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FEATURES OF CLINICAL MANIFESTATIONS AND PATHOMORPHOLOGICAL CHARACTERISTICS OF BORDETELLOSIS IN DOGS

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Summary. The results of the studies showed that the clinical signs of *Bordetella* infection in dogs are pronounced and have characteristic manifestations. These signs typically last for three to four months and then disappear. The most pronounced symptom was coughing, ranging from a rare, mild, productive cough to a severe, dry cough accompanied by suffocation attacks and vomiting. We recorded the most pronounced damage and typical pathomorphological changes in the respiratory organs of all dead dogs. The mucous membranes of the trachea and bronchi were hyperemic, and their lumens contained foamy sputum. Signs of catarrhal or catarrhal-purulent inflammation, emphysema, and hyperemia were detected in the lungs. A heterogeneous, spotted, marbled pattern was observed on the surface of the lungs, especially in the dorsal regions, which is characteristic of this infection. According to our studies, the infection was found in animals of all age groups. However, it was more frequently recorded and proceeded particularly severely in puppies aged two to five weeks and six months. In most cases, bordetellosis resulted in recovery following adequate etiological and symptomatic therapy; however, complications and animal deaths were frequently reported. According to our observations, mortality was low in complicated cases, ranging from 7% to over 22%

Keywords: *Bordetella bronchiseptica*, tracheobronchitis, bronchoalveolar lavage, nasopharyngeal secretion

Introduction. Currently, bordetellosis is recognized as an independent nosological entity caused by the bacterium *Bordetella bronchiseptica* (Versalovic et al., 2011). This bacterium is a medium-sized, Gram-negative, ovoid rod that was first isolated by N. S. Ferry in 1910 from the respiratory tract of a dog in the early stages of the disease (Avramenko, 1999). This pathogen causes respiratory diseases in a wide range of mammals, including dogs, horses, rabbits, pigs, rodents, and cats (De la Torre et al., 2012; Gueirard et al., 1995). In dogs, it causes tracheobronchitis, also known as 'kennel cough.' In rabbits, it is the main cause of rhinitis. In cats, it causes pneumonia. In piglets, it causes atrophic rhinitis (Huebner et al., 2006; Khanna et al., 2005; El Khatib et al., 2015). Infection with this pathogen can lead to chronic, asymptomatic colonization of the upper respiratory tract in animals (Versalovic et al., 2011). In humans, disease caused by *B. bronchiseptica* is uncommon. More often, it is characterized by asymptomatic carriage of the bacterium on the mucous membrane of the respiratory tract (Gueirard et al., 1995). *B. bronchiseptica* is an opportunistic pathogen that can be detected in humans with other infections. Severe manifestations and relapses are more characteristic of immunocompromised individuals, such as premature infants, patients with acute leukemia, cystic fibrosis, and HIV infection, as well as individuals after bone marrow and lung transplantation. The onset of infection is often associated with contact with sick animals (Gueirard et al., 1995; Huebner et al., 2006; El Khatib et al., 2015; Spilker, Liwienski and LiPuma, 2008).

Data from animal studies indicate that the pathogen binds to cells of the ciliated respiratory tract epithelium due to the presence of pertactin, fimbriae, the tracheal colonization factor, and filamentous hemagglutinin. The dermonecrotic factor, adenylate cyclase hemolysin, and tracheal cytotoxin cause local epithelial damage and increased mucus production. Consequently, mucociliary clearance is impaired, resulting in the accumulation of viscous secretions. This reduces the patency of small bronchi and bronchioles, causing atelectasis, bronchopneumonia, and emphysema (Avramenko, 1999). The pathogen evades the host's protective mechanisms by inhibiting phagocytosis through adenylate cyclase toxin. This contributes to its prolonged persistence in epithelial cells of the respiratory tract and alveolar macrophages. Such intracellular parasitism prevents the pathogen from being rapidly eliminated from the body and leads to bacteriocarrier status and chronic and recurrent infection (Gueirard et al., 1995; MHU, 2005, 2007; Hadzevych, 2024).

