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Significance of *Bordetella bronchiseptica* in Respiratory Tract Infections of Canines

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ABSTRACT

Bordetella bronchiseptica is one of the major pathogens affecting canine respiratory tract. The agent is transmitted through aerosol mode, contact with contaminated faces or urine as well as through infected fomites. The agent affects all age groups and breeds of dogs. It is a contagious one that can transmit to other dogs easily and it's having zoonotic potential. Because of all these reasons *Bordetella bronchiseptica* infection got attention of researchers. *Bordetella bronchiseptica,* a gram-negative cocci bacterium causes respiratory discomfort in dogs as well as other animals. The sequential synthesis of virulence factors decides the pathogenesis of *B. bronchiseptica viz.,* adhesions, toxins, and other bacterial products that may alter host functions, facilitate immune evasion, or otherwise assist in transmission or survival BvgAS controls bacteria's virulent components. *B. bronchiseptica* causes a variety of clinical signs which develop within 3 to 4 days of exposure and, may persist for up to 14 days. Signs are coughing, gagging or retching and mild serous oculo-nasal discharge. Affected dogs usually remain non–febrile, alert and active. *B. bronchiseptica* diagnosis depends on isolation, identification using biochemical, serological, and molecular techniques. Culture, isolation molecular diagnostics are required to fully understand the bacteria details. Precaution such as isolation before re-entering the population, routine monitoring for the emergence of clinical signs with the group, and quarantine protocols for dogs should be done. Additionally, obtaining vaccinations and taking precautionary measures can help in reducing the chances of getting the animal infected.

KEYWORDS: *Bordetella bronchiseptica*, bacillus, canine, cough, kennel, respiratory

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1. INTRODUCTION

Bordetella bronchiseptica was recognized as a respiratory pathogen of mammals since 1910. Ferry in 1910 first isolated *B. bronchiseptica* and was identified as *Bacillus bronchocanis* (Gueirard et al., 1995). The name *Bacillus bronchocanis* was chosen since the organism was isolated from the respiratory tract of dogs suffering from distemper. Bordetella attaches itself to the ciliated epithelium during its colonization of the upper respiratory tract in mammals. The attachment is achieved by expression of the adhesions such as fimbriae, filamentous haemagglutinin and pertactin (Edwards et al*.,* 2005).

B. bronchiseptica are small (0.2–0.5 μm in diameter and 0.5–2.0 μm in length) gram negative, motile, aerobic coccobacilli belonging to class of Beta Proteobacteria, order Burkholderiales and the family Alcaligenaceae (Kadlec and Sehwarz, 2018). *B. bronchiseptica*, an upper respiratory tract pathogen, infects a wide variety of host including domestic and wild animals and also opportunistically infects humans. Bordetella is involved in the diseases such as kennel cough in dogs and atrophic rhinitis in swine (Melvin et al., 2014). Based on a number of criteria, including morphological, physiological and antigenic properties, DNA hybridization studies and phage typing, these three species were recognized as closely related and were classified in the genus Bordetella (Gerlach et al*.,* 2001).

B. bronchiseptica had been known by many names earlier *viz*., *Bacillus bronchicanis, Alcaligenes bronchisepticus, Alcaligenes bronchicanis, Haemophilus bronchisepticus* and later on Moreno–Lopez named it as *B. bronchiseptica. B. bronchiseptica* in pigs causes atrophic rhinitis, snuffles in rabbits, suppurative bronchopneumonia in cats, suppurative necrotizing bronchopneumonia in guinea pigs, atrophic rhinitis in rats and respiratory infections in humans (Durgut et al., 2003).

B. bronchiseptica is a broad-range mammalian pathogen, which infects animals of a wide variety; pigs, horses, cattle, dogs, cats, rodents, and sometimes humans, both immunocompetent and immuno-compromised. In host organisms *B. bronchiseptica* causes both symptomatic and asymptomatic infections (Zlamy, 2016).

B. bronchiseptica causes respiratory infections in many different mammals including mice, rats, guinea pigs, skunks, rabbits, raccoons, cats, dogs, pigs, ferrets, hedgehogs, horses and occasionally humans. The best described and most important natural infections are in pigs and dogs. Bordetella is an important cause of respiratory disease in dogs and cats. The most prevalent and well-known sign of this infection in dogs is tracheobronchitis, which causes the self-limiting clinical condition known as kennel cough or canine infectious respiratory disease (CIRD) (Lavan and Knesl, 2015).

