Clinical aspects of bovine virus diarrhoea virus infection

J.C. BAKER *

Summary: Bovine virus diarrhoea virus (BVDV) infection of cattle results in a wide range of clinical manifestations. This article reviews the clinical responses associated with BVDV and discusses these diseases in terms of acute infection in immunocompetent cattle, fetal infection, infection in cattle immunotolerant to and persistently infected with BVDV and finally mucosal disease.


Bovine virus diarrhoea virus (BVDV) was first recognised in the United States in association with outbreaks of acute and often fatal disease characterised by diarrhoea and erosive lesions of the digestive tract (54). Bovine virus diarrhoea virus has a worldwide distribution. Infection with the virus is common, as indicated by the high prevalence of seropositive cattle (22, 23, 25). The biology of BVDV is complex, and multiple and diverse clinical manifestations may occur in cattle infected with the virus (Table I). This diversity was difficult to understand based on previous explanations of pathogenesis. For example, bovine virus diarrhoea (BVD) and mucosal disease are two clinically distinct and different diseases caused by the same virus. Recent investigations have brought a better understanding by focusing on cattle born immunotolerant and persistently infected with BVDV, and the role of these animals in the pathogenesis of mucosal disease. This article will review the spectrum of clinical responses that occur in cattle following BVDV infection.

ACUTE INFECTIONS

Subclinical infections

It has been estimated that 70 to 90% of BVDV infections in susceptible, immunocompetent cattle are subclinical (1). Cattle undergoing subclinical infections may have a mild elevation of body temperature and leukopenia that is followed by the development of specific neutralising antibody. This form of infection undoubtedly accounts for the high prevalence of cattle with serum antibody to BVDV without previous history of disease (25).

* Department of Large Animal Clinical Sciences, College of Veterinary Medicine, Michigan State University, East Lansing, Michigan 48824-1314, USA.
Acute bovine virus diarrhoea infection

- Subclinical infections
- Bovine virus diarrhoea
- Venereal infections
- Immunosuppression
- Bovine virus diarrhoea infection in neonatal calves
- Mixed infections (bovine respiratory disease)

Congenital infections

- Fetal abortion, stillbirths, fetal resorption and mummification
- Congenital defects
- Weak and undersized calves
- Normal calves born seropositive to BVDV
- Calves born immunotolerant to BVDV

Chronic infections

- Persistent viraemia

Mucosal disease

- Acute mucosal disease
- Chronic mucosal disease

Bovine virus diarrhoea

The disease referred to as bovine virus diarrhoea (BVD) represents an acute infection in seronegative, immunocompetent cattle (14). As previously mentioned, subclinical infection is most common in this group. When infections become clinical, they generally do so in cattle ranging in age from six months to two years (7). In susceptible herds, morbidity may be high but mortality is low to none. The incubation period is 5 to 7 days and is followed by transient fever and leukopenia (22). Viraemia occurs 4 to 7 days after infection (16) and in some cases may persist up to 15 days (22). Clinical signs include depression, anorexia, ocular nasal discharge and occasionally oral lesions characterised by erosions or shallow ulcerations. A rapid respiratory rate may develop secondarily to pyrexia which may be incorrectly diagnosed as pneumonia (56). Diarrhoea may also be present, and in lactating cows milk production may decrease. Virus appears to be shed in low concentrations when compared with persistently infected cattle (22). Diagnosis can be made by isolation of virus from blood, body secretions and excretions, or by demonstration of seroconversion. Virus neutralising antibodies generally appear in the serum two to four weeks after infection and probably persist for life. Following the immune response it is likely that there is life-long protection to disease caused by BVDV, but these individuals may be susceptible to periodic reinfection (16).

