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Canine leptospirosis: Clinical spectrum, diagnostic challenges, and zoonotic implications

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Abstract
Leptospirosis is a globally distributed zoonotic disease caused by pathogenic spirochetes of the genus *Leptospira*, affecting humans and a wide range of domestic and wild animals. Transmission occurs via direct contact with infected urine or indirectly through contaminated water or soil. The disease exhibits a broad clinical spectrum, from subclinical infections to severe multi-organ involvement, including hepatic, renal, and hemorrhagic complications. Diagnosis relies on serological assays such as the microscopic agglutination test (MAT) and ELISA, molecular methods like PCR, culture, and histopathological evaluation. Effective management includes prompt antimicrobial therapy, supportive care for organ dysfunction, and monitoring for complications. Preventive strategies emphasize vaccination, environmental sanitation, rodent control, and public health education. This review consolidates current understanding of leptospirosis, addressing epidemiology, pathogenesis, clinical features, diagnostic approaches, treatment modalities, prevention, and future research directions, providing a comprehensive resource for veterinarians, clinicians, and public health professionals.

Keywords: Leptospirosis, zoonosis, diagnosis, treatment, prevention, epidemiology

1. Introduction

Leptospirosis is an acute or chronic infectious disease of global significance that affects a wide range of mammalian species including dogs, livestock, wildlife, and humans (Levett, 2001) [23]. It is caused by pathogenic species of the genus *Leptospira*, which are spiral-shaped, motile bacteria belonging to the order *Spirochaetales* (Bharti *et al*., 2003) [8]. Among domestic animals, dogs are considered one of the most susceptible species and also play a critical role as reservoir hosts for certain leptospiral serovars, thus contributing to the epidemiological cycle of the disease (Faine *et al*., 1999) [14].

Canine leptospirosis is recognized as a re-emerging zoonotic disease worldwide, with increasing incidence in urban as well as rural canine populations (Goldstein, 2010) [15]. Environmental and ecological factors such as rainfall, flooding, poor sanitation, and rodent proliferation contribute significantly to the prevalence of leptospirosis (Ko *et al*., 2009) [20]. In India, canine leptospirosis has been reported from several states, particularly those with warm and humid climates, highlighting the endemic nature of the disease (Srivastava and Singh, 2013) [27]. The clinical importance of leptospirosis in dogs stem from its multisystemic involvement including renal, hepatic, pulmonary, and hematologic disorders, which can vary from mild subclinical infections to fulminant, life-threatening illness (Sykes *et al*., 2011) [28]. Canine leptospirosis is often underdiagnosed or misdiagnosed due to the nonspecific nature of clinical signs, overlapping with other systemic diseases such as infectious canine hepatitis, parvoviral enteritis, and immune-mediated disorders (Adler *et al*., 2010) [1, 4].

Historically, leptospirosis in dogs was primarily associated with *Leptospira interrogans* serovars Canicola and *Icterohaemorrhagiae* (Greene & Sykes, 2006) [17]. However, in recent decades, there has been an epidemiological shift with emerging serovars such as Grippotyphosa, Pomona, Bratislava, and Autumnalis being increasingly implicated in canine infections (Ward *et al*., 2002) [31]. This change poses a challenge for vaccination strategies, as immunity is largely serovar-specific (André-Fontaine, 2013) [6].

The pathogenesis of canine leptospirosis involves penetration of the bacteria through abraded skin or mucous membranes, dissemination via the bloodstream, and subsequent colonization of the renal tubular epithelium and other organs (Ellis, 2015) [13]. This results in a spectrum of disease presentations, including acute kidney injury, hepatic dysfunction, vasculitis, pulmonary hemorrhage syndrome, and coagulopathies (Schuller *et al*., 2015) [26]. Chronic carriers can intermittently shed organisms in urine, perpetuating the risk of environmental contamination and zoonotic transmission (Adler, 2014) [2].

Leptospirosis is an important zoonosis, with dogs serving as both sentinels and sources of human infection, particularly for veterinarians, kennel workers, and pet owners (WHO, 2003) [32]. Human leptospirosis ranges from mild febrile illness to severe forms like Weil's disease (characterized by jaundice, renal failure, and hemorrhage) and pulmonary haemorrhagic syndrome, conditions that are associated with high mortality rates (Haake *et al*., 2015) [18].

The diagnosis of canine leptospirosis remains challenging due to limitations in currently available tests. The microscopic agglutination test (MAT) continues to be regarded as the gold standard, but it requires paired sera, specialized laboratories, and interpretation challenges due to cross-reactivity (OIE, 2018) [25]. Polymerase chain reaction (PCR) assays have emerged as a rapid and sensitive alternative for early detection, while ELISA and other serological kits provide supportive evidence (Harkin *et al*., 2003) [19].

Therapeutic management of leptospirosis in dogs is aimed at eliminating the leptospiremic phase and reducing renal colonization, typically using antimicrobials such as penicillin derivatives and doxycycline (Sykes *et al*., 2011) [28]. Supportive care including intravenous fluids, antiemetics, hepatoprotectives, and dialysis in severe cases plays a critical role in patient survival (Kohn *et al*., 2010) [21-22].