Although bordetellosis in domestic animals is a pressing global issue, the disease has not been adequately studied in our country. There is a severe lack of data on the spread, detection, and elimination of the disease. Many questions remain unanswered. These questions are about its epizootic characteristics and epidemiological role. The primary reservoir of *B. bronchiseptica* remains unknown, and the intraspecies and interspecies transmission of the pathogen in nature is not well understood. Additionally, there is conflicting data on the seasonality of the disease and the role of rodents, wild birds, and synanthropic birds in its spread. There is no clear information on the pathogenicity of *B. bronchiseptica* for laboratory animals. In fact, the available information is contradictory. There is no objective data on the ability of the bordetellosis causative agent to kill white mice in bioassays. Furthermore, the

pathomorphology of this disease has only been described in a few reports in foreign specialized publications. In our opinion, the clinical presentation of the infection, pathogenesis, and pathomorphological characteristics require further study and broader coverage. Information about the characteristics of the clinical picture and pathomorphological changes in sick animals is the primary basis for making a preliminary diagnosis and deciding on further actions. These actions include implementing the necessary anti-epizootic measures, such as applying appropriate diagnostic, preventive, and therapeutic procedures. These procedures help prevent or locate the source of infection promptly.

This stage of the study **aimed** to investigate the clinical manifestations of bordetellosis in dogs of different ages and to determine the patterns of pathomorphological changes in acute and chronic forms of the disease. To achieve this goal, the following tasks were set: (1) study and analyze available literature on related issues; (2) determine the epidemiological situation of bordetellosis in kennels in Kharkiv Region; (3) investigate clinical features in dogs with bordetellosis; (4) perform pathological autopsies on carcasses of animals that died from a *Bordetella* infection; and (5) determine macroscopic characteristics of the disease.

Materials and methods. The following methods were used in the research: epizootiological, medical history collection, clinical, morphological, and biochemical studies of animal blood, pathological, bacteriological, serological, and virological studies, and statistical analysis. Epizootiological research methods included studying the patterns of occurrence, spread, and dynamics of infection, as well as developing preventive measures and means of combating the disease. These methods included observation, comparison, measurement, experimentation, analysis, and synthesis. The isolation and identification of *B. bronchiseptica* clinical isolates from dogs with or suspected of having bordetellosis were carried out in accordance with the methodological guidelines for the microbiological diagnosis of whooping cough and paracough (MHU, 2005). During an epizootiological survey of kennels in Kharkiv Region of Ukraine, biological material was collected from sick and deceased animals for laboratory testing. This material included parenchymal organs, nasal and conjunctival swabs, stabilized blood, blood serum, scrapings, milk, and nasopharyngeal secretions. Nasopharyngeal secretions were sampled for *Bordetella* isolation using a sterile, disposable probe swab from the mucous membranes of the tonsils and pharynx. During the epizootiological examination, special attention was paid to retrospective epizootiological analysis (Korniienko et al., 2008; Gerlach et al., 2001; Milanko, Kholodylo and Dushkyn, 1995).

In cases of severe tracheobronchitis in animals, bronchoscopy was performed, and tracheal and bronchoalveolar lavage samples were collected. The resulting fluid was examined bacteriologically to determine the sensitivity of the microflora to antibiotics. We determined and recorded antibiotic sensitivity using the disc diffusion method in accordance with the methodological guidelines 'Determination of the Sensitivity of Microorganisms to Antibacterial Drugs' (MHU, 2007).

To exclude other microorganisms and viruses associated with respiratory tract damage accompanied by coughing, appropriate bacteriological, mycological, virological, and serological methods, as well as PCR diagnostics, were performed. First, *B. bronchiseptica* was differentiated from canine adenovirus, canine parainfluenza virus, canine respiratory coronavirus, influenza A virus, carnivore plague virus, reovirus types II-III, herpesvirus, *Mycoplasma cynos*, β-hemolytic streptococci, and *Pasteurella multocida* (Versalovic et al., 2011; Gadzevich, 2024; Mylanko and Dushkin, 1996; Milanko, Gerilovich and Dushkin, 1995).

During pathological autopsies of dead animals, macroscopic changes in organs and tissues were recorded and described. During autopsy, pathological material was collected for further histological examination by means of incomplete evisceration.