Thrombocytopenia has been reported as a feature of acute BVDV infection (68). While it could not be determined if the animals in the study were acutely infected, or persistently infected with non-cytopathic BVDV and undergoing superinfection with a cytopathic strain of BVDV resulting in mucosal disease, it is likely that some were acute infections as they recovered. The thrombocytopenia resulted in clinical signs of bleeding manifested by bloody diarrhoea, epistaxis, petechial haemorrhages, ecchymotic haemorrhages and bleeding from injection sites. The pathogenesis of the thrombocytopenia was not determined. However, disseminated intravascular
coagulation was not thought to be involved and most likely the thrombocytopenia occurred as a result of BVDV-induced myelosuppression or bone marrow necrosis.

It has been difficult to reproduce BVD in immunocompetent cattle under experimental conditions and the reason for this is not readily apparent. Infection with BVDV is known to cause immunosuppression (discussed later) and thus under field conditions the addition of environmental stressors and other pathogens may result in the manifestation of clinical disease. There is the possibility that different strains of the virus determine the ability to produce clinical disease, but this has not been proven.

Therapy for acute BVDV infection is non-specific. Depending on the severity of disease, hydration should be maintained with oral or IV fluids and electrolytes. Antimicrobial therapy may be considered in the presence of or for the prevention of secondary bacterial infections. Because the virus can cause immunosuppression, treatment with corticosteroids would be contraindicated but non-steroidal anti-inflammatories would be a therapeutic consideration.

Venereal infections

Semen from bulls persistently infected with BVDV contains virus which is infective to susceptible cows (6, 18, 44, 47). Semen from immunocompetent bulls undergoing acute BVDV infection may be transiently infected (55, 91). Acceptable semen quality and fertility has been reported for persistently infected bulls (6), although semen quality may be affected in both persistently infected and acutely infected bulls and is characterised by decreased motility and morphologic abnormalities (44, 55, 71). Significantly reduced conception rates have been observed in seronegative cattle exposed to the virus at the time of breeding, by serving cows with persistently infected bulls (44) and experimentally by intra-uterine administration of BVDV (2, 3, 24, 92). Seronegative cows inseminated with infective semen generally fail to conceive until they develop an immune response to the virus (44). However, a recent report indicated that twelve seronegative heifers inseminated with semen from a persistently infected bull all conceived, seroconverted and gave birth to normal appearing calves, although one calf was persistently infected with BVDV (47). The risk of venereal infection with BVDV appears to be highest with natural service (24). Bovine virus diarrhoea virus does not appear to inhibit conception of either seropositive or seronegative cattle when inoculated by an oral or intranasal route or infused into the uterus of a seropositive cow at the time of breeding (92). The adverse effect of this virus on conception is attributed to fertilisation failure (24).

Thus, BVDV may cause a herd problem characterised by repeat breeding if the infection is by the venereal route in seronegative cows. The most notable finding would be an increase in the number of services per conception. Such a problem will be transient in nature (until an immune response occurs), and it may be difficult to incriminate BVDV if there are no other concurrent signs of infection in the herd.

BVDV has been associated with ovaritis in infertile heifers (81). It was possible to re-isolate BVDV from the ovaries many months after they had seroconverted with no evidence of persistent viraemia. BVDV has also been incriminated in a disease similar in appearance to pustular vulvo-vaginitis caused by infectious bovine rhinotracheitis virus (1, 83). Pustular lesions have been described in the area of the vulva and vagina and have been associated with pruritus.

Immunosuppression

The topic of BVDV-induced immunosuppression has recently been reviewed (60) and is covered in detail elsewhere in this volume. In light of the current understanding
of the pathogenesis of mucosal disease there may be a tendency to decrease the importance of the role of BVDV in causing disease in immunocompetent, seronegative cattle. Although the predominance of evidence indicates that most infections in such cattle result in subclinical or mild disease, there is potential for more severe disease because of immunosuppressive effects of the virus. Thus, BVDV infection may potentiate or enhance the pathogenicity of co-infesting pathogens, such as parainfluenza virus type 3, infectious bovine rhinotracheitis virus, coronavirus, rotavirus, Pasteurella spp., Salmonella spp., Actinomyces pyogenes, coccidia and helminths (1, 4, 60). Whether immunosuppression, induced by BVDV infection, leads to the development of disease, it is undoubtedly tied to the complex interactions between host, environment and infectious agents. If management and environmental conditions are suboptimal and animals are in contact with a variety of potential pathogens, BVDV may enhance development of a secondary disease. Even modified live BVDV vaccines have been demonstrated to be immunosuppressive (76). Although the use of such vaccines in unstressed, well managed cattle herds may pose only a small risk, their use under some conditions (e.g. entry to a feedlot of recently weaned, comingsled, transported or otherwise stressed calves) may be ill-advised.

