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Bovine Respiratory Syncytial Virus Infection

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Bovine Respiratory Syncytial virus (BRSV) has quickly become one of the major respiratory viruses in cattle in the U.S. Literary reports from various areas of the U.S. and Canada show that BRSV is present in 38 to 81 percent of the beef and dairy cattle tested.1,3

Bovine Respiratory Syncytial virus, first isolated in Switzerland in 1970 from an outbreak of respiratory disease involving dairy cattle, is a nonhemagglutinating pneumovirus in the family Paramyxoviridae.2,3,11 The virus was named for its characteristic property of promoting fusion of cells into multinucleated giant cells (syncytial cells).

Pathogenesis
The mechanism by which BRSV causes disease is not clearly understood. A common association of secondary bacterial pneumonia in outbreaks of BRSV indicates that this virus has a catastrophic effect on the respiratory tract defense mechanisms. Scanning electron photomicrographs made after experimental infections have demonstrated destruction of ciliated respiratory epithelium.4 By causing extensive damage to the mucous membranes, the virus leaves the respiratory tract susceptible to dust, debris, and secondary infectious agents such as bacteria and other viruses.

The pulmonary edema-emphysema and pneumonia caused by BRSV is, in theory, due to a hypersensitivity reaction.4,16 This theory is based on the observation that two separate clinical disease syndromes are observed.2 The initial stage is characterized by mild respiratory signs, except when complicated by secondary bacterial pneumonia. The second stage, which may follow improvement or recovery from the initial stage, is characterized by severe respiratory distress. Because of this biphasic respiratory disease syndrome, it is thought that the initial exposure may sensitize the animal, with subsequent exposure inducing an anaphylactic or hypersensitivity reaction.16

Disease Incidence
The virus affects both dairy and beef cattle, ranging in age from ten days to maturity.17 Age, management, and environment seem to affect the animals' susceptibility.

In beef calves, the infection is most severe during the fall, when they are 5 to 10 months of age.2 Such outbreaks coincide with weaning, feedlot shipping, dietary changes, and other stressors that suppress the immune system. An association has also been reported between more severe outbreaks and well managed calves on good rations.2

The virus is most likely transmitted to susceptible calves by aerosol, direct contact, and droplet contact from subclinically or clinically infected calves.2 The spread of the disease is especially efficient in closely confined herds. The virus is very labile outside the body and is therefore unlikely to persist in the environment. Cattle are probably the reservoir for the virus.6 Experiments with BRSV infected dams have shown that calves are not protected by colostral or passively derived antibodies.15 This is important to BRSV susceptibility of very young calves.

The severity of disease varies considerably from group to group, and within groups of cattle. However, almost all calves within a group will be affected, even though fever may be the only abnormality.6 Death rate also varies greatly, 0 to 25 percent, with the highest mortality occurring in well-bred animals on a high plane of nutrition.2,6 Certain feeds like corn silage have been associated with an increased incidence or severity of disease.

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Clinical Signs

The incubation period for the disease is three to five days and cattle show few clinical signs before this time. Early clinical signs of disease include mild nasal, oral, and ocular discharge, increased respiratory rate, decreased feed and water intake, and an increased frequency and severity of coughing. Many of the calves, even those that appear normal, will have elevated temperatures ranging from 104 to 108°F. Affected calves may stand alone with their heads down, although they brighten up remarkably well when an observer walks among them. Consequently, to the inexperienced observer, the first clinical sign may be death.

Early signs may be followed by acute disease within hours. At this time extreme respiratory distress is seen, including pronounced dyspnea, mouth breathing, a dry hacking cough, and frothing of saliva from the mouth. Subcutaneous edema may be evident, especially around the eyes, throat and neck. The animal may be seen with tongue hanging out and neck extended. The calves do not eat or drink, they lose fill, and become dehydrated. Diarrhea has been reported but tends to be transient, since decreased feed and water intake leads to constipation. Duration of the disease ranges from one to two weeks.

Auscultation of the lungs reveals increased bronchovesicular sounds with fine crackles, which are consistent with the presence of emphysema. Rales and rhonchi are also commonly heard due to the presence of secondary bacterial infections in the lungs.