Results and discussion. The results of the studies showed that the clinical signs of canine bordetellosis are pronounced and have characteristic manifestations. The signs are prolonged and disappear within three to four months. The manifestation of symptoms depends on the extent of respiratory system damage, the type of inflammation, the course of the disease, the aggressiveness of the pathogen, complications from other pathogens, and the dog's age. Sometimes, the disease manifested only as a cough, with or without phlegm. In other cases, severe, life-threatening pneumonia developed. The initial signs of infection are small drops of serous exudate forming on the dog's nasal wings (Fig. 1) and difficulty breathing through the nose, accompanied by snorting sounds. After three to seven days, the animals developed depression, sneezing, anorexia, and pyrexia. Profuse serous discharge was observed from the eyes and nose, which then became purulent due to secondary infection. Initially, respiratory sounds were usually unchanged; however, in some cases, bronchial breathing was heard. Later, crepitus and wheezing were recorded. At the onset of uncomplicated bordetellosis, we most often observed only coughing, which appeared three to four days after contact with the pathogen. For example, this occurred after a dog visited a veterinary clinic or an exhibition where it came into contact with other sick animals. The cough could be dry (nonproductive) or wet (productive), rough or soft, and frequent or infrequent. Sometimes, a coughing fit was accompanied by phlegm, which was usually white or yellowish in color. Frequent regurgitation of large amounts of phlegm sometimes provoked vomiting. It should be noted that animal owners often mistook profuse phlegm for vomiting when it was actually a productive cough. After a few days, the animals' health typically worsens due to secondary infections and complications.

The results of the studies showed that bordetellosis in kenneled dogs in Kharkiv Region occurred in acute, subacute, chronic, latent, and atypical forms. The acute form was characterized by pronounced depression, frequent labored breathing, intermittent fever, an increase in body temperature to 40–41°C, refusal to eat, conjunctivitis, and rhinitis (Fig. 2 and Fig. 3). The most pronounced symptom was coughing, ranging from mild, productive coughs to severe, dry coughs accompanied by suffocation and vomiting. It should be noted that coughing in dogs with *Bordetella* infection had distinguishing characteristics. Initially, coughing was mainly observed at night in the form of short coughing fits. During the day, no coughing was observed. After a short time (two to three days), the frequency and nature of the cough usually worsen significantly. The cough became dry, raucous, and prolonged. Attacks of suffocation occurred, which were complicated by vomiting. Puppies often develop abdominal hernias due to excessive straining while coughing. The cough could easily be triggered by slight irritation or touching the trachea. Profuse, bilateral mucopurulent discharge was observed from the nasal cavity. The animals lost significant weight within one to three weeks. They lost their appetite, eventually refusing to eat altogether. Their 'voice' became hoarse. During examination of the oral cavity, redness and hyperemia of the soft palate and tonsils were noted. The cervical and submandibular lymph nodes were enlarged. The animals became lethargic and reluctant to get up; they also moaned. According to the results of clinical and morphological blood tests, neutrophilic leukocytosis with a left shift was noted, and pneumonia was diagnosed based on focal or diffuse opacity detected by chest X-ray.

The acute form lasted one to two weeks — this was the first wave of the endemic disease. During autopsies of dead animals, macroscopic changes were recorded and described in various organs and tissues. These changes were found in dogs infected with bordetellosis. During the external examination of carcasses, it was noted that the skin was dry and inelastic and that the visible mucous membranes were pale and dry. All animals showed signs of exhaustion (Fig. 4). We established that the most pronounced damage and typical pathomorphological changes were recorded in the respiratory organs of all dead dogs. The mucous membranes of the trachea and bronchi were hyperemic, and their lumens contained foamy sputum. The lungs showed signs of catarrhal or catarrhal-purulent inflammation (Fig. 5), emphysema, and hyperemia (Fig. 6).

