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AB0130

SERUM ATHEROGENICITY IN WOMEN WITH UNTREATED SYSTEMIC LUPUS ERYTHEMATOSUS

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Background: Systemic lupus erythematosus (SLE) is associated with an unexplained increase cardiovascular risk. The nature of the factors that contribute to progression of atherosclerosis were identified using the method for determining the atherogenicity of blood serum in cell culture in cell culture (in vitro). The term "atherogenicity" is meant as the ability of the serum and/or its components to induce intracellular accumulation of cholesterol in cultured cells.

Objectives: To compare atherogenicity of blood serum in women with untreated SLE and healthy women.

Methods: Thirty seven women (median age 30[21;39] years) with active SLE (median disease duration 45[3;102] months; SLADAI 17 [8;34]) were enrolled in the study. Lupus nephritis are defined in 15 (41%), Antiphospholipid syndrome (APS) – in 8 (22%) of 37 SLE patients (pts). The control group consisted of 30 women, median age 31[25;39] years.

Atherogenicity of blood serum was determined in the culture of murine macrophages. Peritoneal macrophages were isolated from the ascitic fluid of the line mice according to the generally accepted method J. Goldstein et al (1979y). Serum atherogenicity was determined by the accumulation of intracellular cholesterol induced by 10% of the blood serum of the patients, and expressed as a percentage of the content of cholesterol in the control cells.

Results: Elevated atherogenicity of blood serum was detected more frequently in SLE pts (24/72 (65%) vs healthy controls (5/30 (17%), $p < 0.01$). The blood serum of SLE pts caused a 3-7-fold accumulation of intracellular cholesterol, which significantly differed from healthy women ($203 \pm 136\%$ vs $127 \pm 42\%$, $p < 0.001$). The ability to stimulate the accumulation of cholesterol esters in murine macrophages was not associated with age, duration of the disease, lipid spectrum and was the highest in pts with nephritis ($305 \pm 141\%$ vs $180 \pm 52\%$, $p < 0.05$) and APS ($253 \pm 130\%$ vs $119 \pm 75\%$, $p < 0.05$).

Conclusion: Serums of women with untreated SLE may stimulate the accumulation of cholesterol in mouse macrophages unlike of healthy women.

The highest atherogenicity was found in blood serum of SLE pts with nephritis and APS.

Disclosure of Interests: None declared

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AB0131

RESPIRATORY TRACT POLY(I:C) STIMULATION ACCELERATES SALIVARY GLAND IMMUNE DYSFUNCTION IN SPONTANEOUS SJÖGREN'S SYNDROME ANIMAL MODEL

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Background: Sjogren's syndrome is one of the most common autoimmune diseases, with a prevalence of 0.33% to 0.77% in Chinese people, characterized by focal infiltration of lymphocytes in glands and the production of multiple autoantibodies. Studies have shown that virus infection may play a crucial role in the occurrence and development of this disease.

Objectives: It has been shown that airway stimulation with poly(I:C) can mimic respiratory tract viral infection to some extent. Thus, this study was aimed to investigate the dynamic immune responses in salivary gland after respiratory tract poly(I:C) stimulation in NOD mice.

Methods: The 5-week-old NOD mice were given respiratory tract poly(I:C) stimulation to mimic the respiratory virus infection once every other day for a total of 5 times (the total dose is 100µg), and the control group were given the same dose of sterile PBS. After 8 weeks, the mice were sacrificed to obtain and analyze the salivary gland tissues.

Results: We found that the salivary gland flow rate was decreased and the blood glucose was influenced by the Viroid stimulation during the early stage in poly(I:C) stimulated group compared with that in PBS group. Accordingly, the pathological injury of salivary gland tissues in poly(I:C) stimulated group was more serious, including decreased volumes of the salivary glands, increased number of pathological focus score and the increased area of lymphocyte infiltration. Furthermore, we found that the expression of IL-33 in salivary glands of

poly(I:C) stimulated NOD mice was increased, especially the expression of IL-33 in the acini and ducts. Moreover, the expression of IFN-I and IFN-II is up-regulated in salivary glands.

Figure1: Functional change of the NOD mice in different groups.

A. The dynamic change of the salivary gland flow rate; B. The dynamic change of the blood glucose

Figure2: Pathological changes of salivary glands in mice after different treatments.

A. The pathological changes of different groups; B. The quantitative analysis of the volume of salivary glands in different groups; C. The quantitative analysis of the numbers of pathological lesions of salivary glands in different groups; D. The quantitative analysis of the areas of pathological lesions of salivary glands in different groups.

Figure3: The differential expression of IL-33 in the salivary glands.

A. The expression of IL-33 in different sites of salivary glands; B. The quantitative analysis of the expression of IL-33 in the acini cells site; C. The quantitative analysis of the expression of IL-33 in the ductal cells site; D. The quantitative analysis of the expression of IL-33 in the lymphocytic aggregation site.

Conclusion: The results of this study suggest that respiratory tract poly(I:C) stimulation accelerates salivary gland immune dysfunction in spontaneous sjogren's syndrome NOD mice, which mechanisms need to be further investigated.

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