Vaccination is the cornerstone of prevention in dogs, though limitations exist due to serovar specificity and short-lived immunity (André-Fontaine, 2006) [5]. Modern vaccines include bivalent and multivalent formulations that aim to cover the most clinically relevant serovars, but continued surveillance is required to monitor shifts in epidemiological trends (Schuller *et al*., 2015) [26].

Overall, canine leptospirosis remains a disease of veterinary and public health significance, necessitating a multidisciplinary approach encompassing clinical management, vaccination, environmental control, and zoonotic awareness (Adler, 2014) [2]. This review highlights the etiology, epidemiology, pathogenesis, clinical manifestations, diagnostic methods, therapeutic strategies, preventive measures, and public health implications of canine leptospirosis with emphasis on current trends and challenges (Levett, 2001) [23].

2. Etiology and Classification

Leptospirosis in dogs is caused by pathogenic bacteria belonging to the genus *Leptospira*, which are obligate aerobic, Gram-negative, tightly coiled spirochetes (Levett, 2001) [23]. The genus *Leptospira* was first described by Noguchi in 1917 [24] following the discovery of the etiological agent of Weil's disease in humans (Noguchi, 1917) [24]. These organisms are characterized by their unique helical morphology with hooked ends, periplasmic flagella, and rapid motility, which aid in their ability to penetrate mucosal surfaces and abraded skin (Bharti *et al*., 2003) [8].

The genus *Leptospira* is taxonomically classified into pathogenic, intermediate, and saprophytic groups based on genetic and phenotypic features (Adler, 2014) [2]. Pathogenic species are responsible for disease in animals and humans, whereas saprophytic species are free-living organisms commonly found in soil and water, posing no direct threat (Vincent *et al*., 2019) [30]. Intermediate species demonstrate variable pathogenicity and their role in disease remains partially understood (Cerqueira and Picardeau, 2009) [9].

More than 22 species of *Leptospira* have been recognized using molecular techniques, and these are further divided into over 300 serovars based on antigenic differences in the lipopolysaccharide (LPS) structure. In veterinary medicine, serovars are grouped into serogroups, which share common antigens detectable in serological assays such as the microscopic agglutination test (MAT) (Faine *et al*., 1999) [14].

In dogs, the most important pathogenic species are *Leptospira interrogans* and *Leptospira kirschneri*, which include serovars historically associated with canine infections such as Canicola and Icterohaemorrhagiae (Greene & Sykes, 2006) [17]. However, epidemiological surveys have documented emerging serovars such as Pomona, Grippotyphosa, Autumnalis, and Bratislava, reflecting regional variations in prevalence (Ward *et al*., 2002) [31]. These variations pose challenges to prevention, as immunity is primarily serovar-specific and vaccination against one serovar does not reliably confer protection against others (André-Fontaine, 2006) [5].

Dogs are considered the maintenance host for *L. interrogans* serovar canicola, which is adapted to persist in the canine renal tubules with minimal clinical disease, enabling chronic urinary shedding (Ellis, 2015) [13]. Conversely, dogs act as incidental hosts for other serovars such as Icterohaemorrhagiae, Grippotyphosa, or Pomona, which tend to cause acute and severe clinical disease (Adler *et al*., 2010) [1, 4]. This host-serovar interaction is critical in determining the epidemiology, severity, and outcome of canine leptospirosis (Levett, 2001) [23].

The survival of leptospires in the environment is facilitated by their ability to persist in moist, alkaline soil and stagnant water for weeks to months (Trueba *et al*., 2004) [29]. They are inactivated by desiccation, acidic conditions, and disinfectants such as sodium hypochlorite, but their resilience in aquatic environments contributes to the seasonal nature of outbreaks, especially during monsoon and flood conditions (Ko *et al*., 2009) [20].

On a molecular basis, leptospiral virulence is attributed to surface proteins, lipopolysaccharides, and hemolysins that enable adhesion, immune evasion, and tissue damage (Haake *et al*., 2015) [18]. The outer membrane proteins (OMPs) such as LipL32, LigA, and LigB play significant roles in adhesion to host tissues and modulation of host immune responses (Adler, 2014) [2]. The antigenic variability in these proteins underlies the diversity of serovars and complicates vaccine development.

Thus, the etiological complexity of leptospirosis in dogs lies in the diversity of pathogenic species, the regional distribution of serovars, and the host-pathogen interactions that influence disease expression, epidemiology, and control measures (Ellis, 2015) [13]. A thorough understanding of the classification and biology of *Leptospira* is essential for accurate diagnosis, vaccine formulation, and epidemiological surveillance in canine populations (Schuller *et al*., 2015) [26].

3. Epidemiology and Transmission

Leptospirosis is a globally distributed zoonotic disease, with significant impact on both animal and human populations

(Levett, 2001) [23]. The disease is considered endemic in tropical and subtropical regions, where high humidity, heavy rainfall, and warm temperatures facilitate environmental survival and transmission of the organism (Bharti *et al*., 2003) [8]. In contrast, temperate regions generally experience sporadic outbreaks, often associated with seasonal rainfall and flooding (Ko *et al*., 2009) [20].