The inflammatory process primarily spreads along the blunt edges of the diaphragmatic lobes of the lungs. A heterogeneous, spotted, marbled pattern was observed on the surface of the lungs, particularly on their dorsal surfaces, which is characteristic of this infection (Versalovic et al., 2011). Dense foci of bluish-rusty inflammation were found in various configurations in the apical and cardiac lobes of the lungs. Lesions in the extremities of the lungs were most often recorded and had clear demarcation. During autopsy, small necrotic foci of brown color were found. Additionally, an increase in the size of the bronchial lymph nodes, edema of the mucous membranes of the trachea and bronchi, and accumulations of mucus and pus in their lumens were noted. The enlarged bronchial lymph nodes were pinkish-red in color, both externally and on the parenchyma section. They were juicy, which are morphological signs of hyperplasia and serous-hemorrhagic inflammation. Other non-specific but consistent morphological signs resulting from circulatory disorders and heart failure included an enlarged, flaccid heart with right ventricular dilatation (Fig. 7). The liver was enlarged with signs of passive venous hyperemia. The gallbladder was filled with dark green bile (Fig. 8). In the later stages of the disease, dystrophic processes were observed in the liver (Fig. 9). The kidneys were enlarged and bean-shaped with signs of hyperemia. The spleen was enlarged and lanceolate, with various types of hemorrhages (spotted and striped).

Foci of serous-catarrhal inflammation were detected in the gastrointestinal tract (Fig. 10).

Upon examining the abdominal organs, enlarged mesenteric lymph nodes often attracted attention first. Sometimes, vascular hyperemia and hemorrhages in the serous membrane of the intestine were also observed. The enlarged lymph nodes were pinkish-red on the outside and in the parenchyma section. They were juicy, which are morphological signs of hyperplasia and serous-hemorrhagic inflammation.

In subacute cases, the infection was complicated by secondary microflora; prolonged, convulsive coughing, suffocation, wheezing, mucous discharge from the nasal cavity, thirst, and cracked paw pads were observed. The course of the disease in dogs was typical for bordetellosis. The animals showed a decrease or loss of appetite, lethargy, mucopurulent discharge from the nasal passages and conjunctivitis, enlarged submandibular lymph nodes, increased body temperature to 40–41°C, catarrhal and, subsequently, catarrhal-purulent inflammation of the upper respiratory tract; shortness of breath, frequent sneezing, and a suffocating, painful cough. The cough intensified when standing or moving, changing from wet to dry and severe, accompanied by suffocation and vomiting. As pathogenic microflora accumulated, pulmonary emphysema and pneumonia developed (Fig. 11). The animals exhibited severe abdominal breathing accompanied by wheezing. When a large number of animals were involved, the infection was much more severe, resulting in generalized lung damage. The sick animals were stunted in growth and development, lost weight (Fig. 12), and died en masse. Auscultation of the chest during inhalation revealed distinct noises and wheezing. Starting at four weeks of age, the development of the bone base of the upper jaw in puppies was delayed, and their incisor bite was affected.

In kennels with a history of *Bordetella* infection, a second wave of infection was often observed among animals after weaning. This wave of endemic disease

developed into a chronic form. In chronic cases or when the disease was complicated by secondary microflora, the inflammatory process involved other organs and tissues. Pleurisy, pericarditis, and lung abscesses were detected in such cases, and the affected areas became grayish-yellow in color. The foci of inflammation were both localized and diffuse, and the pathological changes depended on the duration of the process. In the localized form, separate foci were found in the lungs. In the diffuse form, which lasted more than two months, lobar foci were detected that covered the anterior, cardiac, accessory, and sometimes diaphragmatic lobes.

Bordetellosis was most commonly observed in young, old, or chronically ill animals. According to our research, the infection was found in animals of all ages, but it was more frequently recorded, and was particularly severe, in puppies aged from two–five weeks to six months. We diagnosed the disease in dogs ranging in age from birth to two years and older; however, the percentage of dogs diagnosed with bordetellosis decreased with age. Typically, bordetellosis in adult dogs manifests only as sneezing and rare coughing, which could periodically intensify or subside. Our research showed that *Bordetella* infection sometimes caused abortions (Fig. 13) in pregnant dogs with bordetellosis or resulted in the birth of non-viable offspring. In kennels unaffected by the disease, animals were most often able to carry and give birth to puppies. However, we often recorded exacerbations of *Bordetella* infection and increased coughing in bitches during the first days after giving birth. During this time, the puppies also developed symptoms characteristic of bordetellosis (within 2–5 days). It should be emphasized that, when signs of the disease appeared in a bitch or at least one puppy in the litter, *Bordetella* infection was usually observed among all animals in the litter that had been in contact with each other within a short period of time (2–5 days).

