DISEASES BY PARAMYXOVIRIDAE

(enveloped RNA virus)

Paramyxoviruses

Mumps

Parainfluenza - 1 (swine), Parainfluenza - 2 (canine), Parainfluenza - 3 (bovine, ovine) and Parainfluenza - 4 (human)

Morbilliviruses

- Measles
- Canine distemper
- Peste-des-petits-ruminants (PPR)
- Rinderpest

Rubulaviruses (newly k.a. Avulaviruses) : have both

- Avian paramyxovirus - 1 - Newcastle disease (Ranikhet disease)
- Avian paramyxoviruses (type 2-9)

Pneumoviruses

Respiratory syncytial virus (bovine)

- Avian pneumovirus (Turkey rhinotracheitis, TRT)
- Swollen head syndrome (SHS) of chickens

Peste-des-petits-ruminants (PPR)

(Commonly known as PPR, Goat Plague, Erosive Stomatitis and Enteritis of goats, and stomatitis-pneumoenteritis complex; Kala chhera)

INTRODUCTION & ETIOLOGY

"Peste-des-petits-ruminants (PPR)" is a French name and means "plague of small ruminants", i.e., goats and sheep. The disease was called "plague" because it inflicted heavy mortality. 'Plague' is a Latin word for 'blow'. Therefore, in past, any epidemic disease which caused high mortality was called 'plague'. Thus, rinderpest was first called "cattle plague"; swine fever as "swine plague", and avian influenza as "avian plague". PPR is thus commonly known as "**goat plague**".

PPR is an acute, highly contagious disease of goats and sheep, caused by PPR virus, which is ss RNA virus of genus **morbillivirus** (paramyxoviridae). Official name of this virus was changed in 2016 to ***Small ruminant morbillivirus* (SRM)** but still it is commonly known as PPRV by people working in the field. The disease is more severe in goats than in sheep, and is rapidly fatal in young animals. It is a ***NOTIFIABLE Disease*** in India.

The disease was first recognized in 1942 in Ivory Coast, in French West Africa. In India, it was first reported in **1989** in native sheep flocks in Villupuram district of Tamil **Nadu**, and is now widely spread in the country. PPR is characterized by fever, anorexia, **Lymphopaenia**, **Erosive Stomatitis**, diarrhoea, oculo-nasal purulent dischage, and respiratory distress. It is almost rinderpest-like disease, and is **highly fatal in goats**. PPR is antigenically closely related to the viruses of rinderpest, canine distemper, and measles (of humans).

Two signs often seen in PPR and not in RP are (i) crusting scabs along the lips and (i) development of Bronchopneumonia in later stages of disease.

SPREAD

Large amounts of the virus are present in all body excretions and secretions, especially in diarrhoeic faeces. Infection is mainly by inhalation, but could also occur through the conjunctiva and oral mucosa. Close contact with an infected animal, or contaminated fomites, plays important role in disease to spread. The disease is usually fatal in goats.

PATHOGENESIS

The virus penetrates the **retro-pharyngeal mucosa**, sets up a viraemia, and specifically damages the **alimentary, respiratory, and lymphoid systems**. Some virulent strains cause death from severe **diarrhoea and dehydration**, particularly in young goats before respiratory lesions become severe. In others, death may occur from concurrent secondary diseases such as pneumonic pasteurellosis and coccidiosis.

Lymphoid necrosis is not as marked in this disease as in rinderpest. Most sheep and some adult goats recover and they carry antibodies that confer life-long immunity.

SIGNS

The PPR can be acute or subacute.

The ACUTE FORM is seen mainly in goats, and is similar to rinderpest in cattle. Signs generally appear 3-6 days after being in contact with an infected animal. A **high fever** (above 104°F) is accompanied by **dullness, sneezing, and serous discharges from the eyes and nostrils, develop a dry muzzle & dull coat** . A **leukopaenia** occurs from destructive effect of the virus on lymphocytes, but this is less marked than in rinderpest.