Dogs play an important role in the epidemiology of leptospirosis as both reservoir hosts and incidental hosts, depending on the infecting serovar (Greene & Sykes, 2006) [17]. As reservoir hosts for *L. interrogans* serovar Canicola, dogs can harbor the organism in renal tubules and excrete it in urine for prolonged periods without manifesting overt disease (Ellis, 2015) [13]. As incidental hosts, dogs may acquire infection with serovars such as Icterohaemorrhagiae, Grippotyphosa, Pomona, or Autumnalis, which typically cause acute and severe clinical disease (Adler *et al*., 2010) [1, 4].

The transmission cycle of leptospirosis involves a wide range of reservoir hosts including rodents, livestock, and wildlife, which serve as sources of environmental contamination (Faine *et al*., 1999) [14]. Rodents, particularly rats, are recognized as the most important reservoirs due to their ability to chronically excrete large numbers of leptospires in urine without developing clinical illness (Ko *et al*., 2009) [20]. Infected animals shed organisms into the environment, where leptospires survive in moist soil and stagnant water for extended periods (Trueba *et al*., 2004) [29].

Dogs acquire infection primarily through direct contact with urine of infected animals or via indirect contact with contaminated water, soil, bedding, or food (Goldstein, 2010) [15]. Entry of leptospires occurs through abraded skin, mucous membranes of the mouth, nose, and conjunctiva, or less commonly via ingestion (Haake *et al*., 2015) [18]. Transmission is favoured in environments with poor sanitation, flooding, overcrowded kennels, and rodent infestation (Srivastava and Singh, 2013) [27].

Epidemiological studies have shown that leptospirosis in dogs has a seasonal distribution, with higher incidence reported during the rainy season or following flooding events, when contaminated water provides an ideal medium for organism survival (Sykes *et al*., 2011) [28]. In urban areas, close association between dogs, rodents, and humans increases the risk of zoonotic transmission, while in rural settings, exposure may be linked to livestock reservoirs such as cattle and pigs (Schuller *et al*., 2015) [26].

Globally, the prevalence of canine leptospirosis varies with region and serovar distribution. In North America and Europe, emerging serovars such as Grippotyphosa, Pomona, and Bratislava have replaced Canicola and Icterohaemorrhagiae as the predominant causes of canine infection (Ward *et al*., 2002) [31]. In Asian countries including India, both classical (Canicola, Icterohaemorrhagiae) and newer serovars are implicated, reflecting complex epidemiological dynamics (Srivastava and Singh, 2013) [27]. The variability in serovar distribution underscores the importance of regional surveillance and tailored vaccination strategies (André-Fontaine, 2013) [6].

Dogs are considered sentinel species for human leptospirosis due to their shared environment and similar exposure risks (Adler, 2014) [2]. Canine cases often precede or coincide with outbreaks in humans, highlighting their epidemiological significance (Levett, 2001) [23]. Zoonotic transmission from dogs to humans is particularly important for veterinarians, kennel workers, and pet owners who may be exposed to urine or contaminated fomites (WHO, 2003) [32].

Molecular epidemiological tools such as PCR and multilocus sequence typing (MLST) have been increasingly employed to study leptospiral diversity and track outbreaks. These studies have revealed regional differences in strain distribution and have enhanced understanding of transmission pathways between animals, environment, and humans (Cerqueira and Picardeau, 2009) [9].

Overall, the epidemiology of canine leptospirosis is influenced by climatic conditions, reservoir host diversity, canine lifestyle, urbanization, and vaccination practices (Schuller *et al*., 2015) [26]. Understanding transmission dynamics is essential for developing effective prevention and control strategies, not only for animal health but also for reducing zoonotic risks (Haake and Levett, 2015) [18].

4. Pathogenesis and immunopathology

The pathogenesis of canine leptospirosis begins when leptospires gain entry into the host through abraded skin, mucous membranes of the oral cavity, nasal passages, or conjunctiva following direct or indirect contact with contaminated urine or water (Adler *et al*., 2010) [1, 4]. Following penetration, leptospires rapidly disseminate via the bloodstream during the leptospiremic phase, leading to widespread tissue colonization (Bharti *et al*., 2003) [8]. This systemic spread occurs within 2-7 days post-exposure and is characterized by fever, lethargy, and bacteraemia (Levett, 2001) [23].

Leptospires possess periplasmic flagella and a spiral morphology, enabling them to move efficiently through viscous environments such as connective tissue and extracellular matrix (Haake and Levett, 2015) [18]. The presence of outer membrane proteins (OMPs), including LipL32, LigA, and LigB, facilitates adhesion to host cells, extracellular matrix proteins (fibronectin, collagen, laminin), and endothelial surfaces, aiding tissue invasion (Adler, 2014) [2].

The primary target organs in canine leptospirosis are the kidneys and liver, though lungs, eyes, spleen, and reproductive organs may also be affected (Greene & Sykes, 2006) [17]. In the kidneys, leptospires localize in the renal proximal tubular epithelial cells, where they multiply and induce tubulointerstitial nephritis (Ellis, 2015) [13]. Damage results from both direct cytotoxic effects of leptospiral toxins and the host immune response, leading to acute kidney injury (AKI) manifested as azotemia, polyuria, and oliguria (Sykes *et al*., 2011) [28].

Hepatic involvement results from leptospiral colonization of hepatocytes and Kupffer cells, causing hepatocellular necrosis and cholestasis (Schuller *et al*., 2015) [26]. The clinical consequence is jaundice, increased serum liver enzymes, and, in severe cases, hepatic failure (Adler, 2014) [2]. Unlike viral hepatitis, leptospiral hepatic damage often coexists with renal dysfunction, giving rise to the classic hepatorenal form of leptospirosis (Levett, 2001) [23].

