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RISK FACTORS FOR SYSTEMIC LUPUS ERYTHEMATOSUS

Abstract

This study aimed to investigate the risk factors associated with the development of systemic lupus erythematosus. Between 2008 and 2019, a total of 72 patients with systemic lupus erythematosus and 142 healthy individuals were examined at the 1st Clinic of Samarkand State Medical University. A history of hypertension increased the risk of developing SLE. Patients were more likely to report angina pectoris compared to the control group. A statistically significant association was found between a family history of any autoimmune disease and an increased risk of SLE. Individuals who smoked between 2 to 5 packs of cigarettes per week had a 2.64 times higher risk compared to non-smokers, although none of the results reached statistical significance.

***Keywords:** systemic lupus, risk factors, alcohol, stress, family history.*

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ФАКТОРЫ РИСКА РАЗВИТИЯ СИСТЕМНОЙ КРАСНОЙ ВОЛЧАНКИ

Целью было изучение факторов риска, ассоциированных с развитием системной красной волчанки. В период с 2008-2019 гг. в 1-й клинике Самаркандского государственного университета были обследованы 72 больных с СКВ и 142 здоровых лиц. Риск развития СКВ повышало наличие гипертонии в анамнезе. Больные чаще сообщали о стенокардии по сравнению с контрольной группой. Была выявлена статистически значимая связь между семейной историей любого аутоиммунного заболевания и

повышенным риском развития СКВ. У тех, кто выкуривает от 2 до 5 пачек сигарет в неделю, риск развития СКВ был выше, чем у некурящих в 2,64 раза, однако ни один из результатов по этому воздействию не был статистически значимым.

Ключевые слова: волчанка, факторы риска, алкоголь, стресс.

Introduction. Systemic lupus erythematosus (SLE) is a multisystem autoimmune disease characterized by the production of numerous B cells producing hyperactive autoantibodies and involvement of skin, joints, kidneys, brain, serosal surfaces, blood vessels, blood cells, lungs and heart [1,3]. While genetic and hormonal factors are proved to be significantly important, other risk factors, including different environmental exposure, may have equal importance in the aetiology of SLE. According to recent research, many environmental factors may act collectively to cause SLE in a genetically susceptible person [2,4]. It is hypothesized that some drugs containing aromatic amines have been proposed to cause SLE [5-7]. Therefore, numerous studies investigated environmental agents containing chemical components, especially aromatic amines, such as tobacco smoke and hair dyes. We undertook a clinic-based case-control study to investigate potential risk factors for developing SLE in the 1st Clinic of Samarkand State Medical Institute.

Materials and Methods. Overall, 72 cases and 142 matched controls were interviewed between 2008 and 2019 at the first Clinic of Samarkand State Medical Institute. Clinical data for all cases were obtained from the central patients' database of 1st Clinic of Samarkand State Medical Institute. The diagnosis of SLE was based on the classification criteria of the American Rheumatism Association. Only those patients who met >4 criteria for SLE were included in the study. For each included case, we matched two controls for sex and age. Controls were randomly selected from the population screening database. Only those who provided informed consent were included in the

Results. Our results revealed a negative relationship between higher doses (>200 grams per week) of alcohol consumption and the SLE risk (see Table 1). The odds ratio was 0.49 for those with alcohol consumption of >200 g/week. There was also a greater risk of SLE among smokers compared to non-smokers (OR = 1.4, 95% CI 0.79-2.49). Those who smoke 2 to 5 cigarette packs per week had 2.64 times increased risk of SLE compared to non-smokers (OR = 2.64, 95% CI 0.97-7.18). However, none of the results on this exposure was statistically significant. Participants who reported alcohol and smoking exposure were males. Only participants with a body mass index (BMI) greater than 30 kg/m² tended to have a statistically significant greater risk of SLE when compared to those with BMI less than 18.5 kg/m² (OR = 2.88, 95% CI 1.17-7.07). However, we found no statistically significant dose-response relationship neither among smokers nor among those overweight and obese.

There was a statistically significant association between a family history of any autoimmune disease and an increased risk of SLE (OR 2.25, 95% CI 1.25-4.05). Especially those with a family history of SLE (OR 3.47, 95% CI 1.21-10) or rheumatoid arthritis (OR 2.7, 95% CI 1.04-7.02) tended to have a significantly greater risk of SLE.

People with diagnosed hypertension tended to have an increased risk of the development of SLE (OR 3.7, 95% CI 1.36-7.9). Also, cases were more likely to report angina pectoris compared to controls (OR 4.7, 95% CI 1.6-24). Among the infectious diseases, only pneumonia was borderline significantly associated with SLE (OR 1.9, 95% CI 1.0-3.7). A history of blood transfusion had a greater odds ratio (OR 1.8, 95% CI 0.8-3.6) while not statistically significant.

The hair colouring three or more times per year was not associated with a risk of SLE (OR 1.7, 95% CI 0.86-3.12) compared with less frequent exposure to hair colourants. The proportion of cases who reported occupational exposure to cold was significantly greater among cases rather than controls (32% and 12%

respectively, OR 3.44, 95% CI 1.21-9.5). The proportions with close contact with animals (cow, sheep or dog) were 61% of the cases and 39% of the controls (OR 2.31, 95% CI 0.78-6.3). There was a significant association between SLE and exposure to animals (cow) (OR 2.8, 95% CI 1.1-5.9).

For all four groups of life events classified according to reported by participants information, we observed no association with SLE. However, reported serious accidents tended to have a higher risk of SLE compared to other groups of events (OR 1.7, 95% CI 0.86-3.12).

Discussion. Our data shows that smoking is associated with an increased risk of SLE, although this did not reach statistical significance. Also, obtained results are consistent with previously published results [5-6]. Alcohol consumption has been suggested as a protective factor. This study suggests a dose-response relationship between alcohol consumption and SLE, which was even more pronounced in a multivariate model, which further strengthened the observation. Thus, our results are consistent with two previous studies that specifically addressed this issue. However, a study [2] indicated that data collection was performed from post-diagnostic exposure, which may distort the results and are not comparable with the evidence we obtained. Of course, our data may be and likely was influenced by recall bias, but concordance between our evidence and previous studies strongly suggests that the protective effect of alcohol may exist and smokers have a greater risk of developing SLE. We did not observe any indications of an association between hair dyes and SLE.

Conclusions. Our results do not support several etiological factors, including hair dyes and exposure to animals. This could be because too few subjects were investigated, which is the main drawback of this study. Furthermore, the recruitment rate was slightly higher in cases (82%) than in controls (69%), indicating to a possible selection bias. Another source of bias could be recalling the exposure. Cases would probably be more likely to make an

effort when filling in the questionnaire. Besides, defining disease onset may be complicated in a disease such as SLE. Notably, we did not find any indications that hormonal factors play any role as risk factors for SLE. However, we did find an indication of a link between animal exposure and the risk of SLE, with exposure to sheep. Negative life events did not show any evidence of a connection. As expected, the most obvious risk factor was a close relative with SLE, which was associated with two times the increased risk of SLE. This suggests that environmental and genetic data should be included in future studies.

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