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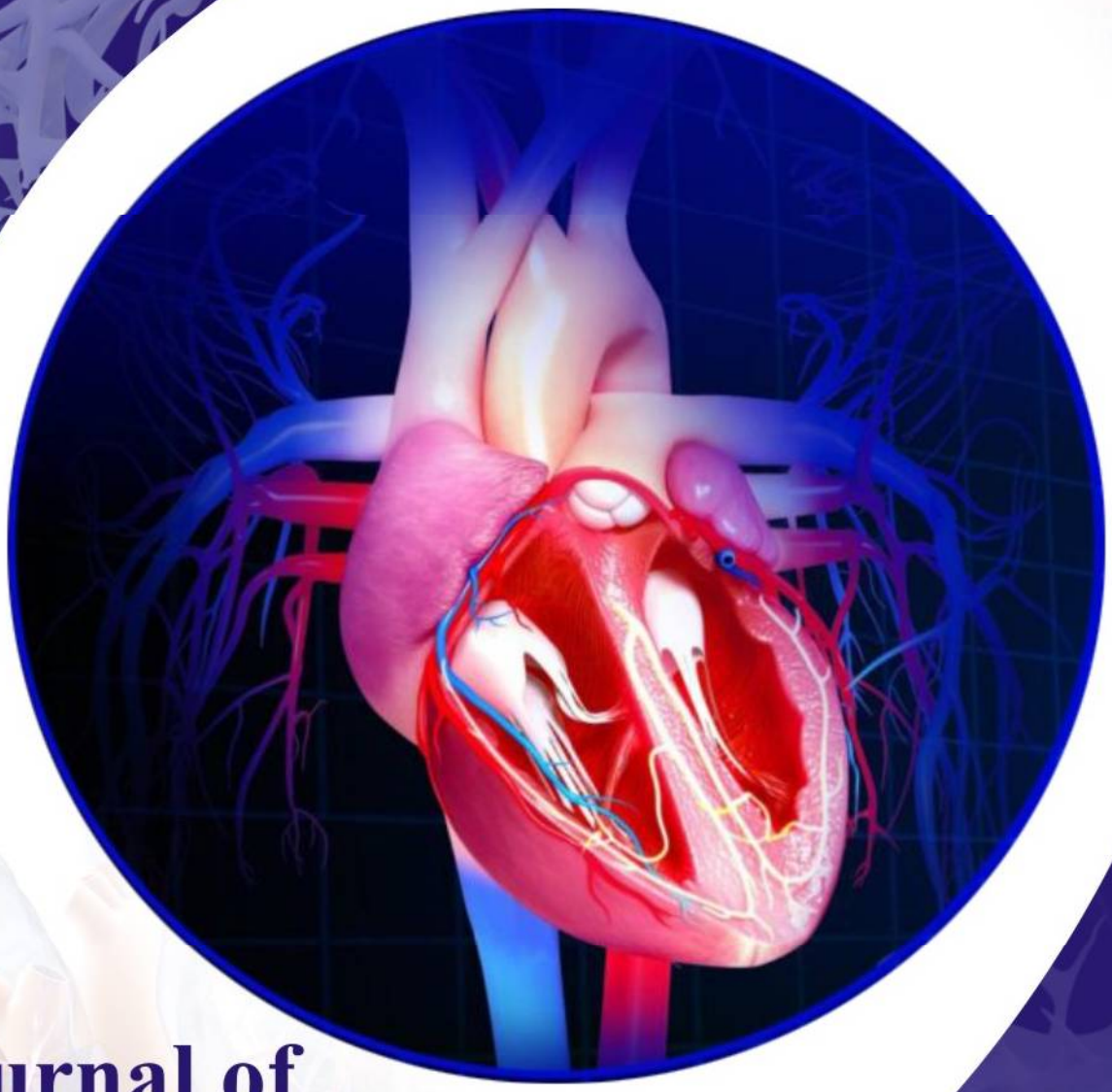

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АССОЦИАЦИЯ ТЕРАПЕВТОВ УЗБЕКИСТАНА

МИНИСТЕРСТВО ЗДРАВООХРАНЕНИЯ РЕСПУБЛИКИ УЗБЕКИСТАН

САМАРКАНДСКИЙ ГОСУДАРСТВЕННЫЙ МЕДИЦИНСКИЙ УНИВЕРСИТЕТ

ИННОВАЦИОННЫЕ ТЕХНОЛОГИИ В ЗДРАВООХРАНЕНИИ:
НОВЫЕ ВОЗМОЖНОСТИ ДЛЯ ВНУТРЕННЕЙ МЕДИЦИНЫ

МАТЕРИАЛЫ

международной научно-практической конференции
(Самарканд, 22 апреля 2022 г.)

