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Phenotypic and functional characteristics of HLA - DR$^+$ neutrophils in Brazilians with cutaneous leishmaniasis

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Abstract

The protozoan *Leishmania braziliensis* causes cutaneous leishmaniasis (CL) in endemic regions. In murine models, neutrophils (PMNs) are recruited to the site of infection soon after parasite inoculation. However, the roles of neutrophils during chronic infection and in human disease remain undefined. We hypothesized that neutrophils help maintain a systemic inflammatory state in subjects with CL. Lesion biopsies from all patients with CL tested contained neutrophils expressing HLA - DR, a molecule thought to be restricted to professional antigen - presenting cells. Although CL is a localized disease, a subset of patients with CL also had circulating neutrophils expressing HLA - DR and the costimulatory molecules CD80, CD86, and CD40. PMNs isolated from a low - density leukocyte blood fraction (LD - PMNs) contained a higher percentage of HLA - DR$^+$ PMNs than did normal - density PMNs. In vitro coculture experiments suggested LD - PMNs do not suppress T cell responses, differentiating them from MDSCs. Flow - sorted HLA - DR$^+$ PMNs morphologically resembled conventional PMNs, and they exhibited functional properties of PMNs. Compared with conventional PMNs, HLA - DR$^+$ PMNs showed increased activation, degranulation, DHR123 oxidation, and phagocytic capacity. A few HLA - DR$^+$ PMNs were observed in healthy subjects, and that proportion could be increased by incubation in either inflammatory cytokines or in plasma from a patient with CL. This was accompanied by an increase in PMN *hladrb1* mRNA, suggesting a possible connection between neutrophil “priming” and up - regulation of HLA - DR. These data suggest that PMNs that are primed for activation and that also express surface markers of antigen - presenting cells emerge in the circulation and infected tissue lesions of patients with CL.

Citing Literature

Supporting Information