

An Overview on Important Transboundary Diseases of Animals: An Editorial

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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) [1]

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

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- 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 ^[2]

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 ^[3]

Many associated potential risk factors re 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 ^[4]

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 within 24-36 hours, bloody diarrhoea
- icterus ,anorexia, abortion

Humans

- influenza like syndrome-fever, headache, muscular pain ,nausea
- retinopathy, 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 [4]

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 [5-7]

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.

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.

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.

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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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