Под редакцией
Ж.А. РИЗАЕВА

ТОМ I

Самарканд-2022

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Инновационные технологии в здравоохранении: новые возможности для внутренней медицины: Материалы международной научно-практической конференции (г. Самарканд, 22 апрель 2022 г.) / отв. ред. РИЗАЕВ Ж.А. - Самарканд: СамГМУ, 2022. – 736 с.

В сборнике собраны материалы, которые содержат статьи и тезисы докладов, представленных на международной научно-практической конференции «Инновационные технологии в здравоохранении: новые возможности для внутренней медицины», проведенной в СамГМУ 22 апрель 2022 г. Значительная часть материалов отражает современные проблемы внутренней медицины, посвященные поиску эффективных методов диагностики, лечения и профилактики заболеваний внутренних органов.

Представленные материалы будут интересны специалистам всех направлений внутренней медицины и широкому кругу читателей, интересующихся вопросами возникновения и профилактики основных заболеваний терапевтического профиля.

МИНИСТЕРСТВО ЗДРАВООХРАНЕНИЯ
РЕСПУБЛИКИ УЗБЕКИСТАН

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ПРЕДИКТОРЫ СИСТЕМНОЙ КРАСНОЙ ВОЛЧАНКИ: КЛИНИЧЕСКОЕ ИССЛЕДОВАНИЕ СЛУЧАЙ-КОНТРОЛЬ

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АННОТАЦИЯ

Системная красная волчанка (СКВ) — полисистемное аутоиммунное заболевание

Цель. Изучить факторы риска, которые предложено связывать с развитием СКВ.

Материал и методы. Всего в период с 2008 по 2019 год в первой клинике Самаркандского государственного медицинского института было опрошено 72 случая и 142 сопоставимых контроля. В исследование были включены только те пациенты, которые соответствовали >4 критериям СКВ. Была разработана анкета, состоящая из вопросов, связанных с факторами риска. Влияние переменных воздействия на СКВ измеряли с использованием отношения шансов (ОШ) и его 95% доверительного интервала (ДИ).

Результаты. При использовании многофакторной модели у пациентов с артериальной гипертензией в анамнезе, как правило, был повышенный риск развития СКВ (ОШ 3,7, 95% ДИ 1,36–7,9). Кроме того, пациенты с большей вероятностью сообщали о стенокардии, чем контрольная группа (ОШ 4,7, 95% ДИ 1,6–24). Выявлена статистически значимая связь между семейным анамнезом любого аутоиммунного заболевания и повышенным риском СКВ (ОШ 2,25, 95% ДИ 1,25–4,05). В то время как у тех, кто выкуривает от 2 до 5 пачек сигарет в неделю, риск СКВ был в 2,64 раза выше по сравнению с некурящими (ОШ = 2,64, 95% ДИ 0,97-7,18). Однако ни один из результатов этого воздействия не был статистически значимым.

Выводы. СКВ может быть связана как с эндогенными, так и с экзогенными факторами. Необходимы дополнительные хорошо спланированные исследования для изучения причинно-следственной связи между этими факторами и СКВ.

Ключевые слова: волчанка, СКВ, факторы риска, курение, алкоголь, стресс, семейный анамнез.

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PREDICTORS OF SYSTEMIC LUPUS ERYTHEMATOSUS: A CLINIC BASED CASE-CONTROL STUDY

ANNOTATION

Background. Systemic lupus erythematosus (SLE) is a multisystem autoimmune disease

Objective. To explore the risk factors proposed to be associated with the development of SLE.

Material and methods. In total 72 cases and 142 matched controls were interviewed between 2008 and 2019 at the first Clinic of Samarkand State Medical Institute. Only those patients who met >4 criteria for SLE were included in the study. The questionnaire consisted of questions related to risk factors were developed. The effect of the exposure variables on SLE was measured using the odds ratio (OR) and its 95% confidence interval (CI).

Results. Using a multivariate model, cases with a history of hypertension tended to have an increased risk of SLE development (OR 3.7, 95% CI 1.36-7.9). Also, cases were more likely to report angina pectoris than controls (OR 4.7, 95% CI 1.6-24). 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). While 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.

Conclusions. SLE may be associated both with endogenous and exogenous factors as well. Additional well-designed studies are needed to explore a cause-and-effect relationship between these factors and SLE.

Keywords: Lupus, SLE, risk factors, smoking, alcohol, stress, family history.

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TIZIMLI QIZIL YUGURIK XABARCHILARI: KLINIK TADQIQOT-NAZORAT

ANNOTATSIYA

Maqsad. SLE rivojlanishi bilan bog'liq bo'lgan xavf omillarini o'rganish.