B. bronchiseptica is the sole pathogen identified in other infections and observations of natural disease and experimental infections suggest that it acts as a primary pathogen (Bemis et al*.,* 1977). In addition to tracheobronchitis, *B. bronchiseptica* can cause pneumonia: it has been identified in 12–78% of dogs with lower respiratory tract infections (Proulx et al*.,* 2014).

B. bronchiseptica is recognized as a zoonotic pathogen that can affect humans, particularly those who have immunosuppression or cystic fibrosis. Healthy dogs or cats or those with signs of respiratory disease, have been implicated as the source of *B. bronchiseptica* infection (Taha-Abdelaziz et al*.,* 2016). While multifactorial infections are common in CIRD, *B. bronchiseptica* has been shown to cause clinical respiratory disease on its own. Despite it's regular role as a commensal agent, *B. bronchiseptica* is often regarded as the most common and significant CIRD pathogen (Ford et al., 2009).

In dog shelters, boarding kennels, and veterinary clinics, where dogs are kept in groups, respiratory disease is typically the most significant issue. Bacteria are important in CIRD as primary pathogens and as a cause of secondary infections. *B. bronchiseptica* is the bacterium most frequently associated with CIRD (Erles and Brownie, 2008).

The high rate of asymptomatic infection with protracted organism shedding makes it challenging to analyse the epidemiology of *B. bronchiseptica* in research colonies, kennels, and pens used for laboratory animal experiments. The analysis of the epidemiology of *B. bronchiseptica* is significantly hampered by the high percentage of asymptomatic infection and prolonged shedding of the organism in lab animal research colonies, kennels, and pens (Pesavento and Murphy, 2014). Infectious tracheobronchitis (ITB) or kennel cough is the term used by veterinarians to describe an acute, highly contagious respiratory disease in dogs affecting the larynx, trachea, bronchi, and occasionally the nasal mucosa and the lower respiratory tract. Mild to severe episodes of cough and respiratory distress are characteristic clinical features recognized in affected dogs. ITB has worldwide distribution and is recognized as one of the most prevalent infectious diseases of dogs. The disease is frequently described in dogs housed in groups and boarding or training kennels (Buonavoglia and Martella, 2007). A significant clinical issue with dogs is respiratory infection, especially in high-density environments like pet stores, breeding and boarding kennels, shelters, research institutions, or veterinary clinics (Ford, 2006).

B. bronchiseptica is very contagious and spreads through aerosol contact. When an organism is inhaled, adhesion molecules (fimbrial adhesions, filamentous haemagglutinin,

pertactin, and lipopolysaccharides) help it stick to the respiratory cilia. Utilising virulence features like the outer capsule or the O antigen, which shields the bacteria from phagocytosis and complement-mediated attacks, the organisms can get around the host defences (Inatsuka et al., 2010). Other virulence factors have also been discussed, such as exotoxins that kill epithelial cells and type III secretion systems that enable bacterial colonisation, adenylate cyclase toxin with anti-inflammatory and immune evasion properties (Shrivastava and Miller, 2009). *B. bronchiseptica* is considered as the primary pathogen which infects a wide range of animal species including humans (Register and Kunkle, 2009).

2. PATHOGENESIS OF BORDETELLA BRONCHISEPTICA

The pathogenesis of *B. bronchiseptica* is dependent on the sequential coordinated synthesis of an array of virulence factors including adhesions, toxins, and other bacterial products that may alter host functions, facilitate immune evasion, or otherwise assist in transmission or survival. Expression of most virulence genes requires co expression of the BvgAS (Bordetella virulence genes) system (Beier and Gross, 2008).

BvgAS, a two-component sensory transduction system, controls the production of biofilms in Bordetella. The two-component BvgAS (Bordetella virulence gene) signal transduction system is used to monitor their environment and control the expression of their genes through at least three phases: a virulent Bvg+phase, a non-virulent Bvg-phase, and an intermediate Bvgi phase. The adhesins (FHA and FIM) and toxins (ACT), among other virulence factors, are encoded by genes expressed in the Bvg+phase. FHA and FIM continue to be expressed during the Bvgi phase (Irie et al., 2004).