Pulmonary involvement, particularly leptospiral pulmonary hemorrhage syndrome (LPHS), is increasingly recognized in canine and human infections (Dolhnikoff *et al*., 2007) [12]. The pathogenesis involves severe pulmonary capillary damage mediated by immune complex deposition and inflammatory cytokines, leading to alveolar hemorrhage, dyspnea, and hemoptysis (Haake *et al*., 2015) [18]. Mortality rates are high when pulmonary complications occur (Gouveia *et al*., 2008) [16].

The immune response to leptospiral infection is biphasic. Initially, innate immunity attempts to clear circulating leptospires through phagocytosis and complement activation, but leptospires have evolved mechanisms to evade these defenses by binding complement regulators and resisting oxidative stress (Cinco, 2010) [10, 11]. As adaptive immunity develops, antibody production; especially IgM in the early phase and IgG in later stages; aids in bacterial clearance (Adler, 2014) [2].

Despite immune clearance from blood and most tissues, leptospires persist in the renal tubular lumen, where they are protected from immune attack and antimicrobial activity due to their location in the apical membrane and urine environment (Faine *et al*., 1999) [14]. This carrier state results in chronic urinary shedding and environmental contamination (Ellis, 2015) [13].

Pathological lesions in canine leptospirosis include renal congestion, cortical petechiae, hepatic enlargement with necrosis, pulmonary edema and hemorrhage, splenomegaly, and serosal petechiation (Greene & Sykes, 2006) [17]. Histopathological changes are characterized by lymphoplasmacytic interstitial nephritis, tubular degeneration, hepatocellular vacuolation, necrosis, and alveolar hemorrhage (Schuller *et al*., 2015) [26].

Immunopathologically, disease severity is linked to the intensity of the host inflammatory response rather than just bacterial load (Haake *et al*., 2015) [18]. Cytokine dysregulation and immune-mediated vasculitis contribute to capillary leakage, hemorrhage, and multi-organ dysfunction (Cinco, 2010) [10, 11]. Thus, both direct leptospiral virulence factors and host immune reactions play pivotal roles in determining clinical outcomes (Adler *et al*., 2010) [1, 4].

Overall, the pathogenesis of canine leptospirosis reflects a complex interplay between bacterial invasion, immune evasion strategies, host inflammatory responses, and persistence in renal tissue, making it a challenging disease to diagnose, manage, and prevent (Ellis, 2015) [13].

5. Clinical Manifestations

Canine leptospirosis exhibits a wide spectrum of clinical presentations, ranging from subclinical infection to acute, life-threatening disease (Sykes *et al*., 2011) [28]. The variability in clinical signs depends on factors such as the infecting serovar, host immune status, age, concurrent disease, and environmental exposure (Levett, 2001) [23].

5.1 General Signs

Dogs with leptospirosis often present initially with nonspecific systemic signs, including fever, lethargy, anorexia, vomiting, and diarrhea (Greene & Sykes, 2006) [17]. Fever may be intermittent or persistent, and in some cases, hypothermia can be observed in severely ill animals (Goldstein, 2010) [15]. Muscle pain, stiffness, and reluctance to move are commonly reported due to leptospiral myositis and inflammatory cytokine effects (Adler *et al*., 2010) [1, 4].

5.2 Renal Signs

Renal involvement is the most common manifestation in canine leptospirosis (Ellis, 2015) [13]. Dogs may develop polyuria and polydipsia during early tubular damage, progressing to oliguria or anuria in acute kidney injury (Sykes *et al*., 2011) [28]. Azotemia, uraemia, and electrolyte imbalances are common laboratory findings (Schuller *et al*., 2015) [26]. Chronic infection may result in persistent renal dysfunction with proteinuria and isosthenuria (Greene & Sykes, 2006) [17].

5.3 Hepatic Signs

Hepatic involvement is more frequent in infections with serovars such as Icterohaemorrhagiae and Pomona (Levett, 2001) [23]. Clinical signs include icterus, pale mucous membranes, hepatomegaly, vomiting, and anorexia (Adler, 2014) [2]. Laboratory abnormalities may include elevated alanine aminotransferase (ALT), alkaline phosphatase (ALP), bilirubin, and coagulopathy (Sykes *et al*., 2011) [28]. Severe hepatic failure may occasionally occur, contributing to a poor prognosis (Schuller *et al*., 2015) [26].

5.4 Pulmonary Signs

Pulmonary involvement, including leptospiral pulmonary hemorrhage syndrome (LPHS), is increasingly recognized in canine cases (Dolhnikoff *et al*., 2007) [12]. Clinical signs include dyspnea, tachypnea, coughing, hemoptysis, cyanosis, and exercise intolerance (Haake *et al*., 2015) [18]. Pulmonary edema and hemorrhage can rapidly lead to respiratory distress and death (Gouveia *et al*., 2008) [16].