Material va usullar. 2008-2019-yillar davomida Samarqand davlat tibbiyot institutining 1-klinikasida jami 72 ta holat va 142 ta mos keluvchi nazorat tekshiruvi o‘tkazildi. Tadqiqotga faqat SLE uchun > 4 mezonga javob beradigan bemorlar kiritilgan. Xavf omillari bilan bog'liq savollarning anketa aralashmasi ishlab chiqildi. EHM o'zgaruvchilarining SLEga ta'siri odds nisbati (OR) va uning 95% ishonch oralig'i (CI) yordamida o'lchandi.

Natijalar. Ko'p o'zgaruvchan modeldan foydalangan holda, gipertenziya tarixi bo'lgan hollarda SLE rivojlanish xavfi yuqori bo'lgan (OR 3,7, 95% CI 1,36-7,9). Bundan tashqari, holatlar stenokardiyani nazorat qilishdan ko'ra ko'proq xabar qilishdi (OR 4,7, 95% CI 1,6-24). Har qanday autoimmun kasallikning oilaviy tarixi va SLE xavfi ortishi o'rtasida statistik jihatdan muhim bog'liqlik mavjud edi (OR 2,25, 95% CI 1,25-4,05). Haftada 2-5 quti sigaret chekadiganlar chekmaydiganlarga nisbatan SLE xavfini 2,64 marta

oshiradi (OR = 2,64, 95% CI 0,97-7,18). Biroq, bu ta'sirga oid natijalarning hech biri statistik ahamiyatga ega emas edi.

Xulosa. SLE ham endogen, ham ekzogen omillar bilan bog'liq bo'lishi mumkin. Ushbu omillar va SLE o'rtasidagi sabab-ta'sir munosabatlarini o'rganish uchun qo'shimcha yaxshi ishlab chiqilgan tadqiqotlar talab qilinadi.

Kalit so'zlar: Lupus, SLE, xavf omillari, chekish, spirtli ichimliklar, stress, oilaviy tarix.

Introduction. Systemic lupus erythematosus (SLE) is a multisystem autoimmune disease characterized by numerous B cells producing hyperactive autoantibodies and involvement of skin, joints, kidneys, brain, serosal surfaces, blood vessels, blood cells, lungs and heart [1]. While genetic and hormonal factors are significantly important, other risk factors, including different environmental exposure, may have equal importance in the aetiology of SLE. According to recent research, many various environmental factors may cooperate to cause SLE in a genetically susceptible person [2]. It is hypothesized that some drugs containing aromatic amines have been proposed to cause SLE [3-4]. Therefore, numerous studies investigated environmental agents containing chemical components incredibly aromatic amines, such as tobacco smoke and hair dyes. The studies that have explored the etiological role of hair dyes in SLE development showed contradictory results [5-6], while smoking tobacco in many studies was associated with an increased risk of SLE [7-9]. In contrast, recent findings demonstrate that alcohol consumption is associated with a decreased risk of SLE [9], with some exceptions [10]. Some other studies suggest that hormone replacement therapy may be associated with an increased risk of SLE [11]. Infectious agents, mainly of viral origin, were also discussed as potential triggers of SLE for many years. [12]. The role of stressful adverse life events in the onset of autoimmune diseases is controversial [13]. 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 the SamMI. The diagnosis of SLE was based on American Rheumatism Association's classification criteria. 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 study.

Socioeconomic, demographic, and clinical factors were compared between cases and controls using the chi-square test for sex and socioeconomic status (categorical variables) and t-test for age (continuous variable). The proportion of SLE was compared between the entire study sample as well as in age, sex, and socioeconomic status subgroups to avoid confounding effects.

Questionnaire. The questionnaire consisted of questions related to education, body height and weight, hair dyes (frequency), smoking (number of cigarette packs per week), alcohol consumption (quantity), hormonal/endocrine factors (hormone replacement therapy), occupational exposure to low temperature, a family history of autoimmune diseases and drug allergy, any history of adverse psychological events (stress, depression etc.)

Data analysis. The effect of the exposure variables on SLE was measured using the odds ratio (OR) and its 95% confidence interval (CI). Estimations were performed by conditional logistic regression. In the multivariate analyses, we also tested whether effect modification was present by including relevant interaction terms in the models. For analyses, we used R studio version 3.6.2.

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 than 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 than 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.

Table 1. Conditional logistic regression results (BMI, alcohol, smoking)

Variables	Cases n (%)	Controls n (%)	Odds ratio (OR)	95% CI
Alcohol consumption (grams /week)				
No	32 (44.4)	48 (33.8)	Ref	Ref
>0-200	21 (29.2)	45 (31.7)	0.70	0.35-1.39
>200	19 (26.4)	49 (34.5)	0.49	0.25-0.97
Smoking (packs week)				
0	29 (40.3)	69 (48.6)	Ref	Ref
>0-2	22 (30.6)	45 (31.7)	1.16	0.6-2.27
>2-5	11 (15.3)	19 (13.4)	1.38	0.58-3.26
>5	10 (13.9)	9 (6)	2.64	0.97-7.18
BMI				
<18.5	19 (26.4)	51 (35.9)	1.0	Ref
18.5-24.9	21 (29.2)	48 (33.8)	1.17	0.56-2.45
24.9-29.9	17 (23.6)	29 (20.4)	1.57	0.71-3.49
>30	15 (20.8)	14 (9.8)	2.88	1.17-7.07

Family history of autoimmune diseases

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) (Table 2). 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.