Both the DNA-binding response-regulator protein BvgA and the sensor kinase protein BvgS are present in this locus. BvgAS regulates the expression of a spectrum of phenotypic phases that transitions between a virulent (Bvg+) phase and a non-virulent (Bvg2) phase in response to environmental signals. The BvgAS system is completely functional and many of the known virulence factors are expressed during the pathogenic Bvg+phase (Cotter and Jones, 2003). Contrarily, during the Bvg2 phase, when BvgAS is inactive, the highest expression of motility loci, virulence-repressed genes (vrg), and genes required for urease production occurs (McMillan et al., 1998).

Planktonic bacteria first attach to a surface, forming a monolayer. This is occasionally followed by the formation of clusters and microcolonies, and finally the development of differentiated structures in which both the individual bacteria and the entire community are encircled by an extracellular matrix. They have demonstrated that Bvg-mediated control of biofilm growth occurs after the initial attachment of the bacterial cells to a surface in the case of *B. bronchiseptica.* Additionally, we have demonstrated that the *B. bronchiseptica* Bps polysaccharide, which is not controlled by BvgAS, encourages biofilm formation at stages after attachment, particularly in the production of three-dimensional structures (Conover et al., 2012).

The variables and regulatory mechanisms that mediate later phases of biofilm development for *B. bronchiseptica* have therefore been the subject of some research, but nothing is known about the early stages of biofilm growth. The genes and proteins that biofilm cells express are different from those of planktonic cells, leading to different physiological states and phenotypes (Anderson and Otoole, 2008).

Unexpected revelation that the production of *B. bronchiseptica* biofilms is tightly regulated and begins with the early expression of genes encoding flagella, a hallmark Bvg2 phase trait. Flagella are important during the first stages of biofilm formation, and they have gathered strong data to support this. Their findings imply that the regulatory system controlling biofilm formation in *B. bronchiseptica* generates a classical Bvg2 phase phenotype under Bvg+phase circumstances (Nicholson et al., 2012).

Transcription of BvgAS and subsequent expression of the BvgAS inducible genes (Bvg+state) are triggered by rise in the temperature for growth and the organism transits from the external environment into the respiratory tract. At temperatures of about 77°F (25°C) or lower, BvgAS genes are not expressed, and the resulting Bvg state is not permissive for the synthesis of toxins, adhesins, and other known or suspected virulence proteins, whereas there is maximal expression of motility genes and virulence-repressed genes (Akerley et al., 1992).

The transcription of BvgAS and subsequent expression of the BvgAS-inducible genes (Bvg+state) are influenced by environmental conditions, such as an increase in growth temperature that happens as the organism transitions from an external environment into the tissues of the respiratory tract. Phenotypic modulation is a fully reversible process that is an essential part of an organism's adaptive response to environmental changes. Previous research using phase-locked and ectopic expression mutants showed that the Bvg+phase encourages *B. bronchiseptica* colonisation of the respiratory tract while the Bvg phase encourages survival in nutrient-deprived environments, such as those that may be present in an environmental reservoir (Cotter and Miller, 1994).

The expression of all BvgAS-activated genes is irreversibly abolished by a tiny percentage of growing cells spontaneously

acquiring deletions or frame shift mutations, regardless of the growth conditions, which also affects the BvgAS genes. Expression of "late" genes, such as a number of toxins, doesn't start until there are enough of the BvgAS gene products in the body. As *B. bronchiseptica* cycles through transmission, colonisation, growth and spread, immune evasion, and shedding, the presence of the BvgAS system suggests that precise control of the temporal expression of virulence factors in response to a changing environment is important for maximising growth and survival (Vergara-Irigaray et al., 2005).

The inflammatory response to *B. bronchiseptica* in experimental dogs was characterized by neutrophilic invasion of the ciliated respiratory mucosa. During the first weeks of experimental infection, numerous bacteria were seen in the cilial layer. It has been suggested that these bacteria are trapped among the cilia or in the layer of mucus above the cilia (Thompson et al., 1976).