The nature of immunosuppression may differ depending on the form of BVDV infection which the animal is undergoing. Thus, the immunosuppression associated with acute BVDV infection appears to be different from that described for cattle persistently infected with BVDV. Possibly another form of immunosuppression is associated with acute and chronic mucosal disease.

**BVDV infection in neonatal calves**

The importance of BVDV as a cause of neonatal calf disease has been controversial. In light of the current understanding of the pathogenesis of BVDV infections and its association with the occurrence of persistently infected calves, the role of this virus in calfhood diseases needs to be reevaluated. Calves born persistently infected with BVDV (discussed below) may have poor viability and succumb to early disease and death (22). Disease in these persistently infected calves may be manifested as pneumonia and enteritis (5, 90). Thus, it becomes important to know if calves experiencing enteritis and pneumonia in association with BVDV are persistently infected or are immunocompetent and undergoing an acute infection. This information is not available in earlier reports, making it difficult to determine the importance of acute BVDV infection in immunocompetent calves.

In general, BVDV is considered rarely to cause disease in immunocompetent calves less than six months of age but it has been associated with and may contribute to outbreaks of pneumonia and enteritis (65, 84, 87). Fetuses may become infected during later stages of gestation or calves may become infected in the neonatal period and may develop severe enteritis which is sometimes fatal (1). Fatal enteritis has been experimentally produced in both colostrum-fed as well as colostrum-deprived neonatal calves (38). In older colostrum-fed calves (4-6 months), experimental infection resulted in mild clinical disease with rapid recovery (51). It has been suggested that BVDV may have a major role in neonatal calf diarrhoea (37, 38) but this contention has not been well substantiated. There is some field and experimental evidence that when present with other infective agents, BVDV can increase the severity of neonatal diarrhoea (89).

Passively acquired antibody in calves appears to provide protection against both systemic and respiratory infections with BVDV (27, 29, 80). Passively derived antibody
declines to undetectable levels between 105 and 230 days, but with initial high concentrations, antibody may persist up to 398 days (19). After decay of passively acquired antibody, titres increase following natural exposure and infection (36).

BVDV may have an important role in neonatal diseases of calves in the event of failure or partial failure of passive transfer of antibody, because of the suppressive effects of the virus on the calf’s immune system. Such a problem may be overcome in a herd through immunisation programmes in dams, followed by appropriate measures to ensure adequate intake of quality colostrum.

**BVDV and bovine respiratory disease**

It has been controversial whether BVDV has a role, and if so, to what extent it is involved in bovine respiratory disease. A recent review concluded that the existing published literature failed to support the contention that BVDV has a role in the pathogenesis of undifferentiated bovine respiratory disease (67). Although evidence is only circumstantial, BVDV may have a role in bovine respiratory disease because of its immunosuppressive effects on the host. The majority of research focusing on viral-bacterial synergism in bovine respiratory disease has dealt with infectious bovine rhinotracheitis virus and parainfluenza virus type 3 (94) and BVDV virus has not been as well studied in this regard. There still remains evidence that is suggestive of a role for BVDV in bovine respiratory disease (29, 30, 59, 69, 70, 85, 87). Despite the fact that BVDV is generally considered to be lymphotrophic and enterotrophic, it has been identified in outbreaks of respiratory disease, usually in association with other pathogens. In one study BVDV was the virus most often isolated from pneumonic lungs in cases of shipping fever in feedlot cattle and was usually found in association with *Pasteurella haemolytica* (70). A recent study reported BVDV to be the most frequent virus found in association with multiple virus infection in outbreaks of respiratory disease in calves (72). A seroepidemiologic study demonstrated an association between antibody titres to BVDV (as well as other viruses) and the treatment of respiratory disease (42). These findings suggest that BVDV may be involved as an initiator in bovine respiratory disease.

