**Sjögren’s syndrome (SS) is a chronic autoimmune disease characterized by a progressive lymphocytic and plasmacellular infiltration of the salivary and lacrimal glands leading to a progressive dryness of the mouth and eyes.**

1. **Introduction**

   Sjögren’s syndrome (SS) is a chronic autoimmune disease characterized by a progressive lymphocytic and plasma cell infiltration of the salivary and lacrimal glands leading to the serostasis and xerophthalmia (secia complex) (1,9,35). SS is associated with the production of autologous factors (RF) and a variety of antigenic autoantibodies among them autoantibodies against SS-A/Ro and SS-B/La are the most important. SS displays a broad clinical spectrum extending from sicca symptom to the extraglandular (systemic) disease affecting the lungs, kidneys, blood vessels, and muscles. SS can occur alone (primary) or in association with other autoimmune diseases (secondary) (24).

   The trapping of leukocytes from the blood stream and their subsequent rolling along the activated endothelial cell lining of postcapillary venules are the earliest signs of inflammation. Rolling is an essential element of the multi-step cascade leading to the leukocyte recruitment into sites of inflammation. It is mediated by the interactions between E-selectins on the surface of activated endothelial cells and their ligands, which are heavily glycosylated surface molecules of leukocytes e.g. CD15 molecule of granulocytes. Granulocytes, monocytes and a subset of memory T cells could be bound through this interaction. Next step is mediated through the interaction between adhesion molecules and their ligands, which are heavily glycosylated surface molecules of leukocytes e.g. CD15 molecule of granulocytes, leukocytes, eosinophils, basophils, and monocytes (11,16,21,37).

   **Membrane adhesion molecules are shed into the body fluids by the proteolytic cleavage or by alternative splicing on the level of mRNA (15). The elevated levels of soluble adhesion molecules are found in the serum of patients with various inflammatory diseases, and may provide some useful diagnostic or prognostic informations (14).**

   Neopterin is produced by macrophages after activation with interferon gamma, a cytokine produced by CD4+ helper-inducer lymphocytes. Increased neopterin concentration thus reflects the activation of specific cellular immunity. An elevation of serum or urine neopterin levels has been proven in autoimmune disorders including rheumatoid arthritis, systemic lupus erythematosus, inflammatory bowel disease, or sarcoidosis (13,39).

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**Original Article**

**SEROUS SOLUBLE ADHESION MOLECULES (sICAM-1, sVCAM-I, sESELECTIN) AND NEOPTERIN IN PATIENTS WITH SJÖGREN’S SYNDROME**

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Summary: Sjögren’s syndrome is a systemic autoimmune disease characterized by focal lymphocytic infiltration of the salivary and lacrimal glands. Expression and up-regulation of adhesion molecules and activation of cellular immune system is essential for the migration of inflammatory cells into tissues. Soluble forms of adhesion molecules sICAM-1, sVCAM-I, sE-selectin and neopterin were analyzed in serum of 17 patients with primary Sjögren’s syndrome and 11 patients with secondary Sjögren’s syndrome together with 26 age-matched healthy blood donors. There were significantly higher serum concentrations (mean ± 1SD) of sICAM-1 (362.0 ± 67.9 ng/ml, p<0.001), sE-selectin (78.7 ± 28.1 ng/ml, p<0.001) and neopterin (17.9 ± 6.4 nmol/l, p<0.001) in primary Sjögren’s syndrome patients in comparison to control group (sICAM-1: 128.3 ± 46.9 ng/ml, sE-selectin : 46.3 ± 39.5 ng/ml, and neopterin : 7.6 ± 2.3 nmol/l). Sera from patients with secondary Sjögren’s disease contained significantly higher levels of sICAM-1 (356.0 ± 62.4 ng/ml, p<0.001), sE-selectin (65.5 ± 27.0 ng/ml, p<0.05), and neopterin (18.8 ± 9.8 nmol/l, p<0.001) in comparison with control group. There were no significant differences between patients with primary and secondary Sjögren’s syndrome in any parameters tested. No statistically significant differences in serum levels of sVCAM-1 were found either in patients with primary or secondary SS compared to control group.

