THE SCIENCE WORLD
A Monthly e Magazine
ISSN:2583-2212
Popular Article
November 2024 Vol.4(11), 5036-5037

Infectious Bovine Rhinotracheitis

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https://doi.org/10.5281/zenodo.14097444

Introduction

Infectious bovine rhinotracheitis (IBR) has been described as "an acute; contagious, febrile infection of cattle, characterized by an intense inflammation of the upper respiratory passages and trachea, and accompanied by dyspnea, depression, nasal discharge, and loss of condition".

It was known by names such as "red nose", "dust pneumonia" and "infectious necrotic rhinotracheitis. The first published report on respiratory IBR came from Schroeder and Moys in 1954. The following year, Miller recognized that this condition was related to the one described from California and commented that the cause was undetermined but presumed to be a virus. That same year, the accepted name for the disease became infectious bovine rhinotracheitis.

Etiology:

Bovine Alphaherpesvirus type 1 (BoAHV1) is a virus of the Herpesviridae family, subfamily Alphaherpesvirinae, genus Varicellovirus, known as the causative agent of infectious bovine rhinotracheitis (IBR).

Species affected:

Cattle are believed to be the only significant natural host of the virus, serological and isolation techniqueshave been used to implicate several other species in natural infections, including goats, pigs, deer, water buffalo, and several African species, including buffalo, eland, antelope, wildebeeste, impala and hippopotamus, and man.

Transmission:

Infection occurs via the respiratory and genital routes. The virus is spread both within and between herds mainly by horizontal transmission such as direct and indirect contact (fomites) and aerosol droplets, or from infected bulls by coitus and in infected semen either by artificial or natural insemination. Frozen semen is held under optimal conditions for virus survival. As with other herpesviruses, infection with BHV-1 results in lifelong latent infection. This may occur in the absence of clinical signs and in the absence of detectable serum antibody.

Pathogenesis:

Replication of BHV-1 takes place in the mucosal epithelial surfaces of the upper respiratory tract and genital mucosa and virus is shed in nasal and genital secretions. Semen may be contaminated during ejaculation. Local nerve cell endings are infected and the virus is transported to trigeminal and sacral ganglia where it establishes a lifelong latent infection. Once infected, animals become lifelong carriers of the virus. The latent infection may be reactivated periodically, with or without clinical signs; the virus is transported back to the site of entry and is shed with potential transmission to other animals. Most, if not all, seropositive animals are latently infected and virus shedding can be reactivated following stress or corticosteroid treatment. Viremia is rarely detected, but does occur.

Clinical signs

There are three forms: respiratory, genital and encephalitic, the first two are more common.

Respiratory form:

The respiratory form of the disease is most frequently observed in cattle managed under intensive conditions (for example, feedlots) and is not often noticed in cattle under grazing conditions. The mucosa of the nares becomes reddened and shallow erosions may be present. Some animals develop excessive salivation. Oral lesions, which are uncommon, consist of shallow erosions of the oral mucosa. Some animals develop unilateral or bilateral conjunctivitis and have a clear ocular discharge, which may later become muco-purulent. In feedlot or other intensively managed cattle, there can be a severe necrotising laryngotracheitis and pneumonia that is complicated by secondary bacterial infections. These infections are usually encountered within the first 3-4 weeks of animals entering a feedlot.

Genital form:

Genital infection with BHV-1 occurs in both sexes and is a more frequent manifestation of this herpesvirus infection in cattle on pasture. The infection may result in the development of vesicles, pustules and erosions or ulcers in the mucosa of the vulva and vagina or on the penis and prepuce.

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Published 11/11/2024

In females a painful condition known as infectious pustular vulvovaginitis (IPV), may be observed within a few days of mating. Frequent micturition and rising of the tail are the first clinical signs. There may be hyperaemia or oedema of the vulva and the posterior third of the vagina. Small red to white ulcers develop into pustules (0.5–3 mm in diameter). There may be a thick yell.

The disease in bulls is known as infectious pustular balanoposthitis (IPB). After a 2–3-day incubation period, pustules appear on the mucosal surface of the penis and prepuce.10 These pustules can progress to ulcers with a mucopurulent discharge and may prevent a bull from serving. A proportion of infected bulls will also excrete virus in their semen. In turn, infected semen can infect susceptible females, by natural or artificial insemination. ow or white mucopurulent exudate, especially in cases complicated by secondary bacterial infection.The course of the disease is variable among individual animals and, apparently, among outbreaks. Uncomplicated cases of respiratory and genital forms usually resolve in 5-10 days.

In encephalitic form the brain may be affected in calves below 6 months causing high mortality.

Diagnosis:

Samples for Diagnosis: Blood and serum samples should be chilled (2-8^{o}C) during transport to the laboratory and samples should be processed in the laboratory as soon as possible after arrival. Freezing sera at -20^{o}C or lower is preferred for long-term storage but repeated freeze-thaw cycles should be avoided. Swabs should be placed in viral transport medium (phosphate buffered gelatine saline containing antibiotics – PBGS) immediately after collection and chilled while being transported.

If semen sample is used for diagnosing for each individual batch of semen that has been submitted, a minimum of 5 straws of extended semen is required. If there are multiple collections from the same animal on different days, these requirements still apply: 5 straws are needed from each semen batch. Due to the low levels of virus that may be present and the likelihood that the biological material (especially semen) has been diluted after collection, it is expected that a specified minimum volume of material will be collected. In most cases this will be 1–2 mL, representing 5–10 straws of commercially collected extended semen.

Antibodies are detected in the serum of most animals within 2–3 weeks of infection. Maternally-derived antibodies may be detected for up to 7 months, but usually disappear in about 4–5 months. The serological tests commonly used for testing for BHV-1 antibody are the virus neutralisation test (VNT) and Ezyme-linked immunosorbent assay (ELISA) for antibody detection.Virus isolation is used routinely for diagnostic purposes. Usually virus isolation is attempted on swabs that have been collected from lesions in the respiratory or reproductive tracts as early as possible during the course of the disease. The transport medium into which

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the swabs have been placed is subsequently used to inoculate susceptible cell cultures. The presence of BHV-1 virus is detected by the development of characteristic changes in the monolayer cell cultures. Any cytopathogenic agent detected is then identified by neutralisation with specific antiserum or by immunoperoxidase or immunofluorescent staining to confirm its identity. The reliable diagnosis of BHV-1 infection by virus isolation depends on the availability of cell cultures that have proven susceptibility to BHV-1 virusA range of bovine cells are suitable for BHV-1 isolation and serology that include Primary cells usually derived from testis, lung or kidney tissue. Recently PCR has been used to detect BHV-1 virus in semen. Although this assay was not evaluated for disease diagnosis, the level of sensitivity described should allow its application for diagnostic purposes.Electronmicroscopic evaluation of the vesicular fluid or scrapings.

Prevention and Control:

Purchase new animals only after testing them and induct only negative animals to farm. Though vaccination is a method of prevention, no vaccines are produced in India presently for IBR Consult a veterinarian immediately if the above symptoms are seen to prevent the disease from spreading. The control efforts are based on hygiene management, isolation procedures and broad-spectrum antibiotic treatments to avoid the challenge of other bacterial infections. Eradication is recommended in the areas where the prevalence of the prevalence of the infection is not high.

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