Results of experimental attempts to reproduce respiratory disease with BVDV have been variable. In several studies, experimental infection of calves with BVDV has only resulted in mild interstitial pneumonia (48, 58). When BVDV was administered by aerosol exposure there was no effect of the virus on the mean clearance rate of *Pasteurella haemolytica* from the lung (41). In another study a synergistic association between BVDV and *P. haemolytica* was demonstrated (63). Experimentally, initial infection with BVDV impairs the ability of calves to clear infectious bovine rhinotracheitis virus from the lung and to contain it at the infection site (62). Strains of BVDV may differ in their pneumopathogenicity. In one study, cytopathic and non-cytopathic strains caused respiratory tract disease when followed by inoculation with *P. haemolytica*, but the cytopathic strain was associated with more severe disease (61).

In conclusion, there is data suggestive of BVDV having a role in bovine respiratory disease, but further research is needed to document this contention. Such research should include further experimental studies as well as epidemiologic field studies.

**CONGENITAL INFECTIONS**

BVDV infection in an immunocompetent pregnant heifer or cow would be similar to that described under subclinical infections and BVD. The main importance of
BVDV infection during pregnancy is the outcome if transplacental spread of the virus to the conceptus occurs. Transplacental infection appears to be a probable event and occurs with remarkable efficiency if the dam undergoes an acute infection during pregnancy or is persistently infected (22, 73). The main determinant of fetal response is the gestational age of the fetus at the time of infection (74) and the most serious consequences follow exposure during early gestation. The possible outcomes include fetal resorption, abortion, mummification, congenital malformations and the birth of calves weak and undersized, calves persistently infected with BVDV or normal calves seropositive to the virus (Fig. 1).

**FIG. 1**  
Possible outcomes of fetal infection with bovine virus diarrhoea virus

**Fetal abortion, stillbirths and mummification**

BVDV does not appear to be a major cause of early embryonic death (64, 92) but fetal infection from 50 to 100 days of gestation may result in fetal death followed by abortion, stillbirth or mummification (17, 21, 22, 32, 35, 83). Expulsion of the fetus may occur up to several months after infection (34). The overall incidence of abortion caused by BVDV appears to be low (2-7%) but in non-immune herds a higher percentage may be encountered (1, 23, 33, 83).

Diagnosis of abortion induced by BVDV is complicated by several factors. Transplacental infections do not always cause abortion, therefore demonstration of virus, viral antigen or specific antibody from an aborted fetus does not necessarily mean the cause was BVDV. Paired serotesting of the dam may be unsuccessful in diagnosis because antibody titre may have already increased at the time of the abortion.
Congenital defects

Infection of the fetus between approximately 100 to 150 days of gestation can result in a variety of congenital defects (22). This period of development involves the final stages of organogenesis of the nervous system as well as the development of the fetal ability to mount an inflammatory response. Infection during this period may result in damage and destruction of stem cells, resulting in congenital defects at birth. The teratogenic lesions recognised in association with fetal BVDV infection have been described and are summarised in Table II (11, 12, 13, 17, 21, 22, 31, 32, 66, 74, 83, 88, 93). It should also be noted that modified live BVDV vaccines when administered to susceptible pregnant cows can cause fetal infections with the development of congenital defects.

