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Popular Article

Bovine Respiratory Syncytial Virus (BRSV): Most Underestimated Killer in Young Calves

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Introduction

In the early 1970s Bovine respiratory Syncytial virus (BRSV) was recognized as a pathogen in cattle which was responsible for an acute respiratory disease syndrome in beef and dairy calves (Wellemans et al.1970). BRSV is a non-segmented, enveloped, negative-stranded RNA virus belonging to the *Pneumovirus* genus within the subfamily *Pneumovirinae* (Socha et al. 2009; Blodörn et al. 2015). BRSV infection frequently occurs in young calves aged between 2 weeks and 9 months (Baker et al. 1993; Hoppe et al.2018). It is most significant causes of lower respiratory tract infections in calves while in case of adult animals it causes subclinical disease which act as major source of infection in the herds so reinfections are quite common (Baker et al. 1993; Hoppe et al.2018). These infections reported to cause 60% to 80%, morbidity rate and around 20% of mortality rates (Urban-Chmiel et al. 2015). It is highly widespread isolated from cattle in Europe, America and Asia and most important viral cause of bovine respiratory disease (BRD) worldwide (Valarcher and Taylor 2007; Blodörn et al. 2015).

BRSV is closely related to Human Respiratory Syncytial Virus (HRSV), which is a major cause of respiratory disease in young children; both viruses are similar and have similar epidemiological, clinical, and pathological characteristics (Valacher et al. 2007). Before 2015, BRSV was classified into the Pneumovirus genus, Paramyxoviridae family (ICTV 2015). However, currently revised list entered BRSV as bovine orthopneumovirus species and classified it into the Orthopneumovirus genus within the newly created Pneumoviridae family (ICTV 2018).

Genomic RNA of BRSV has around 15,000 nucleotides in length encoding 10 proteins. BRSV has lipid envelope consisting of three surface glycoproteins, defined as the attachment glycoprotein (G), fusion (F) protein, and the small hydrophobic (SH) protein (Sarmiento-Silva et al. 2012). The F and G genes are the major targets of the immune system and plays important roles in viral infectivity (Valarcher and Taylor 2007, Guzman and Taylor, 2015). BRSV also encodes an RNA regulatory protein M2-2 and two non-structural proteins, NS1 and NS2. These two non-structural proteins were more in infected cells. The envelope encloses the matrix protein (M) on the inner face while nucleoprotein (N), phosphoprotein (P), the viral RNA-dependent polymerase (L) and transcriptional anti-termination factor known as M2-1 constitute the nucleocapsid. The M2-2 protein is expressed at low levels in infected cells; however, it there incorporation into virions is not clear (Sarmiento-Silva et al. 2012; Guzman and Taylor, 2015).

BRSV outbreaks commonly occur in winter, although infection can occur throughout the year. Younger age groups are more susceptible to BRSV infection. BRSV infection causes high morbidity up to 80% and mortality can reach up to 20% in certain outbreaks (Sarmiento-Silva et al. 2012; Klem et al. 2013). Management factors also affects spread of disease like animals co-housed densely sharing same feeder trough and water container leads to ascending infection. The close exposure of animals to animals plays critical role in spreading of virus among animals (Goswami et al. 2017) and predisposes calves to secondary respiratory infections, especially bacterial pneumonia (Prieksat and Thompson, 1988). Exotic breeds were more susceptible compared to crossbred cattle for BRSV infection (Hazari et al. 2002)

BRSV infection has incubation period of 2 to 5 days. It induces a well-defined pathology starting from fever, cough, and mucoid nasal discharge. The fever could increase up to 40 °C along with depression, increased respiratory rate and anorexia. Wheezing is recorded in auscultation of the lungs of most severe cases. Overproduction of mucus is particularly detrimental as the airways can quickly become obstructed. If appropriate innate immune response is present, it can be beneficial but uncontrolled responses lead to disease.

BRSV has characteristic cytopathic effect, giant multinuclear cells (Syncytial cells) formed by the fusion of several cells, in infected tissue. BRSV has a predilection for the lower respiratory tract, primarily in ciliated airway epithelia cells and type II pneumocytes, where it can cause varying degrees of pneumonia by itself (Welliver et al. 2008; Guzman and Taylor, 2015; Zoetis, 2019).Virus infected epithelial cells are cleared by apoptosis followed by phagocytosis by neighboring epithelial cells and virus present in respiratory lumen are cleared by neutrophils (Viuff et al. 2002).

On experimental inoculation BRSV was recovered from nasal secretions on days 2 to 6 and viral antigen was shown deep in the lungs 2 to 4 days (Brodersen, 2010). Clinical signs like fever, cough, dyspnea, respiratory distress, abdominal breathing were most prominent features (Peixoto et al. 2000**;** Sacco et al.2014). Gross lesions are commonly seen in cranioventral lung lobes, characterized by atelectasis. Consolidated lesions are distributed multifocally throughout the cranial, middle, and accessory lobe (Sacco et al. 2014). Histologically, extensive moderate to severe broncho-interstitial pneumonia as well as purulent bronchitis and bronchiolitis present (Caswell and Williams, 2008; Blodörn et al. 2015). BRSV infects both type I and II pneumocytes, causing type I pneumocyte necrosis and induces proliferation, hypertrophy and necrosis in type II pneumocyte. Syncytial cells also seen in alveoli (Peixoto et al. 2000; Elsayed et al. 2014).

Molecular approaches are quickly becoming the gold standard for the correct identification and characterization of BRSV in clinical cases. To detect viral genes in nasal or bronchoalveolar lavage nucleic acid-based detection methods have become quite common. These methods which includes conventional RT-PCR also helps in studying molecular epidemiology of BRSV infections (Socha et al. 2009; Yaegashi et al. 2005; Kovarcik & Valentova, 2004).

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