infection.

Prevention
- Modified live vaccines are administered to dogs between six and 16 weeks of age, usually at 2 - 3 week intervals. This multiple dose regimen is necessary because the maternal antibody in puppies greatly hampers the efficacy of vaccination by neutralizing viral antigen.
- Dogs older than three months with unknown immune status should be vaccinated twice, 2 - 4 weeks apart, all dogs should receive periodic (one to two years-interval) boosters.
- Pregnant bitches should not be vaccinated with modified live vaccines.

Rinderpest

Cause

Rinderpest virus. It is closely related to the viruses of canine distemper, peste des petits ruminants and measles. There is considerable variation in the virulence of virus strains. Experimental infection of cattle with some highly virulent strains is uniformly fatal.

Occurrence

Although sheep, goats, swine, deer, yak, camels, hippopotamus, warthog, and other wild animals are susceptible to rinderpest, cattle and water buffaloes are mainly involved in outbreaks.
The disease occurs in remote parts of Asia and Africa, and for centuries has been the cause of enormous losses of cattle and buffaloes. It has not occurred in North America, and has not been reported in Europe since World War I.

Transmission

Spread is usually by food, water, and litter contaminated with secretions and excretions from infected animals. Infection is by inhalation or ingestion. Virus is shed in large amounts in secretions and excretions during the clinical phase of the disease.

Pathogenesis

Initial replication occurs in the mandibular and pharyngeal lymph nodes. Viremia follows in about three days with infection of the mucous membranes of alimentary and respiratory tracts, and lymphoid tissue in general.

Clinical & Pathologic Features

Rinderpest is a highly fatal, contagious, febrile disease seen mainly in cattle and water buffalo. The disease is usually less severe in other hosts and subclinical infections occur in swine.
The incubation period is 4 - 9 days followed by high fever and acute inflammation of the mucous membranes of the digestive and upper respiratory tracts. There is a mucopurulent nasal discharge and ultimately a watery diarrhea with blood-stained feces, a characteristic arching of the back, and rapid loss of condition. Damage to lymphoid cells results in leukopenia and immunosuppression. Convalescence in surviving animals is slow.
A milder disease may be seen where the disease is endemic and less susceptible species are involved. Characteristic

necropsy lesions are congestion and hemorrhage of intestinal epithelium with occasional necrosis of Peyer's 'patches. Erosions are found in the mouth and upper respiratory tract.

Diagnosis
- Clinical specimens: urine, blood, nasal discharge, feces, lymph nodes, and spleen collected in the acute phase of the disease; serum from surviving animals.
- The severe disease is frequently diagnosed clinically in endemic regions.
- Isolation and identification of the virus in cell cultures. Cytopathic changes, including cytoplasmic and nuclear inclusions, are produced in primary bovine kidney cell cultures and in the Vero cell line.
- Detection of antigen in extracts of tissues by immunofluorescence, immunodiffusion and counter immunoelectrophoresis.
- Virus neutralization tests in cell cultures can be performed on the sera of animals that have survived long enough to produce antibodies.

Prevention
- This is a reportable disease in most countries and a strict quarantine and slaughter policy is followed when the disease is confirmed. A global eradication plan is being coordinated by the Food and Agricultural Organization of the United Nations.
- Modified live vaccines are employed where the disease is endemic.

Peste des Petits Ruminants
(Pseudorinderpest)

Cause
Peste des petits ruminants virus (PPRV), a Morbillivirus. It is closely related antigenically to the virus of rinderpest.

Occurrence
The disease occurs in sheep and goats in West Africa.

Transmission
Spread is by direct contact with infected animals and by indirect contact with food, water, litter, etc., contaminated with secretions and excretions of infected animals.

Clinical & Pathologic Features
The virus of PPR causes a disease in sheep and goats clinically similar to rinderpest. Following an incubation period of about five days, infected animals become febrile and anorectic. They quickly develop a necrotic stomatitis, and nasal and ocular discharge, followed by severe diarrhea. Goats are more severely affected than sheep, and the mortality rate may approach 90% in young kids. The overall mortality rate varies from about 10 to 90%.
The virus of PPR causes a subclinical infection of cattle.

Diagnosis
- Clinical specimens: Oral lesions, whole blood, spleen, intestine, and acute and convalescent sera.
- A presumptive diagnosis is made on the basis of clinical signs and history in endemic regions. Confirmation requires isolation and identification of the virus or the demonstration of a significant increase in antibody levels between acute and convalescent sera.
- The virus can be cultivated in a variety of cell cultures, including Vero cells, in which cytopathic changes include syncytia, cytoplasmic and intranuclear inclusions.
- The virus of PPR can be differentiated from the virus of rinderpest by cross neutralization tests.

