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Bluetongue

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Bluetongue (BT) disease, caused by bluetongue virus (BTV) is an acute hemorrhagic fever of domesticated and wild ruminants. An inactivated pentavalent vaccine consisting of BTV serotypes 1, 2, 10, 16, 23 is currently being used in some parts of India.

Etiology

BTV is a member of the family Reoviridae and is the prototype virus of the genus Orbivirus. So far, 29 serotypes of BTV have been identified worldwide and a total of 24 serotypes have been reported from India. The BTV genome consists of 10 linear double-stranded RNA segments, which encode a total of seven structural proteins (VP1 to VP7) and six distinct nonstructural proteins (NS1, NS2 and NS3/ NS3a).

Hosts Affected

All ruminants are potential hosts of BTV, though species and breed, among other factors, can play a role in whether BTV infection manifests as clinical disease. BTV is typically transmitted among susceptible hosts through the bite of a competent Culicoides midge. Since BTV is primarily considered a vector-borne virus, its epidemiology is strongly linked to the presence of competent vectors. However, in some cases, vertical (transplacental) transmission has been implicated for certain strains or serotypes.

Routes of Transmission

Since BTV is primarily considered a vector-borne virus, its epidemiology is strongly linked to the presence of competent vectors. However, in some cases, vertical (transplacental) transmission has been implicated for certain strains or serotypes and there are even recent reports of direct contact transmission of some BTV serotypes in goats and cattle.

Only female *Culicoides* midges are hematophagous and a single bite of an infected midge is

sufficient to infect a susceptible sheep. Conversely, the quantity of virus in the host blood considered necessary to infect a competent *Culicoides* midge is relatively low, at approximately 2.5-3 log10 $TCID_{50}$ (50% tissue culture infective dose) per millilitre.

Clinical signs

BTV can infect all known species of domestic and wild ruminants. Severe disease usually occurs in the fine-wool and mutton breeds of sheep as well as some species of deer. BTV infection of cattle, goats and wild ruminant species is mostly asymptomatic or subclinical. In BTV endemic areas, BTV-infected sheep develop only mild or no obvious disease. The blue tongue after which the disease is named is seen only in serious clinical cases.

The clinical signs of BTV are variable. The first clinical sign is the rise in temperature to 41.6- 41.7^{o}C. Within 24 hours of initial rise of temperature, excessive salivation and frothing of mouth develop and are associated with hyperaemia and swelling of the buccal and nasal mucosa followed by erosions and ulcerations. By 4 to 7 days in severe cases, extensive ulcerations may be covered by gray necrotic tissue on the dental pad and dorsal surface of the tongue. Hyperaemia is also observed around the coronary bands of the hooves. The hooves are tender and varying degrees of lameness are also observed. In more severe cases, the animals stand with an arched back line in the wall of the hoof. The lesions in the mouth, the reluctance to move, and the necrosis of the striated musculature lead to weakness depression, and rapid weight loss. These can result in prostration and eventual death in severely affected animals. Sheep that recovered from severe infections may have a break in the well 3 to 4 weeks after the fever has subsided. This can lead to partial or complete shedding of wool. The reproductive form of the diseasevaries greatly. Signs include abortions, stillbirths, and week “dummy lamb” live births.

Cattle: usually do not cause clinical signs. In some cases, mild hyperemia in the buccal cavity and around the coronary band. Vesicular lesions, which lead to ulcerations, in the buccal lesions. Erect hair over the cervical and dorsal thoracic areas. BT is abortogenic and teratogenic in cattle experimentally, but not observed commercially in field conditions.

Goats: bluetongue infection of goats is an inapparent infection similar to that described in cattle.

Lesions: the lesions of bluetongue in sheep vary greatly depending on 1. The strain of virus. 2. Individual animal and breed susceptibility and environmental stress factors. Prominent lesions include facial edema, edematous ears and dry, crusty exudates over the nostrils.

Oral cavity and Digestive System: lesions in the oral cavity include focal petechial haemorrhages that progress to grey necrotized debris over erosions and ulcerations of the lips; on the dorsal, lateral, and ventral surfaces of the tongue; and on the dental pad. The buccal

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mucosa may be cyanotic. Hyperaemia and occasional erosions can occur on papillae and laminae in the reticulum and omasum.

Respiratory system: lesions include cyanosis and edema of the nasal mucosa and pharynx.

Vascular system: lesions include hyperaemia, edema and haemorrhages. A characteristic lesion in haemorrhage at the base of the pulmonary artery. Petechial and ecchymosis (larger than pinhead-size) haemorrhages may be observed at times in the endocardium. Focal grey-white areas of necrosis are often found in the papillary muscles and less frequently in other areas of the myocardium.

Skin: Dermal and subcutaneous edema of the head and ears and an irregular rash with serous amd crusty exudates on the skin are the common lesions.

Muscle: A yellow gelatinous exudate is [resent in the fascia along and between skeletal muscles. On the cut surface of the heavy muscle there may be focal haemorrhages and areas that appear dry and grey-white.

New born lambs: New born lambs with congenital BT have hydranencephaly or porencephaly. Abnormal development of cerebellum and spinal cord are the other lesions.

Those animals that do recover from BT may suffer from a number of long-lasting secondary effects, such as reduction in milk production, weight gain, wool break and temporary infertility.

Diagnosis

Field Diagnosis: Based on clinical signs in the susceptible population, coincidence of occurrence of symptoms with prevalence of insect vectors, characteristic gross lesions, and a flock history of recent wasting (loss of weight) and pododermatitis (foot rot).

Isolation and identification:

Clinical materials:

Live animals: Unclotted blood. Samples have to be kept at 4^{o}C and send to the lab.

Dead Animals (Fresh carcasses): Spleen, lymphnodes, blood from the heart, foetuses, dead animals (old carcases) & bone marrow.

Serogrouping: BTV are serogrouped on the basis of their reactivity with specific antisera that detect protein such as VP7 that are specific within each serogroup. Monoclonal antibodies specific for VP7 are used in the assay. The immunological assay that are performed using monoclonal antibodies against VP7 protein are immunofluorescence, Competitive ELISA.

Serotyping: Serotyping of BTV is carried out using serum specific for each of 24 serotypes. Virus neutralization assays are performed to serotype BTV. Four different types of neutralization assay are performed in BHK-21, Vero cells. These tests are Plaque reduction tests, plaque inhibition test, microtitre neutralization test and fluorescent inhibition test.

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Treatment: Supportive treatment by use of antiseptic is a solution of 1% potassium permanganate for washing the hoof and mouth lesions. In addition, a 5% boro-glycerine lotion is topically applied twice daily over the mouth erosions for early healing. Administration of broad-spectrum antibiotics to combat secondary infections.

Prevention and control

Traditional prevention and control measures for viral livestock diseases include the restriction of animal trade movements or quarantine of sick animals, optimization of zoo sanitary and other biosecurity approaches, treatment when available, vaccination, and eradication or pre-emptive slaughter. An inactivated pentavalent vaccine consisting of BTV serotypes 1, 2, 10, 16, 23 is currently being used in some parts of India as Raksha BLU vaccine.

Circulation of multiple serotypes, often within the same animal and little or no cross-protection between serotypes makes BTV control and prevention problematic.

-transmissible disease characterized by the spastic paralysis caused by the neurotoxin which has been identified as tetanospasmin. Tetanospasmin is produced by an anaerobic bacterium Clostridium tetani in a deep wound under anaerobic conditions. Various animal species are di□erently susceptible to this infection .

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