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Ibragimov Khasan

Head of simulation training department, PhD

Samarkand State Medical University

**PATHOGENETIC RISK FACTORS OF SLE IN MODERN
RHEUMATOLOGY**

Abstract

The purpose of this research was to identify potential contributors to systemic lupus erythematosus. The 1st Clinic of Samarkand State Medical University investigated 142 healthy persons and 72 patients with systemic lupus erythematosus between 2008 and 2019. Having a history of high blood pressure raises the likelihood of getting SLE. In this group, patients reported angina pectoris at a higher rate than in the control group. An elevated risk of systemic lupus erythematosus (SLE) was discovered to be statistically associated with a family history of any autoimmune illness. There was no statistically significant difference between non-smokers and those who smoked 2–5 packs of cigarettes weekly, however the risk was 2.64 times greater for heavy smokers.

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

Ибрагимов Хасан

Заведующий кафедрой симуляционного обучения, PhD

Самаркандский государственный медицинский университет

**ПАТОГЕНЕТИЧЕСКИЕ ФАКТОРЫ РИСКА СКВ В СОВРЕМЕННОЙ
РЕВМАТОЛОГИИ**

Целью данного исследования было выявление потенциальных факторов, способствующих развитию системной красной волчанки. В 1-й клинике Самаркандского государственного медицинского университета в период с 2008 по 2019 год было проведено обследование 142 здоровых лиц и 72 пациентов с системной красной волчанкой. Наличие в анамнезе повышенного артериального давления повышает вероятность развития

СКВ. В этой группе пациенты чаще сообщали о стенокардии, чем в контрольной группе. Было обнаружено, что повышенный риск СКВ статистически связан с семейным анамнезом любого аутоиммунного заболевания. Не было статистически значимой разницы между некурящими и теми, кто выкуривал 2–5 пачек сигарет в неделю, однако риск был в 2,64 раза выше для заядлых курильщиков.

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

Introduction. The skin, joints, kidneys, brain, serosal surfaces, blood vessels, blood cells, lungs, and heart are all affected in systemic lupus erythematosus (SLE), an autoimmune illness that affects several systems [1,3]. Although hormones and genes play a major role, other risk factors, such as various forms of environmental exposure, may be just as essential in the development of SLE. Recent studies have shown that a combination of several environmental variables might trigger SLE in someone who is genetically predisposed to it [2,4]. Some medications that include aromatic amines have been suggested to potentially cause SLE [5-7]. Tobacco smoke and hair colors are only two examples of the many environmental agents that have been the subject of much research due to their chemical components, particularly aromatic amines. At the 1st Clinic of Samarkand State Medical Institute, we conducted a case-control research to look for possible causes of SLE.

Materials and Methods. The first Clinic of Samarkand State Medical Institute interviewed 72 patients and 142 matched controls between 2008 and 2019. All cases' clinical data were retrieved from the 1st Clinic of Samarkand State Medical Institute's central patients' database. According to the American Rheumatism Association's criteria, SLE was diagnosed. Patients were only included in the trial if they satisfied four or more criteria for SLE. We used a sex-and age-matched control pair for every instance that was considered. The population screening database was used to randomly identify the controls.

Participants were excluded from the study unless they gave their informed permission.

Results. According to our findings, the risk of SLE decreased as alcohol intake increased (>200 grams per week) (see Table 1). For those who consumed more than 200 grams of alcohol per week, the odds ratio was 0.49. To add insult to injury, smokers had a higher risk of SLE than non-smokers (OR=1.4, 95% CI 0.79-2.49). Compared to non-smokers, those who smoked 2–5 packs of cigarettes per week had a 2.64-fold higher risk of SLE (OR = 2.64, 95% CI 0.97–7.18). There were no statistically significant results, nevertheless, from this exposure. Men were more likely than females to report having smoked cigarettes and consumed alcohol. A statistically significant increased risk of SLE was seen only among participants with a BMI higher than 30 kg/m² compared to those with a BMI lower than 18.5 kg/m² (OR = 2.88, 95% CI 1.17-7.07). Neither smokers nor the overweight and obese showed a dose-response association that was statistically significant.

A higher risk of systemic lupus erythematosus was associated with a history of any autoimmune illness in the family, according to statistical analysis (OR 2.25, 95% CI 1.25-4.05). A much higher risk of SLE was seen in those who had a history of rheumatoid arthritis (OR2.7, 95% CI1.04-7.02) or SLE in their family (OR3.47, 95% CI1.21-10) relative to SLE.

An elevated risk of SLE development was seen in those with a history of hypertension (OR 3.7, 95% CI 1.36-7.9). Angina pectoris was also more commonly reported by cases than controls (OR4.7, 95% CI 1.6-24). Only pneumonia showed a borderline significant association with SLE among infectious illnesses (OR 1.9, 95% CI 1.0-3.7). Even though it wasn't statistically significant, having a history of blood transfusions increased the odds ratio (OR 1.8, 95% CI 0.8-3.6).

When comparing those who dyed their hair less frequently to those who dyed it three or more times a year, there was no association between the former

and SLE (OR1.7, 95% CI 0.86-3.12). Cases were more likely than controls to report having been exposed to cold temperatures on the job (32% vs. 12%, OR3.44, 95% CI 1.21-9.5). A total of 61% of cases and 39% of controls had close contact with animals (cow, sheep, or dog) (OR 2.31, 95% CI 0.78-6.3). Exposure to animals, namely cows, was significantly associated with SLE (OR2.8, 95% CI 1.1-5.9).

Across all four categories of life experiences, as defined by participant reports, we did not find any correlation with SLE. But compared to other categories of incidents, the risk of SLE was greater for those involving reported major accidents (OR1.7, 95% CI 0.86-3.12).

Discussion. Smoking increases the risk of SLE, according to our data, although the association did not achieve statistical significance. Additionally, the results that were obtained align with those that have been published before [5-6]. Some research has linked alcohol use to a reduced risk. An even stronger correlation between alcohol intake and SLE was found in a multivariate model, lending credence to the study's dose-response hypothesis. Hence, our findings are in line with two prior research that dealt with this matter head-on. Nevertheless, our findings is incongruous with that of a research [2] that collected data after the diagnosis, which might have skewed the results. Although our results might have been skewed by memory bias, they are consistent with other research suggesting that alcohol may have a protective effect and that smokers are more likely to acquire SLE. No evidence of a link between hair dyes and SLE was found by our researchers.

Conclusions. Several potential causes, such as hair dyes and animal contact, are not borne up by our findings. The primary limitation of this study is the small sample size, which may explain this finding. In addition, there was a potential selection bias as the recruitment rate was somewhat greater in the cases (82% vs. 69%) than in the controls (69%). Recalling the exposure is another potential cause of bias. Cases are more likely to put up an honest effort while

responding to the survey. In addition, a condition like SLE might make it difficult to pinpoint when symptoms first appear. It should be noted that we did not uncover any evidence that hormonal variables contribute to SLE risk factors. Nevertheless, we did discover some evidence that being among sheep increased the risk of SLE. There was no indication of a relationship between unfavorable life experiences. The most glaring risk factor was, unsurprisingly, having a close family with SLE; this was linked to a twofold greater risk of SLE. This provides more evidence that future research should use environmental and genetic data.

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