**Key words:** Neopterin; sE-selectin; sICAM-1; Sjögren’s syndrome; sVCAM-1
We found significantly higher serum levels of neopterin, sICAM-1, and sE-selectin in patients with both primary and secondary SS in comparison with healthy controls in this study.

**Material and methods**

**Patients**

This study was approved by the Ethical Committee of Charles University School of Medicine, Hradec Králové. Informed consent of all participants was obtained.

The serum samples were drawn from 17 patients (15 females, 2 males, average age 58 years, range 41-79 years) with primary SS and 11 patients with secondary SS (11 females, average age 64, range 47 - 81 years). All patients met criteria for the classification of SS according to Vitali et al. (38). The age-matched control group consists of 26 healthy female blood donors.

**Blood sampling**

Peripheral blood samples were collected by venipuncture into sterile tube (Sarstedt, Nuremberg, Germany). Samples were left for 1.5 hours at room temperature. Serum samples were stored frozen at -20°C and thawed only immediately before processing.

**Measurement of adhesion molecules**

Serum concentrations of sICAM-1, sVCAM-1, and sE-selectin were obtained by use commercial ELISA kits Parameter Human Soluble ICAM-1, Parameter Human Soluble VCAM-1 and Parameter Human Soluble E-Selectin manufactured by RD Systems, Minneapolis, MN., USA. Serum levels of neopterin was measured by ELISA using a commercial kit (IBL, Germany). All ELISA techniques were performed according to the manufacturer’s instructions. All measurement were done with the same batch, and in a duplicate.

**Statistical analysis**

The analysis was done using SigmaStat 2.0 statistical software (Systat Software, USA). The data were tested for normality. The differences between groups were calculated by t-test or Mann-Whitney Rank Sum test.

**Results**

sICAM-1: Statistically significant differences (p<0.001) in the levels of serum sICAM-1 were found in both groups of SS patients with primary SS (362.0±67.9 ng/ml) and secondary SS (356.0±62.4 ng/ml) in comparison with controls (128.3±46.9 ng/ml)(Fig. 1.).

sE-selectin: Serum levels of sE-selectin were significantly elevated in patients with primary SS (78.8±28.1 ng/ml, p<0.001) in comparison with controls (46.3±39.5 ng/ml) as well as significant increase in concentrations of sE-selectin was observed in patients with secondary SS (65.5±27.1 ng/ml, p<0.05) in comparison to the control group (Fig. 4.).

**Discussion**

Sjögren’s syndrome is a chronic inflammatory disease of unknown origin marked by inflammation and destructio of the salivary and lacrimal glands. The impairment of salivary and lacrimal gland functions is caused by the destruction of acini and ductal cells accompanied by lymphocytic infiltration, which is believed to be immunologically mediated (35). CD4+CD45RO+ memory helper-inducer T-cells are the major subsets infiltrating the exocrine glands of Sjögren's syndrome patients. B cells subsequently appear in the lesions (2). Leukocytes adhesion is a crucial step in the development of both normal immune response and inflammation (7,16). Adhesion of leukocytes is mediated through the multiple interactions between adhesion molecules and their ligands. More recently we have learned that soluble isoforms of these adhesion structures can be found in the circulation and their levels can serve as a surrogate marker of disease activity (14,32). The information obtained from measurement of soluble adhesion molecules can be interpreted at several different levels. The expression and subsequent release of soluble cellular adhesion molecules is mediated by pro-inflammatory stimuli both exogenous and endogenous origins such as endotoxin, histamine, thrombin, and various cytokines. Soluble cellular adhesion molecules may be regarded simply as markers of the presence and intensity of inflammation. The clinical utility of monitoring levels of soluble adhesion molecules is not yet established, but it is supposed that the availability of commercial assay kits should allow their evaluation in many clinical settings (15).