**TABLE II**

**Congenital defects associated with transplacental infection of the fetus with BVDV**

<table>
<thead>
<tr>
<th>Nervous system</th>
<th>Immune system</th>
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<tbody>
<tr>
<td>Microencephalopathy</td>
<td>Thymic aplasia</td>
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<td>Cerebellar hypoplasia</td>
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<td>Hydranencephaly</td>
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<td>Forencephaly</td>
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<td>Hypomyelinogenesis</td>
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<td>Eye</td>
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<td>Retinal atrophy</td>
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<td>Cataract</td>
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<td>Microphthalmia</td>
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<td>Retinal dysplasia</td>
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<td></td>
<td><strong>Integumentary system</strong></td>
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<td>Hypotrichosis</td>
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<td><strong>Musculoskeletal</strong></td>
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<td>Brachygnathism</td>
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<td>Growth retardation</td>
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<td>Arthrogryposis</td>
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<td><strong>Respiratory system</strong></td>
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<td>Pulmonary hypoplasia</td>
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Calves born with cerebellar hypoplasia are reluctant to stand and can only do so with great difficulty. They have a wide base stance and are ataxic. Tremor is associated with calves born with hypomyelinogenesis and is present at birth. Congenital defect of the eye may result in varying degrees of blindness. Cataracts are readily apparent upon ophthalmic examination. Transplacental infection with BVDV may also result in retardation of fetal growth (21, 22). This is manifested by the birth of weak, undersized calves which may die shortly after birth.

**Normal calves born seropositive to BVDV**

Infection with BVDV in the later stages of gestation, after the development of immunocompetence, rarely causes congenital malformations. Calves may be normal at birth and have neutralising antibodies to BVDV (17, 49, 53). If these antibodies are to be detected, serum must be obtained prior to the ingestion of colostrum.
Immunotolerance

Infection during early stages of fetal development, prior to the complete maturation of the immune system, may result in immunotolerance to BVDV. These calves are born persistently infected with BVDV and remain so for life. Immunotolerance and persistent infection occur naturally and have also been produced experimentally. The precise stage of fetal development in which infection must occur to cause immunotolerance is unknown but it is an uncommon outcome after 100 days of gestation and can still occur at 125 days (18, 20, 39, 45). Thus far, immunotolerance and persistent infection appear to be associated only with non-cytopathic strains of BVDV (8, 43).

CHRONIC BVDV INFECTIONS

Persistent infection with BVDV

The prevalence of persistently infected cattle in the general population is not precisely defined and there is considerable variation likely between geographical areas and among individual herds. A study from the United States reports that 1.7 to 1.9% of cattle in selected herds were persistently infected (8). The frequency of persistent infection is probably overestimated in that the survey was not random and several of the herds were selected because of a history of BVDV infection. A study in Denmark revealed that 0.9% of healthy cattle at slaughter were viraemic, but it was not determined if these animals were transiently or persistently infected (46). In the United Kingdom, a prevalence of 0.8% was reported for viraemia and 0.4% for persistent infection (28) and similar findings have been reported from Germany (57). In herds with a history of recent BVDV outbreaks, higher prevalences for viraemia and persistent infection have been reported (46, 79).

Cattle immunotolerant and persistently infected with BVDV are viraemic, constantly shed virus into the environment, have no or low concentrations of specific neutralising antibodies (with the exception of persistently infected calves that have ingested colostrum containing specific BVDV antibody) and may appear healthy (5, 18, 20). Although persistently infected cattle are immunotolerant to BVDV, they are immunocompetent with respect to other antigens (45). Immunotolerance appears to be specific to the infecting non-cytopathic strain of BVDV in that persistently infected cattle are capable of an immune response to heterologous strains (9, 10, 82). In spite of this antibody response the persistently infecting virus continues to infect the host.

From an epidemiologic standpoint, persistently infected cattle are an important source of viral transmission to susceptible animals. Persistently infected cattle are efficient transmitters, shed large quantities of virus into the environment over prolonged periods of time and the spread to susceptible cattle in contact is rapid (22, 73). It appears likely that persistent infection is the major mechanism by which the virus persists in the cattle population. If persistently infected females reach breeding age, offspring will be persistently infected (44, 86). Affected families may thus develop and provide a means to maintain the virus in a herd (40).

