



# An Overview on Important Transboundary Diseases of Animals: An Editorial

Subha Ganguly

Associate Professor, Department of Veterinary Microbiology, ARAWALI VETERINARY COLLEGE (Affiliated with Rajasthan University of Veterinary and Animal Sciences, Bikaner), N.H. – 52 Jaipur Road, V.P.O. Bajor, Sikar – 332001, Rajasthan, India

**Abstract--** Transboundary animal diseases are of huge concern in global trade of animals and animal products. These diseases occupy a significant socio-economic importance and can spread across International borders. These diseases are also associated with severe public health hazards.

**Keywords--** Animals, Transboundary diseases, Public health

## I. PESTE- DES-PETITS- RUMINANTS (PPR, GOAT PLAGUE)

### *Synonyms*

Plague of small ruminants (goats, sheep), Erosive stomatitis, Enteritis of goats

### *Definition*

It is an acute, highly contagious disease of goats and sheep characterized by fever, anorexia, lymphopaenia, erosive stomatitis, diarrhoea, oculo-nasal discharge and respiratory distress

### *Etiology*

Morbilli virus

### *Incidence*

- First reported in 1942 in Africa
- In India, first reported in sheep flocks during 1989 in the Villupuram district of Tamil Nadu

### *Susceptibility*

Disease is more severe in goats than sheep; fatal in young animals

### *Transmission*

Close contact with infected animal -Direct contact, contaminated fomites, inhalation / conjunctival or oral routes, Large amount of virus is present in excretions and secretions

### *Clinical signs*

Acute or subacute form

### *Acute form*

Clinical signs similar to Rinderpest in cattle

- High fever, dullness, sneezing

- Serous discharges from eyes and nostrils which turns mucopurulent later
- Necrotic lesions in mouth, oral mucosa forming diphtheretic plaques
- Diarrhoea within 3-4 years after onset of fever (mucoid or blood tinged)
- Dyspnoea and coughing
- Death within one week of onset of illness
- Pregnant animals abort

### *Subacute form*

- More common in sheep, Mucopurulent discharge from eyes and nostrils
- Low grade fever, Intermittent diarrhoea
- Recovery after 10 – 14 days, long lasting immunity in recovered animals

### *Gross lesions*

- Erosion, necrosis, ulceration on oral mucosa, pharynx, upper oesophagus; abomasum, small intestine
- Haemorrhage and ulcers in ileo – caecal junction, colon and rectum forming “Zebra stripes”
- Retropharyngeal and mesenteric lymph nodes are enlarged and haemorrhagic
- Spleen enlarged
- Mucopurulent exudate from nasal opening to larynx
- Hyperaemia of trachea and bronchi
- Congestion and oedema of lungs, pneumonia
- With secondary bacterial complications, fibrinous bronchopneumonia and pleuritis is common

### *Microscopic lesions*

- Syncytia formation in stratified squamous epithelium of upper respiratory tract
- Degeneration and necrosis of infected cells
- Intracytoplasmic inclusion bodies in epithelial cells of upper respiratory tract or intestine
- Proliferative rhino tracheitis, bronchitis, bronchiolitis

- Intracytoplasmic and intranuclear eosinophilic inclusion bodies in the respiratory epithelial cells / syncytia

*Diagnosis*

- Differential diagnosis from Rinderpest – Pneumonia is not seen in Rinderpest
- Isolation and identification of the virus
- AGID (agar gel immuno diffusion test) or CIE (counter-immuno electrophoresis) for demonstration of antigen in lymphnodes and other tissues
- Immuno capture sandwich ELISA
- RT – PCR

**II. AFRICAN HORSE SICKNESS**

*Synonym:* AHS, EQUINE PLAGUE

*Definition*

AHS is a highly fatal viral disease of horse, mules and donkey caused by orbivirus characterized by either pulmonary involvement or cardiac involvement or both

*Etiology*

Double Stranded RNA virus – Orbivirus

*Susceptibility*

Horses, mules and donkey

*Transmission*

By *Culicoides* mosquitoes

*Pathogenesis*

Orbivirus is a viscerotropic virus and is found in all tissues and fluids of the body

*Four forms*

- Acute pulmonary form
- Subacute cardiac form
- Mixed form
- Mild form

*Acute Pulmonary form: (DUNKOP form)*

- Fever, dyspnea, coughing
- Frothy nasal discharge – pulmonary oedema
- Profuse sweating and nasal discharge
- Death

*Subacute Cardiac form (DIKKOP FORM)*

- Progressive fever
- Progressive edema of lips, eyelids, neck and chest
- Swollen, cyanotic tongue with petechiae
- Paralysis of esophagus – unable to swallow

- Cardiac failure – pulmonary oedema, hydropericardium and endocarditis
- Death

*Mixed form*

- Both pulmonary and cardiac form present

*Mild form*

- No symptoms, mild fever, anorexia, dyspnoea, mild conjunctivitis

*Lesions*

*Pulmonary form: Hydrothorax*

- Pulmonary edema –frothy exudate in bronchi, trachea, pharynx & nasal passages

*Cardiac form*

- Hydropericardium, ascites
- Haemorrhages of myocardium
- Necrosis of myocardium
- Congestion of GI mucosa
- Enlarged & congested liver
- Haemorrhagic lymph nodes – depletion of lymphocytes
- Edema around pharynx – paralysis of oesophagus