Prevention
- Like rinderpest, this is a reportable disease in many countries and is dealt with similarly.
- Vaccines are not available for PPR, but vaccines prepared with the closely related rinderpest virus are effective and are used in endemic regions.

Henipavirus
Hendra Virus Infection
(Acute equine respiratory syndrome, equine morbillivirus pneumonia)

Cause
Hendra virus, closely related to the Nipah virus.

Occurrence
The disease occurs infrequently and has only been reported in horses and humans in Australia.

Transmission
The reservoir of the virus is fruit bats (Pteropus spp.), which occur in Australia and Papua New Guinea. The infection in bats is subclinical. The virus is present in secretions and urine and spread is presumed to be by direct or indirect contact and aerosol. Vertical transmission is also thought to occur. The disease is not highly contagious.

Clinical & Pathologic Features
The principal feature of the disease is an interstitial pneumonia varying in severity. Clinical signs are mainly those of a respiratory infection and they include fever, anorexia, respiratory distress and a characteristic frothy, sometimes blood-tinged, nasal discharge. Wide spread edema and neurologic signs may also be evident. The fatality rate may exceed 60%. The virus mainly targets the vascular system, which begins in the lungs then spreads by viral-infected macrophages to other organs and in some animals to the brain. The virus attacks the vasculature of various tissues and organs including the spleen, liver, kidneys, myocardium and brain.

Diagnosis
- Clinical specimens: Paired sera; lungs, spleen, liver, lymph nodes and brain.
- The virus can be isolated in a number of cell lines and identified by virus neutralization.
- The virus can be detected in tissues by PCR.
- Testing acute and convalescent sera by ELISA or virus neutralization.
- Histopathologic examination and staining of tissues with labeled Hendra virus antiserum.

Prevention
- The disease has been controlled by quarantine and slaughter of all infected animals.
- Given the omnipresence of the virus reservoir there is little that can be done to prevent infections in horses.

Public Health Significance
- The disease in humans, characterized by nonsuppurative encephalitis or interstitial pneumonia, has been frequently fatal.
- Special precautions should be taken to prevent exposure to potential sources of the virus.

Nipah Virus Infection
(Barking pig syndrome, porcine respiratory and neurologic syndrome)

Cause
A recently discovered paramyxovirus referred to as Nipah virus, which is closely related to Hendra virus.

Occurrence
The virus was first recovered from humans with encephalitis in Malaysia and Singapore in 1998 - 1999. It was determined that those infected had been exposed to infected pigs. There is evidence that in addition to swine and humans, horses, dogs and cats are susceptible. The reservoir of the virus is considered to be fruit bats of the genus Pteropus that occur widely in southeast and south Asia.
The outbreaks in Malaysia and Singapore were controlled and there have been no further outbreaks in swine.

Transmission
After introduction of the virus to swine herds spread is rapid and presumably by direct and indirect contact. Further evidence of the high infectivity of the virus was rapid spread among herds.

Clinical Features
A febrile respiratory infection with rapid, labored breathing develops in many pigs in a herd. This gives rise to a severe characteristic cough and thus the name barking pig syndrome. Less common than the respiratory form was an encephalitis seen mainly in boars and sows.
The mortality rate in pigs (> 5%) was lower than that in humans.

Diagnosis
- Isolation of the virus in cell cultures and identification of the virus by serologic means, including virus neutralization and immunofluorescence. Syncytia are produced in Vero cells.
- Viral antigens in tissues can be identified by chemically tagged specific antibodies.

Prevention
- The disease was controlled in Malaysia and Singapore by strict quarantine of premises and slaughter of all the swine in affected herds.

- Because of the bat reservoir of the virus constant surveillance for evidence of the disease must be exercised.

Rubulavirus

Porcine Rubulavirus Infection

(Swine blue eye disease)

Cause

Porcine rubulavirus.

Occurrence

Porcine rubulavirus infection (PRI) of swine was first reported in Mexico in 1980 and still occurs there. It has not been reported as occurring outside of Mexico.

Transmission

The disease is acquired by direct and indirect contact (fomites).

Clinical & Pathologic Features

PRI or swine "blue eye" disease is most severe in pigs younger than three weeks and is characterized clinically by sudden onset of fever, depression, and progressive CNS signs. Affected pigs are weak and ataxic, and may have rigidity of the hind legs and tremors. Dilated pupils, nystagmus, and conjunctivitis may be noted in some pigs, and approximately 1 - 10% of affected pigs develop corneal opacity. The mortality rate may approach 90%.

In older pigs, clinical signs are principally respiratory including sneezing and coughing, and anorexia and fever. Neurologic signs may be seen in pigs more than 30 days old. The mortality rate is usually low. Most infections in adult swine are subclinical; corneal opacity is occasionally observed.