In 2-4 days, gums become hyperaemic, and distinct erosive necrotic lesions develop in the oral cavity with excessive salivation. This extends over the entire oral mucosa, forming **diphtheritic plaques** (mouth & oral mucosa). There is profound **halitosis** (offensive breath) and the animal is unable to eat because of the sore mouth and swollen lips. Serous nasal discharge that later become mucopurulent and resulting, at times, in profuse catarrhal exudate which crusts over and occludes the the external nares (nostrils), with signs of respiratory distress. Serous ocular discharge (Epiphora) is also seen.

Diarrhoea develops 3-4 days after the onset of fever. It is profuse and the faeces may be mucoid and blood-tinged. Dyspnoea and coughing occur later, and the respiratory signs are aggravated when there is secondary bacterial pneumonia. Bronchopneumonia evidenced by coughing is a common feature.

Superficial erosions may occur in the mucosa of the vulva and prepuce. Death usually occurs within a week of the onset of illness. Pregnant animals may abort.

SUBACUTE infection is more common in sheep, and are manifested by **catarrh, low-grade fever, and intermittent diarrhoea**. Most recover after a course of 10-14 days. Animals recovering from the disease (both goats and sheep) have a lasting immunity.

LESIONS

Grossly, discrete (separate) or extensive areas of **erosion, necrosis, and ulceration**, present in the oral mucosa, pharynx and upper oesophagus, may extend to the abomasum and distant small intestine.

Emaciation, conjunctivitis, **Erosive Stomatitis** involving the inside of the lower lips and adjacent gum near the commisures and the free portion of the tongue

Haemorrhagic ulceration is marked in the ileo-caecal region, colon, and rectum, where they produce typical "**Zebra stripes**". Retropharyngeal and mesenteric lymph nodes are enlarged. Spleen may also be enlarged.

Severe lesions are often present throughout the **Respiratory Tract**. Small erosions and petechiae on the nasal mucosa, turbinates, larynx and trachea with **mucopurulent exudate** is seen. **Bronchopneumonia** is a constant lesion. Grossly, the pneumonia is characterized by areas of red consolidation and atelectasis. These usually involve the antero-ventral lobes, particularly the right lobe. With bacterial complications (mostly Pasteurella), there may be purulent or fibrinous bronchopneumonia and pleuritis.

Congestion, enlargement and oedema of most of the lymph nodes is seen. Erosive vulvo-vaginitis may exist

Microscopic lesions in the alimentary tract are similar to those in rinderpest, but are often **more severe**. Intracytoplasmic inclusion bodies may be abundant in necrotic glands of the small and large intestines.

In the respiratory tract, there is proliferative rhino-tracheitis, bronchitis and bronchiolitis, and within the alveoli proliferation of **type II pneumocytes** and formation of huge Syncytial cells (multinucleated giant cells) in stratified squamous epithelium of upper respiratory tract. Intracytoplasmic and intranuclear eosinophilic inclusion bodies are common in the epithelial cells of the airways and type II pneumocytes and syncytial cells.

DIAGNOSIS

Presumptive diagnosis can be made from the clinical signs and postmortem findings. BronchoPneumonia is usually a feature of PPR, but not rinderpest.

Confirmation can be achieved by isolation of the virus and its identification.

For provisional diagnosis antigen in lymph nodes can be detected by agar gel immunodiffusion (AGID) or by counter-immunoelectrophoresis technique (CIEP).

Since PPR and rinderpest in goats are indistinguishable both clinically and pathologically, it is important to confirm PPR virus using '**immunocapture sandwich ELISA'** that employs specific PPRV monoclonal antibodies.

Differential diagnosis is also possible by complement fixation and virus neutralization tests, however confirmatory diagnosis is nowaday achieved using reverse transcriptase-polymerase chain reaction (RT-PCR).

Differential diagnosis

- Contagious caprine pleuropneumonia (CCPP)
- Bluetongue
- Pasteurellosis (also may occur as secondary infection to PPR)
- Contagious ecthyma
- Foot and mouth disease
- Heartwater
- Coccidiosis

RINDERPEST

(also commonly known as **Cattle Plague)**

INTRODUCTION & ETIOLOGY:

Rinderpest is an **acute, highly contagious** disease of cattle caused by a morbillivirus (Paramyxoviridae) and is characterized by high fever, necrotic stomatitis, diarrhoea, and high mortality. Buffaloes, sheep, pigs, goats, and camels are also susceptible. **In** wildlife, rinderpest has been reported in deer, antelope, wild buffaloes, wild boars, bushbuck, warthogs and giraffe. Exotic cattle breeds (HF, Jersey etc.) were more susceptible than indigenous breeds.

