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Levels of Serum Soluble P-Selectin and E-Selectin in Psoriatıc Patients

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Dear Editor:

Psoriasis is an immune-mediated, chronic inflammatory disease[1]. Selectins generate the first adhesive stage contributing to the tethering and rolling of leukocytes into the vascular endothelium for the distribution of immune cells to the inflamed tissue. This essential role is a potential target for the development of novel treatments[2]. The main aim of our study was to determine the levels of serum soluble P-selectin (sP-selectin) and soluble E-selectin (sE-selectin), which contribute to the inflammatory process in the pathogenesis of the disease, in patients and controls; and to assess the association between sP-selectin and sE-selectin levels and the severity of the disease. We also intended to evaluate whether these molecules contribute to the pathogenesis of psoriasis, which is known to have multifactorial etiologies. Between July 2012 and September 2012, twenty-four patients (9 males, 15 females) who were diagnosed with psoriasis clinically and/or histopathologically in our clinic were included in this study. The control group consisted of 24 healthy age-matched individuals with comparable demographical features (10 males, 14 females) and without any infectious, systemic or dermatological diseases, and who were not smokers. All participants signed the consent form prior to the study. Consent was also obtain from the local ethical committee (IRB approval: Necmettin Erbakan University Faculty of Medicine Ethics Committee, 2012-40). Patients who received systemic medication and/or phototherapy and topical antipsoriatic treatment were excluded from the study. Other factors for being excluded from the study were as follows: pregnancy, age less than 18 years, smoking, hypertension, diabetes mellitus, chronic renal failure, liver and cardiac failure, acute and chronic infections, autoimmune diseases and cancer. Peripheral blood samples were obtained from both the study and control groups. The samples were centrifuged at 3,500 rpm for 4 minutes, and the serum was stored at −80°C. Psoriasis area and severity index (PASI) scores of the patients with psoriasis were recorded. The data were statistically assessed. Statistical analysis was done with Mann-Whitney U using SPSS 15.0 (SPSS Inc., Chicago, IL, USA). A *p*-value less than 0.05 was considered significant. The mean age was

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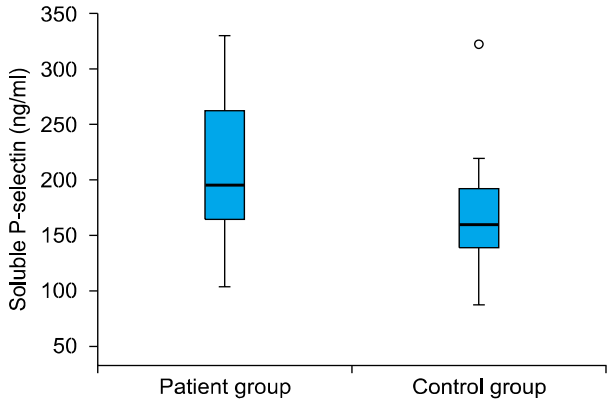

Fig. 1. Serum soluble P-selectin levels in the patient and control groups.

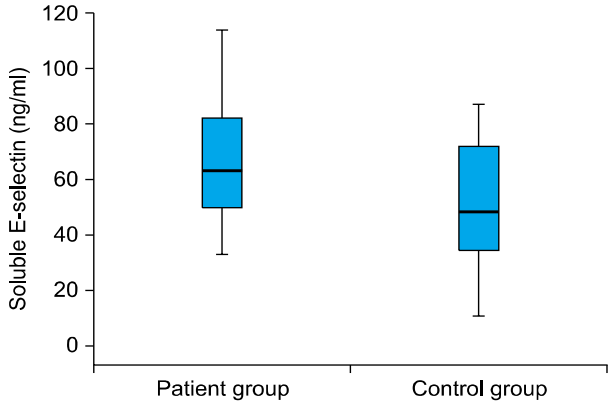

Fig. 2. Serum soluble E-selectin levels in the patient and control groups.