Reproductive problems may occur in pregnant sows. Orchitis and epididymitis may occur in boars resulting in reproductive failure.

Gross necropsy lesions are minimal and nonspecific. Microscopic lesions are those of a nonsuppurative encephalomyelitis and interstitial pneumonitis.

Diagnosis

- Clinical specimens: Brain, lungs, tonsils and affected eyes.
- A presumptive diagnosis has been made on the basis of clinical signs and histopathologic lesions. Epidemiological data is supportive.
- Confirmation requires isolation and identification of the virus. The virus can be cultivated in cell cultures of swine origin (PK-15 cell line) in which it produces syncytia. It is identified by virus neutralization.
- The virus agglutinates erythrocytes of various animal species including the chicken.
- Serological testing of paired sera using hemagglutination-inhibition and ELISA.

Prevention

- This is a reportable disease.
- Prevention is best accomplished by maintaining closed herds. All replacement animals should be isolated and serologically tested.
- Inactivated vaccines have been used.

Canine Parainfluenza Virus 2

This rubulavirus causes subclinical to usually mild respiratory infections in dogs. It may be part of the etiology of kennel cough, together with other microbial agents, including Bordetella bronchiseptica, canine adenovirus, canine herpesvirus, reovirus, mycoplasmas and possibly others.

Diagnosis of kennel cough is based on history and clinical signs. Canine parainfluenza virus 2 may be included in a multiple component vaccine given to dogs prior to exposure in boarding kennels, shows, etc.

Avulavirus

Newcastle Disease

Cause

Newcastle disease virus (avian paramyxovirus type 1). This virus comprises seven or eight closely related antigenic varieties. Each variety causes a characteristic disease manifestation. Alexander and Jones [*] refer to five pathotypes as follows:

1. Viscerotropic velogenic NDVs cause a highly virulent form of disease in which haemorrhagic lesions are characteristically present in the intestinal tract;
2. Neurotropic velogenic NDVs cause high mortality following respiratory and nervous signs;
3. Mesogenic NDVs cause respiratory and sometimes nervous signs with low mortality;
4. Lentogenic respiratory NDVs cause mild or inapparent respiratory infection;

5. Asymptomatic enteric NDVs cause inapparent enteric infection.

However, such groups should be regarded only as a guide as there is always some degree of overlap and some viruses are not easily placed in a specific pathotype.

Occurrence
Newcastle disease (ND) is a highly contagious, worldwide malady that affects chickens, other fowl, and wild and caged birds. It is reported that more than 250 avian species are susceptible to natural or experimental infection.
Caged birds were the source of the 1970 - 1972 epidemic of velogenic viscerotrophic ND in the US. Pigeon-associated ND epidemics occurred worldwide in the 1980s.

Transmission
The disease is mainly spread by infective feces but also by droplet infection, fomites and virus-infected eggs.

Clinical & Pathologic Features
The incubation period is about five days. Clinical signs vary greatly depending on the virus strain (and the avian species), from subclinical infections (lentogenic strains), to acute respiratory disease with central nervous system signs (mesogenic strains), to severe generalized infections with high mortality rates (velogenic strains). Infection with mesogenic strains is characterized by sudden onset, dullness, with coughing and sneezing and reduced egg production. Clinical signs are usually mild in adult chickens with few if any deaths, but young chicks may be severely infected with mortality as high as 50%. Neurologic signs may also develop in young chicks shortly after respiratory signs appear and include wing droop, abnormal positioning of the head and neck, and paralysis.
In the velogenic form, also referred to as the viscerotrophic velogenic form, clinical signs are severe and include marked respiratory distress and diarrhea. Adult chickens and chicks die rapidly after the onset of clinical signs, and mortality may reach 100%. Some velogenic strains of ND virus are principally neurotrophic, causing paralysis of the legs and wings, twisting of the head and neck; circling, trembling, and walking backward. Similar central nervous system signs may be noted in chickens that survive acute viscerotrophic forms of ND.
There are no pathognomonic necropsy lesions associated with ND virus infections. The respiratory system may show hyperemia and congestion with mucus in the trachea. Air sacs may be thickened and cloudy and may contain yellow exudate. These lesions are most severe in velogenic forms. Hemorrhages and necrotic areas involving the proventricular and intestinal mucosa are also noted with velogenic forms.

Diagnosis
- Clinical specimens: Lung, trachea, liver, spleen, brain, and serum.
- Newcastle disease virus is easily isolated by the inoculation of embryonated chicken eggs via the allantoic cavity. The pathogenicity for embryos varies widely with strains; time of death may vary from more than 100 to less than 50 hours after inoculation. Allantoic fluids containing ND virus agglutinate red cells of chickens, guinea pigs, mice, and humans. The virus can be identified by hemagglutination inhibition and virus neutralization tests using specific NDV antiserum. Antibodies in sera can be detected and measured by these same procedures. It should be kept in mind that hemagglutination activity may be due to avian paramyxoviruses or orthomyxoviruses.

