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Original article

Leptin and adiponectin in patients with systemic lupus erythematosus: clinical and laboratory correlations

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ABSTRACT

Objective: To evaluate the serum levels of leptin and adiponectin in patients with systemic lupus erythematosus (SLE) and correlate their levels with disease activity, presence of autoantibodies and clinical manifestations.
Methods: 52 women with SLE and 33 healthy women were evaluated. The patients were divided into two groups, the first with active SLE and the second with inactive SLE. Patients with SLEDAI $\geq$3 were considered active. Serum levels of leptin (ng/mL) and adiponectin (μg/mL) were measured by enzyme immunoassay.
Results: There was a significant difference in leptin levels between SLE and controls (20.7 ± 17.1 vs. 8.0 ± 5.0 ng/mL, $p < 0.001$), but no significant difference in adiponectin levels ($87\ 5 \pm 69.7$ vs. 118.1 ± 70.6 pg/mL, $p = 0.053$). No significant difference in levels of leptin and adiponectin was noted between inactive and active SLE groups. There was a significant association between low levels of leptin and positivity for anticardiolipin (aCL) ($p = 0.025$) and lupus anticoagulant (LA) ($p = 0.003$) and a significant association between high levels of leptin and the presence of renal disease ($p < 0.001$). However, there was no association between adiponectin levels with autoantibodies and clinical features in SLE patients.
Conclusion: Patients with SLE had elevated leptin levels, with association with renal involvement. Leptin and adiponectin were not correlated with disease activity. Low levels of leptin have been associated with the presence of LA and aCL.

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Leptina e adiponectina no lúpus eritematoso sistêmico: correlações clínicas e laboratoriais

Palavras-chave:
Lúpus eritematoso sistêmico
Leptina
Adiponectina

RESUMO

Objetivo: Avaliar os níveis séricos de leptina e adiponectina em pacientes com lúpus eritematoso sistêmico (LES) e correlacionar seus níveis com atividade inflamatória, presença de autoanticorpos e manifestações clínicas.

Métodos: Foram avaliadas 52 mulheres com LES e 33 mulheres saudáveis. As pacientes foram divididas em dois grupos, o primeiro com LES ativo e o segundo com LES inativo. Foram consideradas em atividade as paciente com Sledai ≥ 3. Os níveis séricos de leptina (ng/mL) e adiponectina (ug/mL) foram medidos por ensaio imunoenzimático.

Resultados: Houve diferença significativa nos níveis de leptina entre LES e controle ($20{,}7 \pm 17{,}1$ vs. $8{,}0 \pm 5{,}0$ ng/mL, $p < 0{,}001$), mas não houve diferença significativa nos níveis de adiponectina ($87{,}5 \pm 69{,}7$ vs. $118{,}1 \pm 70{,}6$ ug/mL, $p = 0{,}053$). Entre LES inativo e ativo, não houve diferença significativa dos níveis de leptina e adiponectina. Houve uma associação significativa entre os baixos níveis de leptina e positividade para anticardiolipina (aCL) ($p = 0{,}025$) e anticoagulante lúpico (LA) ($p = 0{,}003$) e uma associação significativa entre níveis elevados de leptina e da presença de manifestação renal ($p < 0{,}001$). No entanto, não houve associação entre adiponectina com autoanticorpos e características clínicas nas pacientes.

Conclusão: Pacientes com LES apresentaram nível elevado de leptina, com associação ao envolvimento renal. A leptina e a adiponectina não se correlacionaram com a atividade da doença. Baixos níveis de leptina foram associados com a presença de LA e aCL.

Introduction

In recent years, a major route linking metabolism and the immune system has been described, after verification of an inflammatory state in obesity.[1] The adipose tissue is an organ that performs neuroendocrine and immune functions, producing various cytokines that include IL-6 and TNF-alpha, leptin, adiponectin and resistin, known as adipokines. These cytokines participate in a variety of physiological processes, such as food intake, insulin sensitivity, atherosclerosis, immunity and inflammation.[2] They represent a new group of proteins released from adipocytes, which act as modulators of the immune system.[3] Studies demonstrate the participation of these substances in rheumatic and inflammatory diseases.[4–7]

Leptin acts on the immune system as a proinflammatory cytokine. In animal models, its deficiency is associated with an increased susceptibility to infection and reducing the inflammation.[8] It promotes the proliferation and activation of T lymphocytes and induces production of Th1 cytokines.[1,9,10] Studies have reported increased leptin levels in systemic lupus erythematosus (SLE) patients.[11–13]

Adiponectin has anti-inflammatory action.[14] It inhibits the proliferation and activation of T lymphocytes, as well as lymphopoiesis and B lymphocytes.[15] High levels of adiponectin were found in patients with SLE,[12,16,17] although there is still controversy.

The aim of this study was to evaluate the levels of leptin and adiponectin in patients with SLE and its possible correlation with disease activity, presence of autoantibodies and clinical manifestations.

Patients and methods

52 female patients who met the American College of Rheumatology (ACR) classification criteria for SLE,[18] hospitalized and/or in outpatient care at the Rheumatology Department, Hospital das Clínicas, Medicine School, Universidade Federal de Goiás (HC/FM/UFG) were included.

The patients were divided into two subgroups: a subgroup of patients with active SLE ($n = 21$) and another subgroup of patients with inactive disease ($n = 31$). The control group comprised 33 healthy women matched for age. The exclusion criteria were: patients younger than 18 years old, pregnancy, history of myocardial infarction or diabetes, liver disease, renal failure, prednisone >20 mg/day and body mass index (BMI) >30 kg/m^2.

The study was approved by the Research Ethics Committee of the HC/UFG and all participants who agreed to participate signed an informed consent form.

The evaluation of patients included demographics, age at disease onset, disease duration, clinical manifestations and physical examination. At the time of evaluation, lipid profile, fasting glucose and tests of inflammatory activity were also obtained for each patient. Autoantibodies found in the clinical record were considered; for those patients for whom autoantibodies were not found, these were requested at the time of their inclusion in the study.

To obtain the profile of autoantibodies, ANA and anti-DNA were used; these tests were performed at the Immuno-Rheumatology Laboratory, HC/FM/UFG. ANA was obtained by indirect immunofluorescence on HEp-2 cells (Hemagen Diagnostics, Inc.) and anti-DNA by indirect immunofluorescence