HISTOLOGICAL ALTERATIONS IN ADIPOSE TISSUE IN HAMSTERS INFECTED WITH L. chagasi

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Introduction: Adipose tissue had a variety of functions, including energy storage, hormonal regulation of homeostatic systems and thermogenesis. Some authors have discussed the importance of adipose tissue in regulation of immune response and inflammation. Leishmaniasis is an infection caused by trypanosomatid Leishmania sp. There are different clinical forms, depending on characteristics of the host and the species of Leishmania. Visceral leishmaniasis (VL) is the most severe clinical form of this infection. Objectives: Investigate the role of adipose tissue in pathogenesis of VL using hamsters infected with Leishmania chagasi as an experimental model. Material and Methods: Golden hamsters (Mesocricetus auratus) were infected with L. chagasi (10⁵ parasites intradermic injected in the ear). Controls (n = 10) and infected animals (n = 10) were followed for nine months and the body weight were monitored weekly. After 3, 5 and 7 months, hamsters were euthanized and serum, spleen, liver and adipose tissue were removed and stored for subsequent analysis. Retroperitoneal and the epididymal adipose tissue were fixed in 10% formalin solution and were processed for histological analysis. The parasite load was evaluated by the limiting dilution technique, in the spleen and liver. Results: After 3 months of infection, hamsters exhibited parasites in spleen (1.15x10⁹ ± 6.52x10⁸ parasites) and liver (6.61x10⁶ ± 5.25x10⁶ parasites). The histological analyses of adipose tissue, showed the presence of basophilic intracytoplasmic inclusions in adipose tissue of infected animals. There were no differences between infected hamsters and controls in the weight of epididymal and retroperitoneal adipose tissue in 3 (2632±574.1mg, 2445±662.6mg, P=0.413; 1803±156.4mg, 1665±353.1mg, P=0.5476, respectively), 5 (1652±197.9mg, 1580±289.8mg, P=0.6612; 1111±346.3, 110.3, P=0.6612) and 7 (2123±629.9mg, 2089±351.2, p=1.000; 1338±290.0, 1399±314.0, p=0.6286) months of infection. Conclusions: These initial results indicate that adipose tissue could be involved in pathogenesis of VL.

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