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The clinical features of leishmaniosis vary widely as a consequence of the numerous pathogenic mechanisms of the disease process, the different organs affected, and the diversity of immune responses mounted by individual hosts (1, 2). In the past, both researchers and clinicians used the clinical classification of asymptomatic, oligosymptomatic and polysymptomatic dogs based only on the results of physical examination. This classification, however, has a limited value because it does not take into account the clinicopathological abnormalities and disregards dogs that have widespread organ dysfunction without apparent visual manifestations. The authors define dogs with clinical leishmaniosis when they have a confirmed *L. infantum* infection and present clinical signs and/or clinicopathological abnormalities which have been attributed to the infection or to the immune response to the infection. Dogs with subclinical infection, or clinically healthy but infected dogs, are defined as those that do not present clinical signs and/or clinicopathological abnormalities detected by routine examination techniques (physical examination, CBC, biochemical profile and urinalysis) but have a confirmed *L. infantum* infection (3).

The clinical features of leishmaniosis vary widely, because of the numerous pathogenetic mechanisms involved, the different organs affected, and the diversity of immune responses mounted by individual hosts. A broad range of immune responses and clinical manifestations have been described in CanL (2, 4, 5). *Leishmania* infection in dogs may be manifested as a subclinical infection, a self-limiting disease, or a non-self-limiting and severe illness. In dogs, the two opposite extremes of this clinical spectrum are characterized by: (1) protective immunity that is CD4 T cell mediated by the release of γ-interferon, IL-2 and TNFα that induce macrophage anti-leishmania activity, and (2) disease susceptibility that is associated with the production of a marked humoral non-protective immune response and a reduced or depressed cell mediated immunity with a mixed Th1 and Th2 cytokines response. Within this spectrum, clinical disease can range from a mild papular dermatitis associated with specific cellular immunity and low humoral responses to a severe renal disease characterized by glomerulonephritis due to immune complex deposition and associated with a massive humoral response and variable parasite loads (1, 2).

Canine leishmaniosis is a systemic disease that may potentially involve any organ, tissue and biological fluid and is manifested by non-specific clinical signs. The main clinical findings found on physical examination in classical CanL include skin lesions, generalized lymphadenomegaly, progressive weight loss, muscular atrophy, exercise intolerance, decreased appetite, lethargy, splenomegaly, polyuria and polydypsia, ocular lesions, epistaxis, onychogryposis, lameness, vomiting and diarrhea (4-15). The variable and non-specific clinical signs make the list of differential diagnosis to CanL widely extensive (1, 2, 4, 5).

Clinical staging systems are aimed to group patients in whom the severity of clinical picture and prognosis are the same. These systems are useful to evaluate the efficacy of different therapies, to decide what therapy is most suitable for each patient and also to consider a prognosis. The criteria of classification have to be simple and clinically-oriented, with the use of easy to perform diagnostic techniques. According to the established criteria, each patient is staged at a certain time point, most often at the moment of admission. Obviously, the stage can change as the disease deteriorates or improves during its course. We propose a system of four clinical stages, based on clinical signs, clinicopathological abnormalities and serological status, along with the type of therapy and prognosis most suitable for each stage (Table 1) (1).

Table 1. Proposal for clinical staging of canine leishmaniosis (1)
### References


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**Table 1. Proposal for clinical staging of canine leishmaniosis**

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<th>Stage</th>
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| **Stage I**  
Mild disease | Dogs with mild clinical signs such as peripheral lymphadenopathy, or papular dermatitis | Usually no clinical abnormalities observed. Normal renal profile: 
creatinine < 1.4 mg/dl, non-proteinuric. UPC < 0.5 | Scientific neglect / Alosurin alone | Good |
| **Stage II**  
Moderate disease | Dogs, which apart from the signs listed in stage I, may present: diffuse or symmetrical cutaneous lesions such as exfoliative dermatitis, alopecia, ulcers, plaques, nodules, lipoatrophy, mucocutaneous lesions, anorexia, weight loss, fever, and cachexia (Ferreira et al., 2008) | Clinico-pathological abnormalities such as mild non-regenerative anemia, hyperuricemia, hyperprolactinemia, serum hyperviscosity syndrome (Ferreira et al., 2008) | Alosurin + Medugani antoaminoside or Mefosine | Good to guarded |
| **Stage III**  
Severe disease | Dogs, which apart from the signs listed in stages I and II, may present signs originating from immune-complex lesions: vasculitis, arthritis, noesitis and glomerulonephritis | Clinico-pathological abnormalities listed in stage II Chronic kidney disease (CKD). IRS stage I with UPC > 1 or stage II (creatinine > 1.4 mg/dl) | Alosurin + Medugani antoaminoside or Mefosine Follow IRS guidelines for CKD | Guarded to poor |
| **Stage IV**  
Very severe disease | Dogs with clinical signs listed in stage III. Pulmonary 
neumonitis or nephrotic syndrome and end stage renal disease | Clinico-pathological abnormalities listed in stage II CKD IRS stage III (creatinine > 2.5 mg/dl) and stage IV (creatinine > 3 mg/dl) Nephrotic syndrome: marked proteinuria, UPC > 5 | Alosurin (alone) Follow IRS guidelines for CKD | Poor |

*Dogs with negative to medium positive antibody levels should be confirmed as infected with other diagnostic techniques such as cytology, nuclease sensitivity PCR.*

**High levels of antibodies are conclusive of a diagnosis of CanL, and are defined as 3-4 fold increased of a well established laboratory reference cut-off.