This fight to control this disease was instrumental in establishment of **first veterinary college in 1762 in Lyon**, France. Mortality varies from 25%-90%, depending on the strains of virus and the resistance of the animals.

The rinderpest virus is anti genically closely related to the viruses of canine distemper, peste-des-petits-ruminants (PPR) of sheep and goats, and measles of humans.

Rinderpest PRESENTLY has been declared **eradicated** from India and officially OIE/ WOAH have recognised India free from Rinderpest on **25 May 2006**.

SPREAD

Rinderpest virus is quite fragile. Therefore, close contact between infected and non-infected animals is necessary for spread of the disease, because the virus does not survive for long outside the host.

Transmission occurs through contaminated feed, or by inhalation of aerosol (infected droplets). The virus is excreted by infected animals in nasal discharges, faeces, urine and sweat also.

PATHOGENESIS

The virus is inhaled in infected droplets. It penetrates through the epithelium of the upper respiratory tract and multiplies in the **tonsils and regional lymph nodes**. From here it enters the blood in mononuclear cells, which disseminate the virus to other lymphoid organs, the lungs, and epithelial cells of mucous membranes. The virus is present in blood and secretions before the symptoms appear.

The virus has a high degree of affinity for **lymphoid tissue and alimentary mucosa**. There is a pronounced destruction of lymphocytes in tissues. This is the cause of marked **Leukopaenia and immunosuppression**. **The virus is present in body, intimately attached to Leukocytes**, only a small proportion being free in plasma. The focal, necrotic stomatitis and enteritis are result of infection and replication in epithelial cells of alimentary tract.

However, since the virus induces a strong antibody response shortly after infection, there is a rapid decrease and elimination of virus from the body as the clinical signs and lesions become visible. Death is usually from severe dehydration, but in less acute cases, death may be from activated secondary parasitic or bacterial infections due to immunosupression.

SIGNS

The onset of illness is indicated by a sharp rise in body temperature (104°-105°F), accompanied by restlessness, dryness of the muzzle, and constipation. Other signs include photophobia (intolerance to light), excessive thirst, starry (shining) coat, retarded rumination, anorexia, and excessive salivation.

A maculo-papular rash may develop on parts of the body, where hairs are fine. The fever usually reaches its peak on the 3rd or 5th day, but drops abruptly with the onset of diarrhoea, even though other symptoms get intensified.

Lesions in the oral mucosa appear by 2nd or 3rd day of fever, but become clearly visible only after the onset of diarrhoea. As diarrhoea becomes severe, it is accompanied by abdominal pain, increased respiration, severe dehydration & emaciation. This is followed by prostration, subnormal temperature, and death, usually after a course of 6-12 days.

A marked **Leukopaenia** occurs at the height of infection. The total count usually falls to below 4000/µl, and is due to an abrupt drop in lymphocytes. Immunity after a natural infection is long and persists for life.

LESIONS

As already mentioned, the rinderpest virus has a particular affinity for Lymphoid tissues and epithelial tissues of the gastrointestinal tract, where it produces severe characteristic changes.

In **Lymphoid Tissue**, the virus causes necrosis of lymphocytes. This is striking in microscopic sections of lymph nodes, spleen, and Peyer's patches. The destruction of lymphocytes is first seen as a fragmentation of nuclei in the germinal centres, and in a short time, most of the mature lymphocytes disappear. Multinucleated giant cells containing eosinophilic cytoplasmic inclusion bodies are often present. Oedema and congestion of capillaries are seen microscopically. The destruction of lymphoid cells leaves a fibrillar, eosinophilic, acellular matrix at sites of the lymphoid follicles. Grossly, changes are most marked in the **Peyer's patches**, which may be darkened with haemorrhages and sloughed out, leaving **deep raw craters (ulcers) in the intestinal wall**.