5.5 Hematologic and Vascular Signs

Leptospirosis often induces thrombocytopenia, leukocytosis, anemia, and coagulation abnormalities due to endothelial damage and immune-mediated mechanisms (Greene & Sykes, 2006) [17]. Petechiae, ecchymoses, and mucosal bleeding may be observed in severe cases, reflecting vasculitis and hemorrhagic tendencies (Adler *et al*., 2010) [1, 4].

5.6 Ocular Signs

Ocular manifestations may occur due to uveitis, conjunctivitis, and retinal hemorrhages (Sykes *et al*., 2011) [28]. Anterior uveitis may cause photophobia, pain, lacrimation, and impaired vision. Chronic ocular involvement can persist even after renal and hepatic recovery (Levett, 2001) [23].

5.7 Reproductive and Other Signs

Leptospirosis can occasionally cause abortions, stillbirths, and infertility in dogs, particularly if infection occurs during pregnancy (Faine *et al*., 1999) [14]. Less common signs include neurological manifestations such as meningitis, seizures, and ataxia due to leptospiral invasion of the CNS (Adler, 2014) [2].

5.8 Subclinical and Chronic Infection

Subclinical infection is frequently observed, especially in maintenance hosts infected with serovar Canicola (Ellis, 2015) [13]. Such dogs may shed leptospires in urine for weeks to months without showing overt clinical signs, serving as silent reservoirs for environmental contamination and zoonotic transmission (Sykes *et al*., 2011) [28].

Overall, the clinical spectrum of canine leptospirosis is broad and multisystemic, which complicates diagnosis and may delay appropriate therapy (Schuller *et al*., 2015) [26]. Awareness of the range of manifestations, including renal, hepatic, pulmonary, hematologic, ocular, reproductive, and subclinical forms, is essential for early recognition and effective management (Levett, 2001) [23].

6. Hematobiochemical alterations

Canine leptospirosis frequently produces distinct hematological and biochemical abnormalities that reflect the severity and organ involvement of the disease (Greene & Sykes, 2006) [17]. Recognition of these changes is crucial for supporting clinical suspicion, monitoring disease progression, and guiding therapy (Sykes *et al*., 2011) [28].

6.1 Hematologic Changes
The most common hematologic abnormalities in dogs with leptospirosis include leukocytosis or leukopenia, thrombocytopenia, and anemia (Levett, 2001) [23]. Leukocytosis typically reflects the host inflammatory response, while leukopenia may occur in severe acute infections due to bone marrow suppression or sequestration (Adler *et al*., 2010) [1, 4].

Thrombocytopenia is frequently observed, resulting from immune-mediated platelet destruction, consumption due to vasculitis, and disseminated intravascular coagulation (DIC) (Haake *et al*., 2015) [18]. Dogs with severe thrombocytopenia may exhibit petechiae, ecchymoses, and mucosal bleeding (Greene & Sykes, 2006) [17].

Anemia in leptospirosis is usually non-regenerative, although hemolytic anemia can occur occasionally due to haemolysin-mediated red blood cell destruction (Ellis, 2015) [13]. Normocytic, normochromic anemia is the most common type reported in canine cases (Schuller *et al*., 2015) [26].

6.2 Renal Biomarkers
Renal involvement in leptospirosis manifests as azotemia, increased creatinine, and blood urea nitrogen (BUN) (Sykes *et al*., 2011) [28]. The pattern may be prerenal, renal, or mixed, depending on hydration status and the extent of tubular necrosis (Levett, 2001) [23]. Urinalysis often reveals isosthenuria, proteinuria, glucosuria, and hematuria, indicative of tubular dysfunction (Ellis, 2015) [13].

Electrolyte disturbances such as hyponatremia, hypokalemia, and metabolic acidosis are frequently observed, resulting from impaired tubular reabsorption and renal failure (Greene & Sykes, 2006) [17]. Severe electrolyte imbalances can precipitate cardiac arrhythmias, neuromuscular weakness, and contribute to morbidity (Adler, 2014) [2].

6.3 Hepatic Biomarkers
Hepatic involvement is reflected in elevated serum liver enzymes, particularly alanine aminotransferase (ALT), aspartate aminotransferase (AST), and alkaline phosphatase (ALP) (Sykes *et al*., 2011) [28]. Hyperbilirubinemia, predominantly conjugated, may cause clinical jaundice and indicates hepatocellular and cholestatic injury (Schuller *et al*., 2015) [26]. Coagulation abnormalities may also arise due to impaired hepatic synthesis of clotting factors (Haake *et al*., 2015) [18].

6.4 Inflammatory and Other Biochemical Changes
C-reactive protein (CRP) and other acute phase proteins are often elevated, reflecting systemic inflammation and endothelial damage (Ellis, 2015) [13]. Hypoalbuminemia can occur due to renal loss, hepatic dysfunction, or inflammation, and is associated with increased severity and poor prognosis (Levett, 2001) [23].

6.5 Diagnostic Relevance
The combination of azotemia, liver enzyme elevation, thrombocytopenia, and abnormal urinalysis strongly supports a clinical diagnosis of leptospirosis in endemic regions (Sykes *et al*., 2011) [28]. However, these alterations are not pathognomonic and may overlap with other infectious, toxic, or immune-mediated diseases (Greene & Sykes, 2006) [17]. Thus, hematobiochemical profiles are used in conjunction with serology, PCR, and clinical findings to confirm the diagnosis (Schuller *et al*., 2015) [26].