*Diagnosis*

- Clinical signs & lesions
- Intracerebral inoculation into mice and then conducting neutralization test using a known antiserum
- Neutralization test

**III. FOOT AND MOUTH DISEASE**

Many associated potential risk factors responsible for the introduction and spread of the FMDV infection in the region. Among these are biosecurity, movement of live animals and animal products, swill feeding and access to landfill waste. The absence of significant clinical signs in sheep in particular, and the increased livestock migration particularly during the festival seasons give rise to specific concerns. Active surveillance for early detection of FMDV infection in wildlife could be a useful addition to an effective passive surveillance system in domestic animals.

**IV. RIFT VALLEY FEVER**

*Etiology:* RVF virus is negative-sense, ss-RNA virus of the family *Bunyaviridae* with in genus *Phelebovirus* (only one strain)

*Host:-* cattle, sheep ,goat,buffaloes, humans (very susceptible)

*Transmission:-* certain Aedes sp. Act as reservoirs for RVF virus during inter epidemic period.  
*infected Aedes*

Feed preferentially on domestic ruminants which act as an amplifier of RVF

Direct contamination and mechanical transmission also occur.

*Diagnosis:*

Incubation period – 1 to 6 days ,12 to 36 hours in lambs

*Clinical diagnosis:-*

*Cattle*

- bloody diarrhoea
- abortion, lacrymation, nasal discharge and excessive salivation,anorexia,fever

*Sheep/Goat*

- biphasic fever (40-42c),rapid abdominal respiration prior to death
- death with in 24-36 hours, bloody diarrhoea
- icterus ,anorexia, abortion

*Humans*

- influenza like syndrome- fever, headache, muscular pain ,nausea
- retiopathy, meningoencephalitis, haemorrhagic syndrome with jaundice, death

*Lesions*

- FOCAL HEPATIC NECROSIS(white foci - 1mm)
- Brown –yellow colour of liver in aborted fetuses.
- Cutaneous haemorrhages, petechial to ecchymotic haemorrhage on parietal and visceral serosal membranes.

*Laboratory diagnosis:*

- Serological tests-virus neutralisation , ELISA, HI
- Identification of agent-agar gel immunodiffusion, PCR, culture, histopathology

#### V. AFRICAN SWINE FEVER

*Definition:-* ASF is serious, highly contagious, viral disease of pig.ASF is characterized by high fever, loss of appetite, haemorrhages in the skin and internal organ, death in 2-10 days on average. Mortality rate -100%.

ASF is DNA virus of the *Asfarviridae* family.

*Transmission:-*

Natural reservoir: Wart hog. Spread from this reservoir is via the soft tick ornithodoros moubata .

*Clinical sign:-* fever and death in 2-10 days on average. Mortality rate 100%.

- Loss of appetite
- Depression, diarrhoea
- Redness of the skin of the ears, abdomen, legs.
- Respiratory distress, vomiting. Bleeding from the nose or rectum
- Abortion

*PM Findings:-*

- Cyanosis of skin
- Haemorrhages in the internal organs like liver, spleen, lymph nodes, kidneys, larynx, bladder
- Splenomegaly
- Oedema of the digestive tract effusion in natural cavities

*Diagnosis:*

- History and clinical sign
- ELISA, FAT, PCR
- Haemadsorption test

#### VI. LUMPY SKIN DISEASE

Susceptible hosts include cattle and goats and wild animals like giraffes, impalas and African buffaloes.

*Geographical distribution:*

In 1929, the first epidemic of lumpy skin diseases occurred in Zambia and affected huge population of cattle in African continent since then. The infection also spread in Egypt, Sudan and South Africa followed by 1989 outbreak in Israel. [2]

*Transmission:*

The disease is transmitted by biting insects and midges.

*Clinical signs and symptoms:*

The incubation period of the disease ranges from 2-4 weeks and clinical signs and symptoms include necrotic skin lesions with fever and ocular and nasal discharge. The lymph nodes become swollen due to edema of the limbs. Morbidity in the disease is high with low mortality rates.

*Diagnosis:*

Differential diagnosis should be made with pseudo-lumpy skin disease during the early stages of infection.



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*Control and prevention:*

A live attenuated version of the Neethling virus and another live attenuated version of the sheep pox virus can be used as vaccines for vaccination against the disease.

VII. CONCLUSION

Sheep pox and goat pox are fatal diseases of concern with characteristic symptoms of vesicle formation and eruption on skin. The occurrence of the diseases is confined to parts of southeastern Europe, Africa, and Asia. Both the sheep and goat poxviruses (capripoxviruses) are closely related to each other in their antigenic and physico-chemical behavior. Both the sheep and goat pox are related to the virus of lumpy skin disease.

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**Corresponding author: Dr. Subha Ganguly** PhD, DSc; Sr Honorary Editorial Board member [*IJETAE/ IJRDET Online*]; **Email:** [ganguly38@gmail.com](mailto:ganguly38@gmail.com)