Persistently infected, immunotolerant cattle are at risk for development of mucosal disease if superinfection with a cytopathic strain occurs (discussed under mucosal disease). In addition to mucosal disease, persistently infected calves appear to be at
risk to other diseases, although the pathogenesis has not been defined. Persistently infected calves may have a death rate of 50% in the first year of life (22) and may be smaller at birth, have a slower rate of growth and may die or be culled from the herd for being a "poor doer". In one study there was a significantly higher death rate among calves born to cows known to be infected with BVDV while pregnant than those of seropositive dams (5). Persistently infected calves may be predisposed to infection by other micro-organisms which are manifested most often as enteritis and pneumonia (5, 90). Immunosuppression has been reported in persistently infected calves (60, 75, 77), which may account for the increased susceptibility to diseases such as enteritis and pneumonia. Subclinical disease in the form of glomerulonephritis and encephalitis has been described in persistently infected, but otherwise normal appearing cattle (20).

**MUCOSAL DISEASE**

Mucosal disease is a sporadic form of BVDV infection in cattle generally between the age of six months and two years. The disease is characterised by severe clinical signs, low morbidity and high case fatality. Recent studies have indicated that mucosal disease occurs when cattle that are immunotolerant and viraemic with non-cytopathic virus, become superinfected with a cytopathic strain of BVDV which shares close homology with the persistently infecting non-cytopathic strain (9, 15, 43). Pathogenesis of mucosal disease is discussed in detail elsewhere in this volume.

The clinical signs, clinical pathology and post-mortem findings associated with the acute and chronic form of mucosal disease have been described (1, 4, 7, 14, 22, 23, 26, 31, 34, 50, 56, 66, 78, 83) and the following descriptions have been derived from these references.

**Acute mucosal disease**

As previously mentioned, acute mucosal disease is a sporadic form of BVDV infection and generally involves cattle ranging in age from six months to two years. Usually less than 5% of the herd is affected but case fatality rate approaches 100%. Occasionally epizootics may involve up to 25% of the animals in a herd.

Acute mucosal disease is characterised by pyrexia (40.5 to 41°C), depression, weakness and anorexia. Heart and respiratory rate are elevated (polypnea and tachycardia). Emaciation and dehydration with acidosis develop as the disease progresses. In lactating cattle, milk production decreases.

Careful examination of the oral cavity may reveal erosive lesions involving the lips, gingival margins, tongue, dental pad, commissures of the mouth and posterior part of the hard palate. Lesions may coalesce to form large areas of necrosis and sloughing in the oral cavity. Erosive lesions can develop on the external nares and in the nasal cavity and there are reports of similar lesions on the vulva and teats. Blunting of oral papillae may develop.

Ptyalism (salivation) often accompanies the oral lesions which are generally found in 75 to 80% of the cases. Mucopurulent nasal discharge is often observed, with lacrimation and corneal oedema being sometimes observed.
Lameness, reluctance to move and recumbency may be seen, and these are attributable to erosive lesions and necrosis of skin in the interdigital cleft. Laminitis and coronitis may also be observed.

Profuse watery diarrhoea generally develops two to three days after the onset of clinical signs, but in peracute cases death may occur prior to the onset of diarrhoea. Faeces are foul-smelling and may contain variable amounts of fresh or clotted blood. Fibrinous intestinal casts may be passed. Straining at defecation is often observed. Ruminations are decreased and mild to moderate bloat may develop.

Severe leukopenia may be recognised in the early stages of disease. Neutropenia without a left shift, lymphopenia and thrombocytopenia may develop. Secondary bacterial infections are common. Death may occur in the acute phase of the disease, but more frequently takes place at three to ten days after onset of signs.

The distribution of lesions in mucosal disease relates to the affinity for and direct necrotising effects of BVDV on epithelial tissues of the gastro-intestinal tract, integument and respiratory tract. The virus also has an affinity for endothelial cells of vessels, particularly in the intestines and lymphoid tissue.