Although clinical signs and epidemiologic features observed during outbreaks of respiratory disease may make a veterinarian suspicious of BRSV involvement this diagnosis should be confirmed by laboratory tests, since there are many different agents involved in the bovine respiratory complex.

Pathological Findings

On necropsy, there is a diffuse interstitial pneumonia with subpleural and interstitial emphysema and edema. The lungs appear reddish pink, are firm and have the meaty texture of adenomatosis. The lungs do not collapse when the thoracic cavity is opened, and often imprints of the ribs can be seen on the surface of the lungs. A foamy fluid flows from the affected portion of the lung when cut, and there is froth in the trachea and bronchi. If secondary bacterial pneumonia is present, areas of the lungs may be consolidated, especially in the cranioventral regions.

Histopathologic changes associated with BRSV vary with the duration of infection, and with the presence or absence of secondary infection. The most severe changes are seen in small bronchi, bronchioles, and alveoli. These changes include necrotizing and hyperplastic bronchitis and bronchiolitis, multifocal to locally extensive interstitial pneumonia, and often in fatal cases, interstitial emphysema. The most characteristic histological hallmark of BRSV infection is the formation of multinucleated syncytial cells in the epithelial cells lining the bronchi, bronchioles and alveolar septa.

Diagnosis

A definitive diagnosis of BRSV cannot be confirmed without laboratory tests. However, it is often difficult to obtain a positive laboratory diagnosis because the virus is difficult to isolate, and it is very labile in transit under field conditions. As a result, practitioners must collect proper specimens, and process and package them properly before sending them to a lab.

When collecting samples for testing, the obviously sick animal is not the ideal candidate because it is usually not shedding the virus. In addition, the animal's antibody response may neutralize the virus, thus preventing virus isolation. The best samples are from those calves in contact with the sick animals, or with calves that are febrile and will be sick within four days. Calves incubating BRSV are more likely to shed the virus. The best samples to submit for diagnostic testing include deep nasopharyngeal swabs, transtracheal samples, serum, and, if the calf dies or is euthanatized, fresh lung samples from areas caudal to those of bacterial lesions. The procedures which are currently available for diagnosing BRSV include virus isolation, immunofluoresence testing, paired serotesting, and histopathology which is only suggestive of BRSV infection.

Since isolation is time consuming and lacks sensitivity for detecting BRSV, it should be used in conjunction with other tests. The most common samples for virus isolation are nasopharyngeal swabs, lacrimal discharge, bronchial lymph nodes, and lung. The virus is labile and rapidly loses viability during transport or if subjected to freezing and thawing. To increase the chances for isolation, the samples should be taken early in the course of an infection. If isolation is going to be made on lung tissue, the samples must be taken as soon as possible after death. To enhance the viability of the virus, several researchers recommend the use of a sucrose transport medium, and storage under iced or frozen conditions.
Immunofluorescence has proven to be a highly sensitive and specific test for the rapid diagnosis of BRSV infection. Frozen lung and bronchial sections, along with nasal swabs collected from calves early in the course of the disease, are the best samples for this test. Nasal swabs should be rolled onto microscope slides, fixed in acetone for ten minutes, and then sent to the lab for fluorescent antibody testing. Such testing can be performed in a few hours, is specific, and inexpensive.

Serologic testing and comparison of paired serum samples will allow the practitioner to evaluate a herd for the presence of BRSV. Serologic diagnosis is made by comparing acute and convalescent serum antibody titers for evidence of seroconversion (fourfold rise in antibody titer). When attempting a serological diagnosis of BRSV, samples should be submitted from ten animals or ten percent of the herd, whichever is greater. Antibody titers to BRSV rise very rapidly within two to three days after the onset of clinical signs. Therefore, it is important that serum samples be collected as early as possible in a disease outbreak. Convalescent samples can be collected as early as two weeks.

A serologic diagnosis is not possible in calves with passively derived antibody because the passive immunity, which is not protective, suppresses the humoral immune response. A newly developed ELISA test for the detection of BRSV serum antibodies has been shown to be a highly sensitive test.

Histopathology is the final method used in diagnosing BRSV infection. The lesions have already been discussed elsewhere in this paper. Histopathology does not give a definitive diagnosis because similar lesions occur in other viral and bacterial diseases of the lung.

