Canine sino-nasal aspergillosis: parallels with human disease

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Abstract

Canine sino-nasal aspergillosis (SNA) is characterized by the formation of a superficial mucosal fungal plaque within the nasal cavity and/or frontal sinus of systemically healthy dogs. The most common causative agent is Aspergillus fumigatus. The fungus does not invade beneath the level of mucosal epithelium but incites a severe chronic inflammatory response that leads to local destruction of nasal bone. These clinicopathological features are equivalent to those of human chronic erosive non-invasive fungal sinusitis. The clinical diagnosis of canine SNA relies on multiple modalities but local instillation of anti-fungal agents is an effective therapy with high cure-rate. Recent studies have investigated the immunopathogenesis of canine SNA. The mucosal inflammatory infiltrate involves a mixture of CD4+ and CD8+ T lymphocytes, IgG+ plasma cells and activated macrophages and dendritic cells expressing class II molecules of the major histocompatibility complex. There is active recruitment of blood monocytes and neutrophils. Real-time quantitative reverse transcriptase polymerase chain reaction (qRT-PCR) analysis of mucosal tissue samples has revealed up-regulation of Th1 (IL-12, IL-18 and IFN-gamma), Th17-related (IL-23) and pro-inflammatory (IL-6, TNF-alpha) cytokine mRNA with evidence of expression of genes encoding monocyte chemoattractant proteins 1-4. Additionally, there is significant transcription of the IL-10 gene consistent with local immunosuppression that prevents secondary immune-mediated sequelae whilst permitting chronicity of the infection. The source of this IL-10 may be a T regulatory population or a Th1 population that switches phenotype during the course of disease. This understanding of the immunopathogenesis of canine SNA establishes this disorder as a valuable model for the equivalent human pathology.

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