The dermonecrotic toxins of *B. bronchiseptica* are of significant virulence factors. The dermo-necrotizing toxin is deadly when administered intraperitoneally to mice and causes skin necrosis when administered intradermally to guinea pigs. They are intracellular, heat-labile toxins (Gentry-Weeks et al., 1988). *B. bronchiseptica* attaches to the epithelial cells lining the nasal mucosa during the early stages of infection. Although attachment to nonciliated epithelia can also occur and may be crucial in the establishment of microcolonies or bio films, organisms exhibit a preference for ciliated cells when adhering to them (Brockmeier et al., 2019).

Fimbrial proteins, a collection of hair-like strands that protrude from the surface of bacteria, have a variety of binding specificities, one of which improves FHA-mediated adhesion. Fimbriae play a role in biofilm production, humoral and cell-mediated immunological responses to infection, and tracheal colonisation and persistence (Gerlach et al., 2007)

The expression of toxins helps the disease advance once *B. bronchiseptica* has taken hold in the respiratory tract. Dermonecrotic toxin (DNT), a protein toxin, inhibits bone growth, which is necessary for the development of pneumonia and turbinate atrophy in both mice and pigs. According to Brockmeier et al. (2002), the DNT is mostly to blame for the pneumonic lesions, which can be deadly, in suckling pigs and are characterized by necrosis, hemorrhage, neutrophil buildup, and ultimately fibrosis. Adenylate cyclase toxin (ACT), a toxin with adenylate cyclase and pore-forming properties, also promotes virulence by impairing innate immunoprotective mechanisms.

A peptidoglycan breakdown product called tracheal cytotoxin (TCT) results from typical bacterial cell wall remodeling during growth. Instead of recycling TCT like most other gram-negative bacteria do, *B. bronchiseptica* releases it extracellularly, where it combines synergistically with lipopolysaccharide to promote ciliostasis and the expulsion of ciliated cells from the mucosal epithelial layer. Early in the course of an infection, mucociliary clearance is impaired, which is probably caused by TCT. Age and immunological function are important (Datz, 2003). BvgAS is a two-component system that controls the virulent factors as a response to external stimuli. The regulatory system is characterized by phase variation and antigenic modulation (Mattoo and Cherry, 2005).

3. CLINICAL SIGNS OF BORDETELLA BRONCHISEPTICA

Clinical signs of infection with *B. bronchiseptica* develop within 3 to 4 days of exposure and, without complications, persist for up to 14 days. They include coughing, gagging or retching and mild serous oculo-nasal discharge. Affected dogs usually remain non-febrile, alert and active. The disease is self-limiting unless complicated by bronchopneumonia which may develop in unvaccinated pups or in older immunosuppressed animals (Ford, 2012).

Altered respiratory epithelial cell function leads to excessive mucus secretion once colonization has been established and impairment of the local innate immune defenses, predisposing the host to infection by opportunistic secondary pathogens. Clinical signs can vary dramatically. In more severe disease cases that involve the lower respiratory tract, signs of systemic illness can be present, including lethargy, decreased appetite, fever, and a productive cough (Siegel and Weiser, 2015).

Clinical signs associated with Bordetella usually relate to upper respiratory tract infection. *B. bronchiseptica* is implicated in a mild form of atrophic rhinitis in pigs and in canine Infectious tracheobronchitis (ITB) (Vieson et al., 2012). Bordetellosis comprises of two clinical forms. Uncomplicated form is associated with dry gagging, hacking cough and retching behaviour. The other form, the complicated form characterized by wet cough, is common in puppies or immunocompromised dogs. The disease is associated with mucoid discharges and signs of systemic infection including pyrexia, anorexia, chorioretinitis, vomiting and diarrhoea leading to death of the pup (Edinboro et al., 2004).

4. DIAGNOSIS OF *BORDETELLA BRONCHISEPTICA*

For diagnosis, *B. bronchiseptica* must first be isolated, and then the organism be can be identified using biochemical, serological, and molecular techniques (Abd

Alfatah, 2019). Microbiological findings in nasal or pharyngeal swabs from dogs should be used to confirm the precise diagnosis of *B. bronchiseptica* infection. History and clinical indicators alone can only suggest that we are dealing with infectious tracheobronchitis brought on by *B. bronchiseptica*. Cooperation with public health diagnostic laboratories is frequently required since they have more advanced tools and can carry out more accurate diagnostic processes (Milanov et al., 2018).