In the DIGESTIVE SYSTEM of cattle, rinderpest virus produces typical lesions. The virus is carried to the oral mucosa by the bloodstream. In the squamous epithelium of the oral cavity, the first change is necrosis of a few epithelial cells in deep layers of the **stratum malpighii**. These cells have pyknotic and fragmented nuclei and eosinophilic cytoplasm. They are shrunken and separated from the adjoining space by a clear space. As these necrotic areas increase in size and extend towards the surface, the **cornified layer above**

them becomes elevated. This causes them to appear grossly as tiny, greyish-white, slightly raised puncta (points). Eosinophilic cytoplasmic inclusion bodies form in the mucosal epithelial cells and multinucleated giant cells form in the stratum spinosum.

Vesicles are not formed in this disease. With time, the foci of necrosis coalesce to form large areas of erosions. Since the basal layer of the squamous epithelium is rarely penetrated, **ulcers seldom form**.

The **EROSIONS** are **shallow, with a red raw floor, and a sharply demarcated margin**. The lesions in the oral mucosa have a selective distribution: inside the lower lip, the adjacent gum, the cheeks near commissures, and the ventral surface of the free portion of the tongue. In severe cases, lesions extend to the hard palate and pharynx, and in fulminant cases, to all the mucous surfaces of the tongue. The oesophageal lesions are similar to those in the mouth, but are less severe.

The rumen, reticulum, and omasum **rarely exhibit any lesions**. **The ABOMASUM is one of the most common sites of the lesions of rinderpest.** They are most severe in the pyloric region, where necrotic foci of microscopic size, resulting in irregular superficial bright red to dark brown streaks. These streaks follow the edges of the broad plicae and extend into the fundus, but become more numerous and diffuse in the pylorus. As necrosis progresses, the epithelium sloughs away, leaving sharply outlined irregular erosions with a red raw floor oozing blood.

In the small intestine, severe lesions are less common, however Peyer's patches are exceptionally vulnerable.

The **LARGE INTESTINE**, is more seriously damaged than the small intestine, with prominent lesions in the ileo-caecal valve, caeco-colic junction, and the rectum.where diffuse hemorrhages with pseudo-membranous bran-like deposits are seen The crest of the folds of mucous membrane throughout the caecum is bright red because of numerous petechiae, which appear more like diffuse haemorrhages. Streaks of hemorrhages & congestion on the folds of mucosa of rectum, gives it a characteristic **"Zebra-striped" (Zebra markings),** or **"barred" appearance**. The mucosa therefore is bright red and lumen contains clotted blood. As disease progresses and the mucosa becomes eroded, diffuse congestion and bleeding from the raw surfaces may occur over large areas. When opened, the mucosa is diffusely red, and the lumen contains partially clotted blood.

The characteristic streaks of cogestion and haemorrhage are more striking in rectum than in colon. The liver is affected only secondarily in rinderpest, with chronic passive congestion resulting from cardiac and pulmonary complications. Lesions in the gallbladder vary from scattered petechiae to diffuse blotches of haemorrhage.

In the **respiratory system, p**etechiae occur on the turbinates and larynx. In trachea, streaks of haemorrhages in mucosa are almost always found. Most common are longitudinal streaks of rusty haemorrhage in the anterior third of the trachea. The lungs appear to be involved only secondarily. Pneumonia not normally seen, as it is in PPR.

The lesions of the heart also appear to be secondary and include subendocardial haemorrhages over the papillary muscles of the left ventricle. The right ventricle is seldom

involved. In the urinary system, lesions comprise oedema around the renal pelvis and desquamation of pelvic epithelium. In the urinary bladder, the epithelium may be desquamated and the underlying stroma infiltrated with erythrocytes. The infiltrates are grossly seen as thin red blotches.

Lesions in the skin, though not common, may be seen as a maculo-papular rash over thinly haired portions of the body, the vulva, and prepuce.

DIAGNOSIS

The clinical, gross, and microscopic features of the disease are adequate for a presumptive diagnosis.

Confirmative diagnosis using immunological methods like Agar gel immunodiffusion (AGID), complement fixation, counter-immunoelectrophoresis (CIEP), immunofluorescence, and immuno-peroxidase tests,

ELISA which is accurate and easy to perform test suitable for field use.

Using **RT-PCR**, the Rinderpest and PPR viruses can now be confirmed and differentiated.