We found significantly elevated serum levels of ICAM-1, VCAM-1, and E-selectin in serum of both primary and secondary SS patients in comparison with healthy control. These findings could reflect hyperactivation of the immune system of SS patients which is well documented (35). The hallmark of SS are hypergammaglobulinemia, presence of autoantibodies as the results of polyclonal activation of B cell system which is under the control of T cell. The putative autoantigen (or autoantigens) in salivary glands of SS patients has to be presented to T cells after binding to HLA class I or II molecules. These specific interactions that are accompanied by the costimulatory and accessory interactions lead to the production of cytokines upregulating the expression of adhesion molecules in affected tissues. It has to be stressed that there are numerous sources of such cytokines in salivary glands, including macrophages, epithelial and endothelial cells, T and B cells and fibroblasts. It could not be possible to distinguish among contributions of particular cell type from the serum levels of soluble adhesion molecules. Saito et al. (30, 31) reported that the expression of ICAM-1 in salivary glands of patients affected by SS is higher than in normal salivary gland. This upregulation of ICAM-1 correlated with the intensity of T cell infiltration, which are the only source of interferon gamma enhancing the expression of ICAM-1. Johansen et al. (18) found higher...
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neopterin: Sera from patients with primary SS contained more neopterin (17.9 ± 6.4 nmol/l, p<0.001) than control group (7.6 ± 2.3 nmol/l)(Fig. 2.). There were also significant differences in serum levels of neopterin between patients with secondary SS and control group (18.8 ± 9.8 nmol/l)(Fig. 2.).

There were no significant differences between groups of patients with primary and secondary SS in any parameter tested.

**Highly significant correlations were observed in patients with SS: between serum levels of neopterin and sICAM-1 (coefficient of correlation r=0.620, p<0.001, Figure 5.), between serum levels of sICAM-1 and sVCAM-1 (r=0.569, p<0.001), and between sVCAM-1 and neopterin (r=0.599, p<0.001), but not in control group (sICAM-1 x neopterin r=0.006, p>0.05; sICAM-1 x sVCAM-1 r=0.171, p>0.05; sVCAM-1 x neopterin r=0.035, p>0.05).

**Discussion**

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Usefulness of procalcitonin for differentiation between activity of systemic auto-immune disease (systemic lupus erythematosus/ systemic antineutrophil cyto-
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leucocytes could adhere through the LFA-1-beta2 integrin which is expressed on all leucocytes. In a sharp contrast, in the case of VCAM-1 the adherence of leucocytes is much more selective because VCAM-1 is a ligand for betal integrin VLA-4. Enhanced expression of VCAM-1 on the endothelium may facilitate the transmigration of leucocytes and CD4+ memory effectors cells, since these cells express VLA-4, whereas "naive" CD4+ T cells do not (17).

Neopterin is an oxidation product of 7,8-dihydrobenzo-
quinone that is synthesized from guanosine triphosphate (GTP) by the enzyme GTP cyclohydrolase I. The production of 7,8-dihydrobenzoinoterin represents a first step in the mechanism leading to the synthesis of tetrahydrobenzoquinone, an enzyme cofactor. For some yet unknown reason the activity of GTP cyclohydrolase I is present in a great excess compared to the enzymes active distally in the tetrahydrobo-
quinone pathway in human macrophages after treatment with interferon gamma leading to a remarkable rise in 7,8-dihy-
drobenzoinoterin. 7,8-dihydrobenzoquinone is in equilibrium with its oxidized form, neopterin, which can be monitored in body fluids (22,23,29).

Increased concentration of neopterin in serum or in uri-
in has been documented in many autoimmune disorders, including rheumatoid arthritis, systemic lupus erythema-
sus, sarcoidosis, or inflammatory bowel disease, and in-
creased neopterin levels have been associated with disease activity (30,32,35).

Numerous factors, genetic, infectious, immunological and environmental are thought to play a role in the patho-
geny of SS. Systemic lupus erythematosus has been recently in-
vestigated in the diagnosis of dysregulation of the immune system in SS patients. Our results support immunopathological e-
tiology of this enigmatic disease.

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