Blood agar, Bordet-Gengou agar, Smith-Baskerville culture media, and MacConkey agar are all excellent substrates for Bordetella species that grow quickly at 37°C. In terms of biochemical test, Bordetella are positive for the utilisation of oxidase, catalase, and citrate and negative for the creation of gelatinase, DNase, indole, and H2S as well as the fermentation of any sugar (Gonzalez et al., 2006).

After 24 hours of incubation, virulent strain colonies on sheep blood agar are tiny, convex, and smooth. Contrary to *B. avium*, which is non-haemolytic, many *B. bronchiseptica* isolates are haemolytic. The isolate produced catalase, urease, and oxidase as well as utilised citrate, but it did not ferment carbohydrates (glucose, sucrose, or arabinose). Light microscopy detected tiny Gram-negative coccobacilli after the slides were dyed (Milanov et al., 2018).

The diagnosis is made in light of a history of recent contact with carrier dogs and recognisable clinical symptoms. Serology and immunisation records may be helpful in identifying the presence of respiratory viruses. For radiographic analysis, to obtain the most information, both right and left lateral views of the chest are necessary and to improve the quality of the radiographic examination of the lung fields, expose the radiograph at maximum inspiration if possible (Elgalfy et al., 2022).

Lung patterns are merely the radiological manifestation of lung illness. Alveolar pattern, interstitial pattern, and bronchial pattern are examples of typical patterns. In cases of bronchitis and kennel cough, a bronchial pattern was seen, which was represented by a diffuse thickening of the airway lines and rings throughout the pulmonary tissue. In cases of pulmonary oedema and pneumonia, an unstructured interstitial pattern was seen that was characterised by an increase in the soft tissue opacity that only partially obscured the blood vessel boundary. In cases of fungal pneumonia, soft tissue nodules with ovoid or rounded shapes that are dispersed throughout the lung tissue reflect an organised interstitial pattern. Alveolar pattern was seen in the same cases as interstitial pattern, but it is more severe. It is characterised by an area of increased soft tissue opacity in the lung tissues that totally block pulmonary blood vessels (Thrall and Robertson, 2022).

In cases of pneumonia and aspiration pneumonia, radiographic images showed a mostly interstitial pattern and alveolar infiltration, with some cases also exhibiting lung consolidation. Megaoesophagus was a contributing factor in certain aspiration pneumonia cases. Some CIRD cases with radiographic views showed pneumonia mostly characterised by a bronchial pattern, whereas other instances displayed a mixed pattern. According to radiographic views of dogs with kennel cough, the pattern was primarily bronchial; however, some cases had mixed patterns (Vindenes et al., 2015).

The onset of therapy should be determined by the clinical signs. For example, in critically ill animals, empiric therapy should begin after swab collection, but in more stable animals, antimicrobial medication should be delayed until the antibiogram results, which take two to three days to come. Treatment should last for approximately two weeks, or seven days after health issues have been resolved (Leekha et al., 2011).

The primers Fla1 (5'CCCCCGCACATTTCCG AACTTC3'), Fla2 (5' AGGCTCCCAAGAGAGAAAG GCTT 3'), Fla3 (5' CACCTGCCCCATCTCC 3'), and Fla4 (5' TGGCGCCTGCCCTATC3') can be used to amplify the upstream region of the fla gene. For disease surveillance and bacterial dissemination management, the prompt identification of Bordetella infections is crucial. This goal cannot be satisfactorily achieved using traditional laboratory techniques (culture and serology). Although very particular, culture lacks sensitivity and takes three to seven days to finish. *B. bronchiseptica* isolates can occasionally be found using a *B. parapertussis* PCR that has been set up (Reizenstein et al., 1993).