In summary, canine leptospirosis is associated with multisystemic hematobiochemical disturbances, with renal and hepatic dysfunction predominating, accompanied by inflammatory and hematologic changes (Adler *et al*., 2010) [1, 4]. Early detection of these abnormalities is essential for initiating timely therapy and improving clinical outcomes (Haake *et al*., 2015) [18].

7. Diagnosis
The diagnosis of canine leptospirosis is challenging due to the nonspecific nature of clinical signs and the wide range of differential diagnoses, including infectious, immune-mediated, and toxic diseases (Sykes *et al*., 2011) [28]. Accurate diagnosis relies on a combination of clinical evaluation, laboratory testing, serology, molecular assays, and sometimes imaging studies (Levett, 2001) [23].

7.1 Clinical Evaluation
A thorough history and physical examination are critical first steps (Greene & Sykes, 2006) [17]. Risk factors such as exposure to stagnant water, flooding, rodents, livestock, or infected dogs, as well as seasonal trends, should be carefully evaluated (Ko *et al*., 2009) [20]. Clinical suspicion increases when dogs present with renal, hepatic, pulmonary, or hematologic abnormalities, especially if multiple systems are involved simultaneously (Schuller *et al*., 2015) [26].

7.2 Haematology and Biochemistry
Hematologic and biochemical profiles provide supportive evidence (Sykes *et al*., 2011) [28]. Common findings include thrombocytopenia, leukocytosis or leukopenia, non-regenerative anemia, azotemia, hyperbilirubinemia, elevated liver enzymes, electrolyte imbalances, and proteinuria (Greene & Sykes, 2006; Ellis, 2015) [17, 13]. While suggestive, these changes are not specific for leptospirosis and should be interpreted in the context of serologic and molecular tests (Levett, 2001) [23].

7.3 Serology
Microscopic Agglutination Test (MAT) remains the gold standard for serological diagnosis (Faine *et al*., 1999) [14]. MAT detects antibodies against specific leptospiral serovars and requires paired serum samples collected 1-2 weeks apart to demonstrate a fourfold rise in titer (Levett, 2001) [23]. A single high titer may indicate exposure but does not confirm active infection (Adler, 2014) [2].

Other serological methods include ELISA for detection of IgM or IgG antibodies, which provide rapid, sensitive, and cost-effective screening (Harkin *et al*., 2003) [19]. ELISA is particularly useful in the early leptospiremic phase, when MAT may be negative due to the delayed antibody response (Sykes *et al*., 2011) [28].

7.4 Molecular Diagnosis
Polymerase Chain Reaction (PCR) is a highly sensitive method for detecting leptospiral DNA in blood, urine, or tissue samples (Haake and Levett, 2015) [18]. PCR can identify infection before seroconversion, allowing early initiation of therapy (Cerqueira and Picardeau, 2009) [9]. It is especially useful in acute-phase disease and in monitoring urinary shedding in carrier animals (Ellis, 2015) [13].

7.5 Culture
Leptospira can be cultured from blood, urine, or tissue, but this is rarely performed due to slow growth, contamination risk, and specialized laboratory requirements (Levett, 2001) [23]. Culture is primarily used for epidemiological studies and serovar identification, rather than routine diagnosis (Faine *et al*., 1999) [14].

7.6 Imaging Studies
Imaging modalities such as ultrasound and radiography can assist in identifying organ involvement (Greene & Sykes, 2006) [17]. Renal ultrasonography may show enlarged, hyperechoic kidneys with loss of corticomedullary distinction, while hepatic imaging may reveal hepatomegaly or parenchymal changes (Schuller *et al*., 2015) [26]. Pulmonary radiographs can detect edema, hemorrhage, or infiltrates, particularly in severe LPHS cases (Dolhnikoff *et al*., 2007) [12].

7.7 Interpretation and Diagnosis
Diagnosis is most reliable when clinical findings, hematobiochemical abnormalities, serology, and molecular tests are considered together (Levett, 2001) [23]. Rapid presumptive diagnosis may be made in endemic regions based on clinical suspicion and supportive lab findings, but confirmation requires seroconversion or PCR positivity (Sykes *et al*., 2011) [28]. Early diagnosis is critical to reduce morbidity, prevent renal and hepatic failure, and limit zoonotic transmission (Adler, 2014) [2].

In summary, an integrated diagnostic approach that combines risk assessment, clinical evaluation, laboratory testing, serology, PCR, and imaging is essential for accurate detection and management of canine leptospirosis (Haake *et al*., 2015) [18].

8. Therapeutic Approaches
The management of canine leptospirosis involves a combination of antimicrobial therapy, supportive care, and treatment of organ-specific complications (Sykes *et al*., 2011) [28]. Early recognition and prompt initiation of therapy are crucial for reducing morbidity and preventing long-term sequelae such as chronic kidney disease (Levett, 2001) [23].

8.1 Antimicrobial Therapy
Antimicrobials are used to eliminate leptospires from the bloodstream and renal tubules. Penicillin and its derivatives, such as ampicillin or amoxicillin, are recommended during the leptospiremic phase to reduce bacteraemia and prevent dissemination (Greene & Sykes, 2006) [17].