The main sources of infection were sick animals and convalescents. One trigger of the disease was endothelium damage caused by toxins or infectious agents, such as a viral infection. This was followed by the accumulation of bacteria, fungi, mycoplasma, and other pathogens. Congenital respiratory system abnormalities, chronic bronchitis, bronchiectasis, and tracheal collapse also contributed to the onset of the disease. Bordetellosis in dogs most often occurred after they visited mass events, such as pet shops, kennels, breeding farms, and animal exhibitions.

Our research has shown that the following breeds of dogs are most susceptible to bordetellosis: Pinscher, Spaniel, Eastern European Shepherd, Boxer, and Doberman. The following breeds are less susceptible: Royal Poodle, Airedale Terrier, Laika, and Irish Setter. However, it is too early to conclude whether a dog's susceptibility to *Bordetella* infection depends on its breed. Additional and more detailed observations are needed.

It should also be noted that a significant proportion of infected animals were asymptomatic. Some animals that recovered from the disease remained carriers of the bacteria for a long time. Both categories posed an increased risk of infection to susceptible animals in contact with them because they were undetectable sources of infection. According to our research, the carrier state can last from four to five weeks to several months. However, according to the literature, the carrier state can last for several years.

In most cases, bordetellosis resulted in recovery following adequate etiological and symptomatic therapy. However, complications and animal deaths were frequently reported. Risk factors for developing complications included having an impaired immune system or chronic respiratory diseases, such as bronchitis, ciliary dyskinesia, and tracheal collapse. Complications of the infection included chronic rhinitis, sinusitis, pneumonia, conjunctivitis, keratitis, cough, exhaustion, dehydration, skin lesions, abortions, and gastrointestinal diseases presenting as vomiting and diarrhea. According to our observations, mortality was low, ranging from 7% to 22%, even in complicated cases.

According to the research, the bacterial agent is most actively released into the environment during the acute phase of the disease. Therefore, samples should be taken from sick animals at an early stage of the disease (but no later than three weeks). At a later stage, the pathogen's viability decreases sharply. The literature indicates that the pathogen can survive for up to 20 days or longer in both external and indoor environments. It has been established that *Bordetella* can survive for a prolonged period in stagnant water, suggesting the potential for interspecies transmission without direct contact.

The infection is transmitted from sick animals by air or contact. Indirect transmission of the pathogen through infected feed, water, rodents, and stray cats is also possible (Fig. 14).

Research has shown that a high concentration of dogs in a confined area (kennel, animal hotel, neighborhood, house, apartment) was one of the main factors contributing to the spread of the disease. The infection should be considered highly contagious, as infection often occurs after brief contact with its source, for example, when visiting a veterinary hospital.

Peaks in enzootic disease were observed in kennels in Kharkiv Region during periods of two to five weeks after birth and after weaning puppies from their mothers. Due to adverse effects from various stress factors (e.g., temperature, biology), repeated outbreaks of infection were often recorded, accompanied by mortality rates of 22% or higher. The severity of the disease was associated with pathogenic factors in *B. bronchiseptica*, such as adhesins (filamentous hemagglutinin, pertactin, and fimbriae), and toxins (dermanecrotic, adenylate cyclase-hemolysin, and lipopolysaccharide).

Due to a sharp decrease in overall immunobiological resistance and the presence of hidden carriers in the kennel, the disease can spontaneously onset at any time of the year. However, the infection was most prevalent during the winter-spring and autumn-winter periods.

Figure 1. Formation of small drops of serous exudate on the wings of a dog's nose.