Table 2. Distribution of autoimmune diseases among close relatives of cases and controls with corresponding unadjusted OR and 95% CI

Autoimmune diseases	Cases n (%)	Controls n (%)	OR	95% CI
Any autoimmune disease	35 (48.6)	42 (29.6)	2.25	1.25-4.05
Rheumatoid arthritis	10 (14)	10 (7)	2.7	1.04-7.02
SLE	9 (13)	7 (4.9)	3.47	1.21-10
Multiple sclerosis	4 (5.5)	3 (2.1)	3.6	0.77-16.9
Systemic sclerosis	2 (2.8)	3 (2.1)	1.8	0.29-11.2

Crohn's disease	3 (4.2)	6 (3.5)	1.35	0.32-5.68
Psoriasis	7 (9.7)	20 (14)	0.95	0.37-2.42
Ankylosing spondylitis	3 (4.2)	1 (0.7)	8.1	0.82-80.4
Diabetes Mellitus (I)	2 (2.8)	4 (2.8)	1.35	0.24-7.69

Comorbidities. 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). Previous blood transfusion had a higher odds ratio (OR 1.8, 95% CI 0.8-3.6) while not statistically significant (see Table 3).

Table 3. Distribution of comorbidities among cases and controls.

Disease	Cases n (%)	Controls n (%)	OR	95% CI
Immunological diseases				
Asthma	2 (2.8)	8 (5.6)	0.7	0.1-3.1
Multiple sclerosis	2 (2.8)	2(1.4)	3.2	0.8-5.2
Crohn's disease	1 (1.4)	2(1.4)	1.6	0.2-12.1
Psoriasis	3 (4.2)	9 (6.3)	0.7	0.13-2.72
Cardio-vascular diseases				
Myocardial infarction	5 (6.9)	2 (1.4)	2.5	0.43-18
Angina pectoris	6 (8.3)	4 (2.8)	4.7	1.6-24
Stroke	4 (5.5)	2 (1.4)	2.9	0.61-12
Hypertension	32 (44.4)	19 (13.4)	3.7	1.36-7.9
Surgery				
Any surgery	11 (15.3)	20 (10)	1.4	0.6-3.1
Blood transfusion	18 (25)	27 (19)	1.6	0.8-3.6
Infectious diseases				
Herpes zoster	5 (6.9)	12 (8.4)	1.1	0.4-2.9
Pneumonia	21 (29.2)	20.4 (14)	1.7	1.0-3.7
Pyelonephritis	8 (11.1)	15 (10.6)	1.3	0.5-3.2

Table 4. Distribution of reported life events among cases and controls

Reported stressors/events	Cases n (%)	Controls n (%)	OR	95% CI
Family (death, divorce, etc.)	14 (11)	22(11)	1.3	0.5-3.6
Financial (any)	3 (1.2)	8 (3.9)	0.4	0.2-3.5
Conflicts (any)	5 (3.7)	13 (6.4)	0.7	0.6-2.6

Accidents (serious)	13(8.5)	16 (7.9)	1.8	0.7-5.7

Other variables (hair dyes, occupational exposure to cold). 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 more significant 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 cows (OR 2.8, 95% CI 1.1-5.9).

Life events. 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. The current point of view on the aetiology of SLE is that several environmental factors act on a genetically predisposed individual to develop or defend against disease. The results of this study suggest that hypertension, a family history of autoimmune diseases are risk factors for SLE, and alcohol is a potential protective factor, however, the latter is based on weak evidence. Our data suggest that smoking has an association with an increased risk of SLE, although this did not reach statistical significance. Our results are consistent with previously published results [5-6]. Alcohol consumption has been suggested as a protective factor. In this study, we also observed 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. Of course, our data may be and likely was influenced by recall bias, but concordance between our evidence and prior 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. Among the reported comorbidities investigated, hypertension was associated with a significantly increased risk of SLE. It is also possible that vascular re-modelling, damage and dysfunction of endothelial cells, which contributes to hypertension in SLE, could be a primary event preceding clinical SLE diagnosis [11-13].

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. Notably, we did not find any indications that hormonal factors play any role as risk factors for SLE. Adverse life events did not show any evidence of a connection. As expected, the most prominent 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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