Clinical update: vaccinating against canine leptospirosis

Despite the ubiquitous nature of leptospires, their zoonotic potential, and widespread vaccination in dogs and cattle, leptospirosis can be considered an emerging disease. We have incomplete knowledge of the epidemiology of leptospirosis. There is a changing pattern in serovar prevalence driven by vaccination, geography and the distribution of reservoir hosts, and reports of clinical disease in vaccinated individuals. This review examines our current knowledge of this disease, its epidemiology, what vaccinations are currently available and the science behind which preparation we should choose.

Key words: Leptospire, leptospirosis, leptospira, vaccination, epidemiology, infectious disease, veterinary, zoonosis, canine, dog

Introduction

Over the last few years, quadrivalent leptospirosis vaccines (L4) have become available in Europe and the UK. Veterinary practices have had to decide whether to continue using the existing bivalent vaccine (L2) or change to the newer vaccines. Change can be challenging, especially when few cases may be seen in daily practice, coupled with an increasing public awareness of unnecessary vaccination and frequency of vaccination. Clients also worry about potential links between vaccination and immunemediated disease, and also about reported side-effects of vaccination, particularly of the increased risk of anaphylaxis in small breed dogs. Change involves increased compliance on behalf of the owner and patient to swap to a new regime and veterinarians have to change to new practice after feeling confident with the existing vaccination schedule over many years. This paper looks at what we know about canine leptospirosis, what vaccinations are available and what the drive is behind the development of quadrivalent leptospirosis vaccines.

What is canine leptospirosis?

Leptospires are gram negative, coiled, motile bacteria that are ubiquitous pathogens and are able to infect a wide variety of mammals. Whilst cats may be infected, they rarely show overt clinical signs, however dogs can become critically ill and die from infection. Leptospirosis is a zoonotic disease.

Leptospires were originally divided into pathogenic (Leptospira interrogans sensu lato) and non-pathogenic (L. biflexa sensu lato) species by phenotype, growth characteristics and pathogenicity. Further subdivision into serogroups and serovars was based on the serological determination of differences in the lipopolysaccharide coat; related serovars form serogroups. There are over 250 serovars from 24 serogroups (Ko et al. 2009). Serovars are adapted to different reservoir hosts, so the classification remains epidemiologically relevant. Genotypic classification has allowed further definition of species, but does not correlate with serological classification.

Epidemiology

Leptospires are environmentally resistant in water and moist soil. Outbreaks of disease are seasonal, linked with rainfall and ambient temperature (Lee et al. 2014; Major et al. 2014). Infection occurs via contact of broken skin or mucous membranes with contaminated water, soil or urine. Reservoir hosts are important in the maintenance of infection. They shed leptospires asymmetrically in urine for prolonged periods of time. The most important reservoir hosts are small rodents (Figure 1).

The major serogroups that infect dogs are: Icterohaemorrhagiae, Grippotyphosa (rare in UK and Ireland), Australis, Sejroe and Canicola (Ellis 2010). In Europe, dogs are more consistently exposed to Icterohaemorrhagiae (maintenance host: rats) and Australis (maintenance hosts: wildlife and dogs); Canicola (maintenance host: dogs) is declining in prevalence. Grippotyphosa and Sejroe are variable as their rodent hosts have differing distribution patterns. The prevalence of leptospirosis has been assessed through serosurveys (which determine exposure by detecting anti-leptospiral antibodies), and the detection of leptospires in urine (which determines active shedding
– either in acute or chronic infection). In an American study, over 20% of healthy, vet-visiting dogs had been exposed to Leptospira serovars (Stokes et al. 2007). Chronic shedding may be present in up to 8.2% of dogs (Harkin et al. 2003). Seroprevalence is higher in dogs in shelters and kennels and in dogs with access to outdoor water sources and/or wildlife (Ghneim et al. 2007; Rojas et al. 2010; Scanziani et al. 2002). However, dogs in urban and suburban environments without obvious exposure to these sources may also become infected. There has been no consensus on risk factors for acute disease.

**Pathophysiology of infection**

Leptospires are haematogenously spread. The capsule has a low endotoxic potential and leptospires can initially evade the immune response by binding inhibitors of complement activation. The host subsequently mounts an immune response to ongoing leptospiraemia. Once the immune system has cleared the infection, localised infection may persist in immunologically privileged sites such as the eye and renal tubules. Leptospires can cause disease in many tissues, including (but not limited to) liver, kidney, lungs, spleen, uvea/retina and endothelial cells. Virulence mechanisms and target organs in dogs may differ by Leptospira genotype (Koizumi et al. 2015). Vasculitis is seen, but is not always a primary event.

Renal infection is characterised as acute interstitial nephritis (De Brito et al. 2006). Glomerular abnormalities are also described (Mastrorilli et al. 2007). The leptospires induce cell damage and inflammation, which leads to a reduction in renal function in most patients. Hepatic infection is characterised by a cholestatic hepatitis; icteric and non-icteric forms can occur. The icteric form is more severe, being strongly associated with death or euthanasia (Major et al. 2014). Leptospiral pulmonary haemorrhage syndrome (LPHS) is a recently recognised severe manifestation of acute leptospirosis. Interestingly, the presence of leptospires within lung tissue is rare and not associated with the lesions. The syndrome is likely immune-mediated, with pulmonary IgG and IgM deposition (Schuller et al. 2015a). However, the pathophysiology is poorly understood and is likely multifactorial, involving both host and pathogen-related factors. Currently, there is no link between particular serovars and disease manifestation.

**Clinical disease**

Infection can lead to a variety of clinical pictures, with severity from sub-clinical to fatal. Clinical signs depend on the age of the animal, the host immune response, the virulence of the serovar and the infectious dose of leptospires received (Figure 2). Initial signs can be non-specific, such as fever, lethargy and inappetance. These develop into signs of multisystemic disease; up to 90% of cases have renal involvement and 10–20% have hepatic involvement (Harkin 2009). However, multiple organ systems can be involved and the clinical profile is often complex (Table 1).

**Figure 1:** Transmission of leptospirosis. Based on Schuller et al 2015b

**Figure 2a**

**Figure 2b**

**Figure 2c**

**Figure 2d**

**Figure 2:** Clinical signs of leptospirosis in dogs. (a) icteric sclera; (b) uveitis; (c) petechial haemorrhages; (d) pulmonary haemorrhage. All or none of these signs may be present, depending on the individual presentation. Images courtesy of Hospital Cliníic Veterinari, Universitat Autònoma de Barcelona, Spain.

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**Table 1:**

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<th>Maintenance Hosts</th>
<th>Environment</th>
<th>Incidental Hosts</th>
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<tr>
<td>Asymptomatic rodents</td>
<td>Soil and surface water</td>
<td>Wild and domestic animals</td>
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