Post-mortem findings include erosions throughout the alimentary tract, commonly in the oesophagus, ruminal pillars, omasum, abomasum and intestines. Ruminal papillae may be reduced in size. The pyloric portion of the abomasum is oedematous and haemorrhagic. Bowel contents are dark, watery and foul-smelling. Catarrhal enteritis may progress to include haemorrhage, erosions and ulcers of the intestinal mucosa. Peyer’s patches often are swollen, necrotic and haemorrhagic. The mucosa of the large intestines may be congested often in a “stripping” pattern which follows the mucosal folds.

Histologic examination reveals varying degrees of necrosis in the germinal centers of lymph nodes and spleen. Oedema and inflammatory cell infiltration is seen in varying degrees throughout the gastro-intestinal tract.

**Chronic mucosal disease**

A small proportion of cattle that develop mucosal disease do not die in the expected time frame and become chronically affected. These cases have been referred to as chronic BVD, but chronic mucosal disease is a more descriptive term and possibly more accurate with respect to pathogenesis. The pathogenesis of chronic mucosal disease has not been defined, but it may share similarities with that of acute mucosal disease. As in acute mucosal disease, both non-cytopathic and cytopathic virus have been isolated (43). It has been suggested that the differences in antigenicity of the superinfecting cytopathic virus may give rise to different clinical responses in the persistently infected animal (16). One extreme may be acute mucosal disease when the superinfecting virus shares close homology with the persistently infecting non-cytopathic strain. The other extreme is no clinical disease, but seroconversion occurring when the superinfecting virus is a heterologous strain. Between these two extremes may be the clinical entity described as chronic mucosal disease. Perhaps another form of mucosal disease exists from which recovery is possible.

Chronic mucosal disease is characterised by inappetence, weight loss, progressive emaciation and an overall unthrifty appearance. Diarrhoea may be continual or intermittent. Chronic bloat may be observed. Nasal discharge and persistent ocular
discharge are frequent findings. Areas of alopecia and hyperkeratinisation may develop, usually on the neck. Chronic erosive lesions often are found in the mouth and involving the skin. Areas of the skin most often affected include the perineal area, prepuce, opening, vulva, skin/horn junction, around the dewclaws, interdigital cleft and the heels. The failure of skin lesions to heal is an important finding in chronic cases of mucosal disease. Chronic lameness may develop because of laminitis, interdigital necrosis and hoof deformities. Secondary bacterial infections are common. Chronic pancytopenia is characterised by anaemia, leukopenia, neutropenia and lymphopenia.

Cattle with chronic mucosal disease may survive up to 18 months and ultimately die from severe debilitation. They should be distinguished from calves born persistently infected, which are unthrifty and "poor doers" from birth. Such calves may potentially develop mucosal disease but, as previously discussed, may succumb to other infections in which case the signs and lesions of mucosal disease would not be present.

Treatment of either form of mucosal disease is unwarranted. Although immunomodulating drugs have sometimes been used in treatment of chronic mucosal disease, it is unlikely that these drugs will have any long-term effect. A diagnosis should be made as rapidly as possible and the affected animal should be removed from the herd. Testing of the herd is then indicated to determine the presence of other persistently infected cattle which should also be removed.

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ASPECTOS CLÍNICOS DE LA INFECCIÓN POR VIRUS DE LA DIARREA VIRAL BOVINA. — J.C. Baker.

Resumen: La infección de bovinos por virus de la diarrea viral bovina (virus BVD) da lugar a gran cantidad de manifestaciones clínicas. Este artículo pasa revista a las respuestas clínicas asociadas al virus BVD y estudia estas enfermedades según la clasificación siguiente: infección aguda de los bovinos inmunocompetentes, infección del feto, infección de los bovinos inmunotolerantes e infectados de manera persistente por el virus BVD y, por último, enfermedad mucosa.

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