Following the acute phase, doxycycline is administered to eradicate leptospires from renal tubules and prevent urinary shedding, typically at 5 mg/kg PO twice daily for 14 days (Ellis, 2015) [13]. In cases where doxycycline is contraindicated, alternative antibiotics such as azithromycin or chloramphenicol may be considered, although evidence is limited (Sykes *et al*., 2011) [28].

8.2 Supportive Care
Supportive therapy is a cornerstone of management and includes intravenous fluid therapy to correct dehydration, maintain renal perfusion, and manage electrolyte imbalances (Schuller *et al*., 2015) [26].

Anti-emetics, gastroprotectants, and nutritional support are provided to address gastrointestinal manifestations such as vomiting and anorexia (Greene & Sykes, 2006) [17]. Hepatoprotective agents may be administered in dogs with hepatic involvement, although their efficacy is primarily supportive (Levett, 2001) [23].

Oxygen therapy is indicated for dogs with pulmonary involvement, particularly in cases of leptospiral pulmonary hemorrhage syndrome (LPHS) (Haake *et al*., 2015) [18]. Mechanical ventilation may be required in severe respiratory compromise (Gouveia *et al*., 2008) [16].

8.3 Renal Support and Dialysis
Severe acute kidney injury may necessitate advanced interventions such as intermittent haemodialysis or continuous renal replacement therapy (CRRT) to manage uraemia and fluid overload (Sykes *et al*., 2011) [28]. Monitoring of renal function through serial measurement of BUN, creatinine, and electrolytes is essential to guide therapy (Ellis, 2015) [13].

8.4 Management of Coagulopathies
Dogs with significant thrombocytopenia or DIC may require blood product transfusions, including fresh frozen plasma or platelet-rich plasma, to manage hemorrhagic complications (Haake *et al*., 2015) [18]. Close monitoring of coagulation profiles and haematocrit is recommended to guide interventions (Adler, 2014) [2].

8.5 Monitoring and Follow-Up
Follow-up evaluation is crucial to ensure resolution of infection, normalization of organ function, and cessation of urinary leptospire shedding (Levett, 2001) [23]. Serological testing and PCR may be repeated to confirm therapeutic success, particularly in dogs that will continue to have contact with other animals or humans (Sykes *et al*., 2011) [28].

Overall, effective management of canine leptospirosis requires a multimodal approach, integrating antimicrobial therapy with supportive care, organ-specific interventions, and diligent follow-up to optimize clinical outcomes and prevent zoonotic transmission (Greene & Sykes, 2006; Ellis, 2015) [17, 13].

9. Prevention and control strategies
Prevention of canine leptospirosis is essential due to its zoonotic potential, environmental persistence, and risk of severe multi-organ disease in dogs (Levett, 2001) [23]. Effective control relies on vaccination, environmental management, rodent control, and public awareness (Adler, 2014) [2].

9.1 Vaccination
Vaccination is the cornerstone of prevention in dogs, targeting the most clinically relevant serovars (André-Fontaine, 2013) [5]. Bivalent vaccines (Canicola and Icterohaemorrhagiae) and multivalent vaccines (including Pomona, Grippotyphosa, Bratislava, and Autumnalis) are commercially available (Greene & Sykes, 2006) [17]. Vaccination induces serovar-specific immunity, reducing the severity of clinical disease and limiting leptospiral shedding (Sykes *et al*., 2011) [28].

Puppies typically receive a two-dose primary series at 8-12 weeks and 11-16 weeks of age, followed by annual boosters, though some regions recommend semi-annual vaccination based on exposure risk (Schuller *et al*., 2015) [26]. Despite vaccination, breakthrough infections can occur due to emerging serovars or incomplete immunity, underscoring the need for continued epidemiological surveillance (Levett, 2001) [23].

9.2 Environmental Management
Environmental measures are critical in reducing exposure to leptospires (Ko *et al*., 2009) [20]. Dogs should be prevented from accessing stagnant water, flood-prone areas, or soil contaminated with urine from rodents or livestock (Goldstein, 2010) [15]. Routine cleaning and disinfection of kennels, yards, and water bowls using effective agents such as sodium hypochlorite or quaternary ammonium compounds reduces environmental contamination (Faine *et al*., 1999) [14].

9.3 Rodent and Wildlife Control
Rodents are major reservoirs of leptospires and contribute to urban and peri-urban transmission (Adler and Peña Moctezuma, 2010) [1, 4]. Implementation of rodent control programs, safe storage of food, and management of livestock or wildlife interactions are recommended strategies to reduce the risk of infection (Haake *et al*., 2015) [18].

9.4 Public Health Precautions
Canine leptospirosis is a zoonotic disease, posing risks to veterinarians, kennel workers, pet owners, and laboratory personnel (WHO, 2003) [32]. Preventive measures include the use of protective gloves when handling urine or contaminated materials, proper hand hygiene, and prompt disinfection of contaminated surfaces (Adler, 2014) [2]. Owners should be educated on avoiding exposure of their pets to high-risk environments (Levett, 2001) [23].

9.5 Surveillance and Reporting
Active epidemiological surveillance is essential to identify emerging serovars, monitor vaccination efficacy, and detect regional outbreaks (Schuller *et al*., 2015) [26]. Reporting cases to local veterinary and public health authorities supports timely intervention, education, and risk reduction strategies (André-Fontaine, 2013) [5].

