BOVINE VIRAL DIARRHOEA (BVD)

(Commonly known as: *Virus Diarrhoea* and other form k.a. *Mucosal Disease*)

Keywords: persistent infection; non-cytopathic virus biotype, cytopathic biotype, in-utero; transplacental transmission

BVD is a contagious disease of cattle, first described in 1946 and was earlier termed "virus diarrhoea". The disease was characterized by high morbidity but low mortality. During the same time, similar disease was observed, but was more severe and had a high mortality and was termed "mucosal disease". The disease generally appears in cattle between 6 months and 2 years of age, and results in death.

ETIOLOGY:

BVD and Mucosal Disease are different presentation of the same viral disease caused by **BVDV, a pestivirus** and this difference in clinical picture of morbidity and mortality is determined by the viral strain variations and mode of infection.

Bovine viral diarrhoea virus (BVDV) occurs in two bio-types: cytopathogenic and non-cytopathogenic on the basis of their effects in tissue culture cells.

The **Bovine Virus Diarrhoea (BVD)** or simply 'Virus Diarrhoea' occurs sporadically in cattle between **6 months and 2 years of age** as a transient, highly contagious acute infection with a high morbidity rate, but is usually a mild disease. It may be caused by both non-cytopathic as well as cytopathic virus variants.

On the other hand, **Mucosal Disease** develops in cattle (neonates) which become infected *in utero* with **non-cytopathogenic BVDV** which become persistent and to which they are immunotolerant. Subsequently when in these persistently-infected animals, the virus transforms from **Non-cytopathogenic BVDV** ⇒ to **Cytopathogenic BVDV** through a process of RNA recombination. (also written as **Non-cytopathic BVDV** ⇒ to **Cytopathic BVDV**)

SPREAD

The virus is transmitted by direct contact between animals, and also by transplacental transmission to the foetus in the infected dam. Persistently viraemic female can remain clinically normal (immuno-tolerant) for several years, and may introduce infection into uninfected animals / herd.

The virus is present in nasal discharge saliva, faeces, urine, tears, milk and semen, each of which would allow wide dissemination and spread of the virus. Discharges from the reproductive tract of an infected cow, including aborted foetuses are potent source of virus, while persistently infected bulls may also introduce the virus into artificial breeding units.

PATHOGENESIS

The pathogenesis of disease due to infection with BVDV is governed by several features like occurrence of viraemia, ability of virus to damage immune system, age of animal, previous vaccination status of animal, occurrence of transplacental infection, induction of immune tolerance (persistent infection), and the emergence of foetal immune competence at about 180 days of gestation.

Persistent infection develops when non-cytopathic BVDV is transmitted transplacentally during the first 4 months (BEFORE 180 DAYS) of fetal development. The calf is born infected with virus, remains infected for life, and usually is immunotolerant to the resident noncytopathic virus. Transplacental infection that occurs later in gestation (after 180 days) can result in abortion, congenital malformations, or birth of apparently healthy calves that have antibody against BVDV.

Apart from those infected with the virus ***in utero*** most cattle are immuno-competent to the virus, and will successfully control a natural infection, develop antibodies, and eliminate the virus so that latency and shedding do not occur.

SIGNS

There are no pathognomonic signs or lesions of BVD. However clinical manifestations can broadly be grouped into following syndromes or types:

A. Inapparent Subclinical BVD
B. Peracute BVD
C. Hemorrhagic BVD
D. Acute Mucosal Disease
E. Chronic Mucosal Disease
F. Reproductive Disorders and Neonatal Disease

Disease induced by bovine viral diarrhea virus varies in severity, duration, and organ systems involved. Lymphoid tissue is a primary target for replication of BVDV, which may lead to immunosuppression and enhanced severity of intercurrent infections.

The clinical signs include high fever (105°-108° F), anorexia, depression, and diarrhoea accompanied by excessive salivation, with stringy mucus hanging from the muzzle to the ground. Ulcers develop in the mouth, nose, and muzzle of severely

affected animals. Other signs include mucous or mucopurulent nasal discharges and cough. The nasal and oral mucosa, and the conjunctiva are congested. Disturbances in distribution of body heat may be observed by touching. Transient **Leukopenia** may be seen with onset of signs of disease. Recovery is rapid and coincides with production of viral neutralizing antibody.

Some isolates of BVDV (BVD type 2) have been associated with **severe clinical disease** that manifests as high fever (~107°F [41°–42°C]), oral ulcerations, eruptive lesions of the **coronary band and interdigital cleft**, diarrhea, dehydration, **Leukopenia**, and **THROMBOCYTOPENIA**. In thrombocytopenic cattle, petechial hemorrhages may be seen in the conjunctiva, sclera, nictitating membrane of the eyes, and on mucosal surfaces of the mouth and vulva. Prolonged bleeding from injection sites also occurs.

CALVES born alive may be persistently infected and later succumb to **mucosal disease**. Congenital cerebellar hypoplasia, cataracts, retinal atrophy, microphthalmia, and optic neuritis have been noted in calves born to infected dams.

LESIONS

The main gross lesions are found in the gastrointestinal tract. There also may be necrosis in lymph nodes and the spleen, which may contribute to immunosuppression.

Irregularly-shaped ulcers or erosions of the mucosa are found on the dental pad, palate, lateral surfaces of the tongue, and inside of the cheeks. Ulcers may also occur on the muzzle and at the external nares. And even on mucosa of pharynx, covered by tenacious grey exudate. Necrotic lesions may be confined to the pharynx, or may extend to the larynx. In the Oesophagus, the entire mucous membrane may contain shallow erosions or ulcers with sharply defined, irregular margins and a red base. These ulcers may coalesce to form **elongated ulcers or erosions**, with necrotic material adhering. The abomasal mucosa, Omasum and Peyer's patches also reveal small haemorrhages and ulcers. Necrotizing Enteritis, probably immune-mediated is seen. In the foetus, lesions similar to those of the adult may be seen in the gastrointestinal tract.

Infection during the first 4 months of fetal development may lead to embryonic resorption, abortion, growth retardation, or persistent infection. Congenital malformations of the eye and CNS result from fetal infections that occur between months 4–6 of development. Fetal mummification, premature birth, stillbirth, and birth of weak calves also are seen after fetal infection.

The most significant congenital defects includes:

- Cerebellar Hypoplasia
- Cerebellar-ocular agenesis
- Brachygnathism
- Musculo-skeletal Deformities
- Alopecia & I/U growth retardation with stunting of long bones
- Stunted calves with curly hair coat

DIAGNOSIS

ELISA and PCR are the most reliable means of diagnosis.

Clinical signs and lesions have to be differentiated from GIT lesions of **Rinderpest** and oral and brain lesions of **Malignant Catarrhal Fever** (MCF)