Adenylate cyclase hemolysin gene-based PCR that can detect *Bordetella spp*. without distinguishing between species has also been created. The PCR based on the pt promoter allows the differentiation between *B. pertussis, B. parapertussis,* and *B. bronchiseptica* by an additional cleavage step with restriction enzymes. The assay becomes less sensitive and more complicated as a result of this additional step. The flagellin structural gene's upstream region, which comprises the sigma consensus site and potential ribosome-binding sites, is the target DNA region. This region seems to be a strong option for a *B. bronchiseptica*-specific PCR test because the flagellum is solely produced in *B. bronchiseptica* (Cotter et al., 1998).

B. bronchiseptica is the only species that can express flagellum under particular circumstances, much like the pertussis toxin, which is only expressed by one of the three species and not by the other two. Although *B. pertussis* and *B. parapertussis* have the flagellin structural gene, it has been demonstrated that they are not mobile, most likely as a result of changes in the gene's upstream region (Parkhill et al., 2003).

Two oligonucleotide primers were then developed based on the aforementioned sequences, Fla2 derived from the DNA sequence that is the same in *B. parapertussis* and *B. bronchiseptica*, and Fla4 specific to *B. bronchiseptica*. A 237-bp PCR result was first discovered using these Bb primers after *B. bronchiseptica* DNA was multiplied. Following DNA amplification from either a *B. parapertussis* or a *B. pertussis* isolate, no PCR products could be identified. The three *Bordetella* spp can thus be distinguished, and *B. bronchiseptica* can be specifically detected, using a proven PCR laboratory diagnostic approach. It was established through sequencing the upstream sequences of the three *Bordetella* spp. a genes that the putative ribosome binding sites and a sigma consensus site are distinct. And it explains why *B. parapertussis* and *B. pertussis* cannot express the fla structural gene despite having it. This made this particular DNA section appear like a solid choice for a PCR test setup (Hozbor et al., 1999).

The *B. bronchiseptica* must first be isolated, and then the organism must be identified using biochemical testing, serological tests, and molecular techniques. Swabs from the throat and nose can be used to collect samples, which can subsequently be processed for isolation (Denes et al., 2006).

For the detection of *B. bronchiseptica* by PCR in more dogs than isolation, PCR was more sensitive than the conventional approach as reported earlier in various investigations. Therefore, multiplex PCR may be suggested for quick diagnosis of suspected Bordetellosis cases (Bhardwaj et al., 2013).

4.1. Microbial culture

Small gram-negative rods with a coccobacillary appearance are *B. bronchiseptica*. These bacteria are typically recognised by their growth traits, biochemical responses, and special capacity to agglutinate red blood cells. Bronchiseptica generate pale, non-lactose-fermenting colonies on MacConkey agar. For the isolation and presumed identification of Bordetella, a selective indicator medium containing bromothymol blue as a pH indicator is used (Smith and Baskerville, 1979).

Blood agar, Bordet-Gengou agar, Smith-Baskerville culture media, and MacConkey agar are all excellent substrates for Bordetella spp that grow quickly at 37°C. In terms of biochemistry, Bordetella are positive for the utilisation of oxidase, catalase, and citrate and negative for the creation of gelatinase, DNase, indole, and H2S as well as the fermentation of any sugar (Denes et al., 2006).

After 24 hours of incubation, virulent strain colonies on sheep blood agar are tiny, convex, and smooth. Contrary to *B. avium*, which is non-haemolytic, many *B. bronchiseptica* isolates are haemolytic. The isolate produced catalase, urease, and oxidase as well as utilised citrate, but it did not ferment carbohydrates (glucose, sucrose, or arabinose). Light microscopy detected tiny Gram-negative coccobacilli after the slides were dyed (Milanov et al., 2018).

4.2. Radiographic and biochemical examination

The diagnosis is made in light of a history of recent contact with carrier dogs and recognizable clinical symptoms. Serology and immunization records may be helpful in identifying the presence of respiratory viruses. For radiographic analysis, to obtain the most information, both right and left lateral views of the chest are necessary and to improve the quality of the radiographic examination of the lung fields, expose the radiograph at maximum inspiration if possible (Elgalfy et al., 2022).