43.12±15.74 years (minimum 18 years, maximum 70 years) in the study group and 42.04±11.63 years (minimum 22 years, maximum 64 years) in the control group. The mean PASI scores were calculated as 11.058±9.21 (minimum 3, maximum 36.6) in the patient group. The mean sP-selectin level was 212.45±68.15 (minimum 104.34, maximum 331.01) in the patient group and 168.20±47.61 (minimum 86.64, maximum 324.34) in the control group. On the other hand, the mean sE-selectin was found as 65.84±20.19 (minimum 33.41, maximum 114.12) in the patients and 51.35±22.10 (minimum 11.50, maximum 87.15) in the controls. A comparison of both serum sE-selectin and sP-selectin levels between the patient and control groups revealed a statistically significant difference ($p<0.05$) (Fig. 1, 2). However, there was no significant correlation between sE-selectin, sP-selectin levels and the PASI scores among the patients ($r=0.014$, $p>0.05$; $r=0.017$, $p>0.05$ respectively). Various studies have been performed regarding the role of selectins in the etiopathogenesis of psoriasis; however, no clear result has been obtained so far. Our aim of this study was to assess the role of serum sP-selectin and sE-selectin levels in the pathogenesis and their effect on disease severity. P-selectin is released to the systemic circulation from platelets and is activated by thrombin or histamine during coagulation and subsequenty facilitates leukocyte-platelet adhesion. Endothelial P-selectin directs platelets to postcapillary venules during the inflammatory response, and P-selectin in platelets causes thrombus formation by platelet and leukocyte congregation. P-selectin, secreted from activated platelets, facilitates leukocytes rolling, the first step in leukocyte extravasation[3]. Tamagawa-Mineoka et al.[4] noted that sP-selectin levels were significantly higher in psoriatic patients and then regressed to prior levels following treatment. Furthermore, Garbaraviciene et al.[3] reported a significant correlation between P-selectin and PASI and concluded that platelet P-selectin expression could be used as a successful biomarker to monitor the treatment of psoriasis. In another study, no correlation was found between sP-selectin levels and the severity of the disease in the patient group compared to the control group[5]. On the other hand, in our study, serum sP-selectin levels were significantly higher in the patient group compared to that in the control group, and there was no correlation between serum sP-selectin levels and disease severity. E-selectin is a 115-kDa single-chain glycoprotein, previously known as endothelial leukocyte adhesion molecule. E-selectin plays an important role in the adhesion of neutrophils, eosinophils, memory T-cells and tumor cells to the vascular endothelium via carbohydrate ligands such as sialyl Lewis X[6]. Factors that determine the processe of conversion to the soluble form are tumor necrosis factor-α, interleukin-1 and lipopolysaccharides. Psoriatic plaques are characterized by strong E-selectin expression on the luminal surfaces of the capillary and the postcapillary endothelial cells. Interestingly, E-selectin is not expressed in the synovial joints affected by psoriatic arthritis. Furthermore, despite continuous E-selectin upregulation and constant cytokine stimulation in the psoriatic skin, E-selectin expression is temporary[7]. Consistent with our study, some studies have reported that sE-selectin levels were increased in the serum of psoriatic patients compared to that of controls[8-10]. However, other studies have found that the levels of sE-selectin were not significantly increased in patients with psoriasis[5]. We did not find any significant correlation between the levels of serum sE-selectin and sP-selectin and PASI scores of psoriatic patients. Czech et al.[8] also did not discover any correlation between these parameters. This may be due to the

small number of patients enrolled in our study. However, there are studies that have reported a correlation between serum sE-selectin levels and disease severity in these patients[9,10]. In conclusion, this study demonstrates that serum sE-selectin and sP-selectin levels of the patient group were significantly higher that those of the control group. However, there was no significant correlation between the levels of sE- selectin and sP-selectin and PASI score in the psoriatic patients. This study emphasizes the potential role of P-selectin and E-selectin adhesion molecules in the complex etiopathogenesis of psoriasis.

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