L. infantum (plus co-infections)-induced inflammation and structural changes of splenic lymphoid tissue in human and canine visceral leishmaniasis.

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Severe forms of visceral leishmaniasis are associated with atrophy and histological disorganization of spleen compartments in both humans and dogs. In this work, we review the changes observed in the spleens of dogs from an area endemic for visceral leishmaniasis. Dogs with susceptibility markers for visceral leishmaniasis display disorganization of the splenic tissue more frequently than dogs without these markers ($\chi^2$-test, $P<0.01$). This disorganization was more intense in the marginal zone and the lymphoid follicle than in the periarterial lymphoid sheath. In addition, the total number of T lymphocytes (Whitney, $P < 0.05$), B lymphocytes (Whitney, $P < 0.01$) and S100+ dendritic cells (Whitney, $P < 0.01$) in the follicles and of B lymphocytes in the marginal zone (Whitney, $P < 0.01$) in animals with disorganized spleen architecture was lower than in animals with normal spleen histology. The architectural disorganization of the spleen is associated with lower expressions of LTα and CXCL13 than those observed in normal spleen. There is also a greater frequency of bacterial skin infections in animals with spleen cultures positive for Leishmania (47%) than in animals with negative cultures (21%). The architectural disorganization of the splenic tissue in dogs with susceptibility markers of visceral leishmaniasis is associated with aberrant expression of cytokines involved in the structuring of the follicles and marginal zone of the spleen. These splenic alterations may impair the leukocyte cooperation required for effective immunity against infection by Leishmania and other pathogens.

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