In summary, prevention of canine leptospirosis requires a comprehensive approach integrating vaccination, environmental hygiene, rodent control, owner education, and surveillance, with attention to regional epidemiological patterns to effectively reduce incidence and minimize zoonotic transmission (Levett, 2001; Haake *et al*., 2015) [23].

10. Public health significance and zoonotic implications
Canine leptospirosis is of significant public health concern due to its zoonotic potential and widespread environmental persistence (Levett, 2001) [23]. Dogs can act as both sentinel animals and sources of human infection, particularly in urban and peri-urban settings where close human-dog interactions occur (Adler, 2014) [2].

10.1 Zoonotic Transmission
Humans are typically infected through direct contact with the urine of infected dogs or indirectly via contaminated water, soil, or fomites (WHO, 2003) [32]. Entry occurs through abrasions, mucous membranes, or ingestion, similar to canine infection (Haake *et al*., 2015) [18]. Certain occupational groups such as veterinarians, kennel workers, farmers, and laboratory personnel are at increased risk (Adler *et al*., 2010) [1, 4].

10.2 Human Disease
In humans, leptospirosis ranges from subclinical or mild febrile illness to severe systemic disease such as Weil's disease, characterized by jaundice, renal failure, hemorrhage, and multi-organ dysfunction (Bharti *et al*., 2003) [8]. Pulmonary hemorrhage and acute respiratory distress are increasingly recognized complications with high mortality (Gouveia *et al*., 2008) [16]. Early diagnosis and treatment are crucial to reduce morbidity and mortality (Levett, 2001) [23].

10.3 One Health Perspective
The One Health approach emphasizes the interconnectedness of human, animal, and environmental health in controlling leptospirosis (Haake *et al*., 2015) [18]. Surveillance in dogs serves as an early warning system for potential human outbreaks (Sykes *et al*., 2011) [28]. Integrated strategies including canine vaccination, rodent control, environmental sanitation, and public education are essential to minimize risk to humans (André-Fontaine, 2013) [5].

10.4 Prevention in Humans
Preventive measures for humans include avoiding contact with potentially contaminated water, using protective clothing, practicing good hand hygiene, and educating at-risk populations (WHO, 2003) [32]. Rapid recognition and isolation of infected dogs, along with adherence to vaccination schedules, reduce the likelihood of zoonotic transmission (Adler, 2014) [2].

10.5 Epidemiological Importance
Dogs act as amplifying hosts for certain serovars and contribute to environmental contamination through chronic urinary shedding (Ellis, 2015) [13]. Mapping canine leptospirosis prevalence can assist in targeted public health interventions, outbreak preparedness, and vaccination campaigns in both humans and animals (Levett, 2001) [23].

In summary, canine leptospirosis has major zoonotic implications and underscores the importance of a One Health strategy involving veterinarians, physicians, public health authorities, and dog owners to prevent transmission, protect human health, and control disease in canine populations (Haake *et al*., 2015; Adler, 2014) [18, 2].

11. Conclusion
Canine leptospirosis is a re-emerging, multisystemic, and zoonotic disease of global veterinary and public health significance (Levett, 2001) [23]. Infection is caused by pathogenic species of *Leptospira*, which are highly adaptable and capable of surviving in diverse environmental conditions, leading to widespread exposure risk for dogs and humans (Bharti *et al*., 2003; Faine *et al*., 1999) [8, 14].

The disease exhibits variable clinical presentations, ranging from subclinical infection to acute kidney and liver failure, pulmonary hemorrhage, coagulopathies, ocular involvement, and reproductive complications (Greene & Sykes, 2006; Sykes *et al*., 2011) [17, 28]. Subclinical carriers serve as reservoirs, perpetuating environmental contamination and posing significant zoonotic risks (Ellis, 2015) [13].

Diagnosis requires a multimodal approach, integrating clinical evaluation, hematobiochemical findings, serology, PCR, and, where necessary, imaging studies (Levett, 2001; Haake *et al*., 2015) [23, 18]. Early detection is critical to reduce morbidity, prevent permanent organ damage, and limit urinary shedding that contributes to human infection (Adler, 2014) [2].

Therapeutic management emphasizes timely antimicrobial therapy, primarily with penicillin derivatives and doxycycline, alongside supportive care, renal and hepatic management, and treatment of coagulopathies (Greene & Sykes, 2006; Ellis, 2015) [17, 13]. Monitoring response to therapy and urinary shedding is essential for ensuring successful resolution and reducing the risk of zoonotic transmission (Schuller *et al*., 2015) [26].

Prevention strategies include vaccination, environmental management, rodent control, and owner education, with emphasis on regional serovar prevalence and risk assessment (André-Fontaine, 2013) [5]. Dogs act as important sentinels for human leptospirosis, underscoring the need for a One Health approach involving collaboration between veterinary, medical, and public health sectors (Haake *et al*., 2015; Sykes *et al*., 2011) [18, 28].

In conclusion, canine leptospirosis is a complex, multifactorial disease requiring integrated strategies for diagnosis, treatment, and prevention. Continuous surveillance, updated vaccination programs, and public health awareness are essential to mitigate disease impact in dogs and reduce the associated zoonotic threat (Levett, 2001; Adler, 2014) [23, 2].

Conflict of Interest
Not available

Financial Support
Not available

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