Prevention
- Prevention is best accomplished by maintaining closed flocks. Any additions should be subjected to testing and quarantine.
- Both live virus (low virulence) and killed vaccines are widely used in countries where ND is endemic. Live virus vaccines are administered in drinking water and by aerosol sprays, whereas killed adjuvant vaccines are given by injection. Healthy chicks may be vaccinated as early as 1 - 4 days of age.
- In countries where ND is reportable, appropriate officials should be notified of outbreaks. Confirmed outbreaks of velogenic forms of ND are dealt with by strict quarantine and slaughter.

Public Health Significance
Humans are occasionally infected, resulting in a mild influenza-like disease, with conjunctivitis.

*In Poultry Diseases, 5th ed. Editors, Jordan et al. WB Saunders, New York, 2001.

Other Avian Paramyxoviruses
Over a number of decades many isolates of paramyxoviruses that differ serologically from Newcastle disease strains have been recovered from chickens, turkeys, ducks and other avian species in the USA and other countries. At least eight serotypes have been identified. The isolates vary in pathogenicity and are most commonly associated with respiratory disease.

Pneumovirus

Bovine Respiratory Syncytial Virus Infection

Cause

Bovine respiratory syncytial virus.

Occurrence

Bovine respiratory syncytial virus (BRSV) affects cattle, sheep and goats and is widely distributed in cattle throughout the world. The disease occurs mainly in young beef and dairy cattle. Recently weaned and transported calves are particularly susceptible.

Transmission Infected cattle shed virus in respiratory secretions, and the disease is spread by respiratory droplets and by direct and indirect contact.

Clinical & Pathologic Features The virus replicates in the respiratory epithelium where it may cause varying degrees of cellular damage including necrosis. Syncytia and intracytoplasmic inclusion bodies are seen in bronchiolar, alveolar epithelial and other cells. The morbidity is generally high in fully susceptible herds; the mortality may approach 20% in severe outbreaks. The occurrence of moderate to high levels of BRSV antibodies in herds not experiencing respiratory disease in the recent past suggest that many BRSV infections may be subclinical or mild. However, some animals develop acute diffuse interstitial pneumonia with or without secondary bacteria. Mortality is not rare in bacteria-complicated, severe cases. Clinical signs include coughing, nasal discharge, and fever. Severely affected animals may display dyspnea and mouth breathing. In the absence of secondary bacterial infections, recovery occurs in 1 - 2 weeks.

Diagnosis

- Clinical specimens: Slide preparations from nasal and conjunctival scrapings, nasal and conjunctival swabs, lung, and acute and convalescent sera.
- A rapid diagnosis can sometimes be achieved by the fluorescent antibody (FA) examination of cytologic preparations of nasal and conjunctival epithelia collected early in the course of disease.
- Similarly, FA or immunoperoxidase staining is used to demonstrate viral infected cells in cryostat sections of lung tissue in animals that have died. In these animals histopathologic lesions are helpful in making a diagnosis of BRSV infection, especially if syncytial cells containing cytoplasmic inclusions are observed.
- The virus, which is antigenically related to human respiratory syncytial virus, can be isolated in cell cultures of bovine origin in which it produces cytopathic effects consisting of syncytia and eosinophilic cytoplasmic inclusions.
- Because of the lability of the virus, isolation attempts are apt to be negative unless the specimens are almost immediately fresh.
- A serologic diagnosis can be made by the demonstration of a significant increase in antibody levels between acute and convalescent sera using ELISA or virus neutralization.

Prevention

- Important considerations are: a closed herd policy, raising young animals apart from older animals, strict sanitation measures and vaccination.
- Modified live and inactivated virus are included in combined products to prevent other important diseases such as infectious bovine rhinotracheitis, bovine virus diarrhea and bovine parainfluenza 3 infection.

Metapneumovirus

Turkey Rhinotracheitis

This disease is caused by the only species in the genus Metapneumovirus. Three and possibly more types of the virus have been identified to date. The disease affects turkeys (3 - 10 weeks) and probably occurs worldwide. It spreads rapidly by infectious droplets. It is a severe, highly contagious upper respiratory infection characterized by sudden onset, swollen sinuses (swollen head syndrome) and coryza. Morbidity and mortality may be high. Because of its similarity to other respiratory diseases, diagnosis should be confirmed by laboratory identification of the virus. Procedures used are isolation and identification of the virus using turkey organ cultures or embryonated eggs. Various serological procedures are available for flock surveys; ELISA is probably the most satisfactory. Live attenuated and oil adjuvant vaccines are used for prevention.