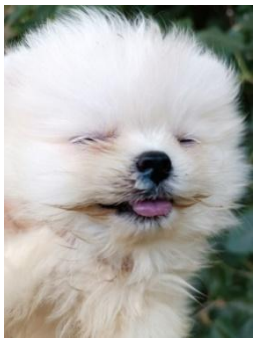

Figure 2. Mucopurulent exudate from the nasal canal and conjunctivitis in a puppy with bordetellosis.

Figure 3. Mucopurulent exudate from the nasal canal and conjunctivitis in a puppy with bordetellosis.

Figure 4. Signs of exhaustion in a dead animal with pale, dry, and dull visible mucous membranes and dry, inelastic skin.

Figure 5. Catarrhal-purulent pneumonia.

Figure 6. Emphysema and lung hyperemia.

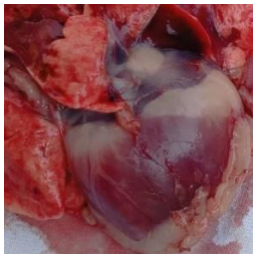

Figure 7. Flaccid, enlarged heart with right ventricular dilatation.

Figure 8. Venous hyperemia of the liver and an overfilled gallbladder.

Figure 9. Dystrophic processes in the liver.

Figure 10. Bloating and serous-catarrhal inflammation in the small intestine.

Figure 11. Catarrhal-purulent pneumonia.

Figure 12. Signs of exhaustion, depression, vomiting, and diarrhea in an animal sick with bordetellosis.

Figure 13. Aborted fetus from a pregnant dog with bordetellosis.

Figure 14. Signs of rhinitis and conjunctivitis in a homeless cat with bordetellosis.

In crowded animal husbandry conditions, bordetellosis most often co-occurs with mycoplasmosis, adenovirus, parainfluenza, and other infections. At the same time, the clinical presentation of the disease and the extent of pathological changes were more pronounced, and the percentage of young animals dying was significantly higher. In cases of associated bordetellosis in dogs, an avalanche-like spread of the infection among kennel animals was often observed. Animals with mixed infections were more severely ill, and etiological and symptomatic therapies were often ineffective.

Conclusions. 1. The results of the studies showed that the clinical manifestations of bordetellosis depended on the extent of respiratory system damage, type of inflammation, course of the disease, aggressiveness of the pathogen, complications from other pathogens, and age of the dog. The most pronounced sign of infection was coughing, ranging from mild, productive coughs to severe, dry coughs accompanied by suffocation and vomiting.

2. The most pronounced damage and typical pathomorphological changes in all dead dogs were recorded in the respiratory organs. The mucous membranes of the trachea and bronchi were hyperemic, and their lumens contained foamy sputum. The lungs showed signs of catarrhal or catarrhal-purulent inflammation, emphysema, and hyperemia. We observed a heterogeneous spotted marbled pattern on the surface of the lungs, especially in their dorsal parts, which is characteristic of this infection.

3. In most cases, bordetellosis resulted in recovery following adequate etiological and symptomatic therapy. However, complications and animal deaths were frequently reported. According to our observations, mortality was low, ranging from 7% to 22%, even in complicated cases.

Prospects for further research. The data we obtained indicate that, under crowded animal husbandry conditions, bordetellosis most often co-occurred with mycoplasmosis, adenovirus infection, parainfluenza, and other infections. These co-occurrences proceeded in accordance with the nature of the associated disease. At the same time, the clinical presentation of the disease and the extent of pathological changes were more pronounced, and the percentage of young animals that died was significantly higher. Animals with mixed infections were severely ill, and etiological and symptomatic therapies were often ineffective. According to the literature, a mixed course of many diseases, including respiratory diseases caused by two or more viral-bacterial agents, has been observed more often in recent years. The polyetiology of respiratory diseases in dogs, the insufficient study of their progression, imperfect diagnostic methods, and the lack of specific preventive measures mean that generally accepted health measures do not always yield positive results. These circumstances highlight the need for a more thorough examination of the epidemiological situation of respiratory diseases in dogs, their characteristics, and the development of treatment and prevention strategies. These issues require careful study and research. The results will serve as the foundation for developing a set of anti-epizootic measures, including appropriate diagnostic, preventive, and therapeutic procedures. These measures will help prevent or promptly identify the source of *Bordetella* infection.

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