Treatment

Early diagnosis improves the chances of successful treatment of BRSV infected animals. All animals in an affected herd should be given antibiotic therapy to prevent the occurrence of secondary bacterial pneumonia. The therapy should begin as soon as respiratory disease is recognized, and continued for several days after apparent recovery. Antibiotic therapy should be initiated while bacteriologic cultures and sensitivity tests are pending.

In some field outbreaks, the use of corticosteroids and antihistamines have shown favorable results in the treatment of BRSV. Because of the immunosuppressive effect of corticosteroids, a diagnosis or strong suspicion of BRSV is indicated before initiation of this form of therapy.

Aspirin and Banamine® (flunixin meglumine) have been used to help reduce rectal temperatures, and bronchial mucosal swelling leading to a reduction in airway obstruction.

Other recommendations in the treatment of BRSV include withholding grain and silage from affected animals for two to three days, and feeding only enough hay to identify those animals that are not eating. Calves that are severely affected and unable to drink need to be given supportive therapy consisting of fluids and electrolytes.

The following two treatment regimens have been used extensively, and have shown considerable success when started early.

1. a. Fast the affected group of calves for two to three days; feed only enough hay to enable identification of anorexic calves.
   b. For two days, give sick calves 250mg pyrilamine maleate twice a day and 10mg dexamethasone once a day.
   c. Start antibiotic treatment by the second day of clinical signs; also give sustained-release oral sulfas; in large groups of calves, treat with antibiotics in the water.
   d. On the second or third day of treatment, give severely dehydrated calves electrolytes and a rumen preparation by stomach tube.

2. a. Change or reduce feed as necessary.
   b. Treat febrile calves with aspirin (240g bolus/306 #) once, or preferably twice.
   c. Treat parenterally with broad-spectrum antibiotics for 4 to 5 days; treat large groups with sulfas in the water and tetracyclines in the feed.
   d. Give required supportive treatment as in regimen 1.

These treatments are for calves in the late syndrome of BRSV. Early syndrome calves can be treated with antibiotics only, to prevent secondary bacterial pneumonia, but individual calves should be watched carefully for respiratory distress, and then treated as above when necessary.

Prevention

Good managerial practices alone do not prevent BRSV, but they will help prevent excessive losses. Because it is difficult to completely control this disease, vaccines have been developed to control BRSV.
BRSV vaccines have recently become available in the U.S., and are produced by Norden Laboratories and Diamond Scientific. These products are modified live vaccines that are given intramuscularly. A report of field trials using one vaccine indicated it was safe and effective in reducing the prevalence of respiratory disease in cattle.8,14 These vaccines are well suited for use in preconditioning programs, or at the time of entry into feedlots. By this time, calves have probably lost their passive immunity to BRSV and are likely capable of mounting an active immune response when vaccinated.16 Dairy calves should be vaccinated when passive immunity is lost. Pregnant beef cows should be vaccinated at the time of pregnancy checking, or closer to calving.

Vaccination for BRSV is not recommended during an outbreak. During an outbreak, the wild strain of BRSV is already in circulation, or the infected animals have already started to develop antibodies to the virus.2 In both cases, the success of vaccination is doubtful.

Summary

BRSV is a disease thought to result from the interaction of BRSV virus with several other etiologic factors which may differ from outbreak to outbreak. When disease is produced, it is usually on a herd basis, perhaps because calves of a highly susceptible age are grouped together, with concurrent exposure to other microbial agents and simultaneous exposure to other stressors, such as cold weather or dietary antigens, lowering herd resistance.

BRSV is often explosive in nature and is characterized by pulmonary edema and emphysema. It has a high morbidity and mortality among young calves. In addition, BRSV plays an important role in predisposing calves to secondary respiratory infections, especially bacterial pneumonia. Both aspects of the disease have a strong economic impact in treatment and loss.

Treatment of the acute disease is usually successful if it is started early in the course of the disease. However, it is expensive and intensive, including the use of antibiotics, antihistamines, and corticosteroids. In the early stages of the disease, fasting or a change of feed may eliminate an outbreak.

Preventing BRSV infection requires sound managerial practices and minimal stress. However, the well-managed, well-nourished herds are often hit the hardest. Vaccines for the prevention and control of BRSV appear to be both safe and cost-effective.

REFERENCES