In cases of pneumonia and aspiration pneumonia, radiographic images showed a mostly interstitial pattern and alveolar infiltration, with some cases also exhibiting lung consolidation. According to radiographic views of dogs with kennel cough, the pattern was primarily bronchial; however, some cases had mixed patterns (Vindenes et al., 2015). The *B. bronchiseptica* must first be isolated, and then the organism must be identified using biochemical testing, serological tests, and molecular techniques. Swabs from the throat and nose can be used to collect samples, which can subsequently be processed for isolation (Denes et al., 2006).

5. TREATMENT AND MANAGEMENT

The onset of therapy should be determined by the clinical signs. For example, in critically ill animals, empiric therapy should begin after swab collection, but in more stable animals, antimicrobial medication should be delayed until the antibiogram results, which take two to three days to come. Treatment should last for approximately two weeks, or seven days after health issues have been resolved (Leekha et al., 2011).

A suitable caloric and fluid intake may be necessary to provide supportive care during the acute phase or extended severe phases occur. Antimicrobials should be administered to dogs that exhibit clinical symptoms that last longer than a week or any indications of bacterial pneumonia, such as pyrexia, lethargy, decreased appetite, or an alveolar pulmonary pattern on thoracic radiographs. *B. bronchiseptica* or opportunistic secondary pathogens is best guided by culture and susceptibility testing (Garcia-de-la-Fuente et al., 2015).

The most widely used antibiotics are clindamycin, azithromycin and amoxicillin. In the tracheobronchial mucosa and lung parenchyma, these antibiotics can be detected in effective concentrations. Gentamicin sulphate has been demonstrated to diminish the amount of *B. bronchiseptica* in the distal trachea and bronchi. It has been suggested to utilise bronchodilators and cough suppressants

to lessen ITB symptoms. Antitussive medications with codeine derivatives, such as hydrocodone or butorphanol, are used to prevent recurrent non-productive coughs. Bronchodilators like theophylline and aminophylline may be used to avoid bronchospasm and so work as efficient cough suppressants, even though ITB does not typically produce bronchial hyperactivity/spasm (Vieson et al., 2012).

Dogs with primary chronic (non-infectious) bronchitis are more likely to not respond to antibiotic therapy. It is indicated to provide additional supportive care, which should be customized to the patient's need (Lappin et al., 2017). Nebulized oxygen therapy and/or nutritional support are two examples of the additional supportive care that can be provided. By eliminating a neck lead and reducing barking triggers, care should be given to prevent further irritation to the trachea (Reagan and Sykes, 2020).

Vaccines against *B. bronchiseptica* are administered orally and intravenously to dogs in veterinary settings. It is advised to vaccinate dogs who are at least three weeks old. Local antibody responses elicited by these mucosal vaccinations are not affected by maternal antibodies. Although antibodies are also found in milk and may help to protect the intestinal mucosa, maternal antibodies are passed from mothers to their puppies *via* the colostrum (Decaro et al., 2004). In dogs, maternal antibodies can last up to 14 weeks (Day et al., 2016).

There are vaccines available for many prevalent CIRDC infections, including *B. bronchiseptica,* CAV-2, CDV, CPIV, CIV H3N8, and H3N2. With the exception of CDV, these vaccinations reduce the severity of clinical symptoms and the amount of pathogen shedding rather than generating sterilizing immunity. All dogs should receive the CDV vaccination, which is a fundamental shot. In dogs at risk of exposure, the remaining immunizations are advised (Schulz et al., 2014).

In situations involving group housing, precautionary measures should be taken, such as isolation periods for dogs before reentering the population, routine monitoring for the emergence of clinical signs with the group, and quarantine protocols for dogs displaying clinical signs linked to chronic infectious respiratory diseases in dogs. The population should not be overcrowded, and stress should also be avoided. Facilities should be available in the event of an epidemic, and there should be an infectious disease strategy in place to restrict exposure to other canines inside the facility, separating sick animals from the general population, and following the appropriate disinfection protocols (Reagan and Sykes, 2020).

6. CONCLUSION

Bordetella bronchiseptica is one of the pathogens associated with upper respiratory tract disease among dogs. The cases can be diagnosed with clinical symptoms, laboratory examination of the samples and PCR will be the gold standard. Antibiotic Sensitivity test can be opted for the selection of antibiotics against *B. bronchiseptica* infection. Cases can be treated with Doxycycline.

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