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THE RELATIONSHIP BETWEEN SMOKING HABITS AND THE INCIDENCE OF CHRONIC URTICARIA IN CIVITAS WIDYA MANDALA CATHOLIC UNIVERSITY SURABAYA

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ABSTRACT

Introduction: Urticaria is a heterogeneous disease with multiple causative factors. A thorough literature review revealed that there is very limited evidence on the influence of smoking and alcohol consumption habits on the clinical features of urticaria.. Atopic allergic which mean urticaria can affect all populations, but is most common in those aged between 20 to >60 years. Smoking has been reported to play a role in the onset of Urticaria. Substances in cigarette smoke can interact with body cells, resulting in the formation of free radicals or Reactive Oxygen Species (ROS). **Objective:** To see the relationship between smoking habit and the incidence of chronic urticaria among civitas at Widya Mandala Surabaya. **Method:** The method used in this study was a case-control study by taking a population of civitas at the Widya Mandala Surabaya who had smoking history from 2020 to 2023. A sample of 94 people was obtained who had met the inclusion and exclusion criteria of the study. The sample of this study was divided into two groups consisting of a case group and a control group. **Result:** The results of the Contigency Coefficient Correlation test showed a significant p value of 0.031 <0.05, which means that there is an important correlation between smoking and the incidence of chronic urticaria among civitas at the Widya Mandala Surabaya. **Conclusion:** It is proven that there is a relationship between smoking and the incidence of chronic urticaria in civitas of Widya Mandala Surabaya.

Keywords: Smoking habit, chronic urticaria, civitas university

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INTRODUCTION

Urticaria is a common and heterogeneous inflammatory skin disease with a lifetime prevalence of up to 20% worldwide[1,2]. Urticaria is initially classified based on its duration as acute urticaria (AU) or chronic urticaria (CU) within 6 weeks.[3] Urticaria is also divided into induced and spontaneous urticaria. In induced urticaria, signs and symptoms are caused by specific triggers that are subtype-specific, for example, cold in cold urticaria (ColdU).[3–5]

The disease is the result of activation and degranulation of cutaneous mast cells, followed by release of histamine and other mediators, which leads to sensory nerve activation, vasodilation, plasma extravasation, and cell recruitment.[3,6] This process leads to the development of disease-defining signs and symptoms, pruritic urticaria and/or angioedema.

In spontaneous urticaria, signs and symptoms occur without warning and there is no obvious trigger, although in some patients, stress, infections, and other exacerbations may increase disease activity. Spontaneous urticaria is more common than provoked urticaria, and both may occur simultaneously in the same patient.[3–5]

The prevalence of AU is highest in children over the age of 30 years.[5,7–11] Adult patients with CSU are older than adult patients; in CIndU (mean age ~30-70 vs. ~20-40 years) and at an older age at onset (~30-50 vs.~20-35 years).

In adults, all types of urticaria are more common in women than in men, except for cholinergic urticaria (CholU), which is more common in both adult men and children. In younger children, the female predominance is absent or less pronounced. [11,44]

The most common types of CIndU are symptomatic dermatomyelia, CholU and ColdU in both adults and children.[11,12] On the other hand, aquagenic urticaria, solar urticaria, heat urticaria, vibratory angioedema, and contact urticaria are rare.[13-18]

Compounds found in cigarette smoke have the ability to connect with cells in the body, resulting in the formation of free radicals or Reactive Oxygen Species (ROS). As a result of the continuous accumulation of ROS, the phenomenon of oxidative stress or an imbalance between pro-oxidants and antioxidants occurs. Furthermore, ROS will interact with lipids or often referred to as the lipid peroxidation process which will lead to a further increase in the inflammatory process in the pathogenesis of Urticaria especially chronic urticaria.[3]

From the research conducted previously, differences in research results

were obtained. Therefore, we would like to conduct another study on the relationship between these two variables.

METHOD

The research method applied in this study is analytical observational using a case- control study design approach with purposive sampling technique. The population taken in this study, namely all male students of the Widya Mandala Surabaya Faculty of Medicine, class of 2020 to 2023.

The data collection procedure in this study was carried out by distributing a preliminary questionnaire in the form of a sheet containing inclusion and exclusion criteria in order to screen the population from other etiological factors that could affect the incidence of chronic urticaria. After that, respondents were asked to fill out a follow- up questionnaire regarding their smoking history. Furthermore, the level of CU incidence was assessed by taking photos using a smartphone camera on three sides of the face, namely the front, left oblique, and right oblique, then the photos were initialed and given to a skin and genital specialist to group the clinical diagnosis of CU. The collected data were then analyzed using the Coefficient of Contingency Correlation test with a significance value of $p<0.05$.

RESULT

Table 1. Number of Smoking Habits in the Sample

Smoking Habits	Total	Percent
Non-smoking	7%	7,4%
Light smokers	0%	0%
Moderate smokers	61	64.9%
Heavy smokers	26	27,7%
Total	94	100%

Table 1 shows the number of smoking habits of the sample consisting of non-smoking, light smokers, moderate smokers. and heavy smokers. The results of data collection showed that 64.9% of the samples did not smoke, 27.7% of the samples were light smokers, 7.4% of the samples were moderate smokers, and there were no heavy smokers.

Table 2. Number of Chronic Urticaria Occurrences in Samples

Incidence of Chronic Urticaria (CU)	Total	Percent
Non-CU	47	50%
CU	47	50%
Total	95	100%

Table 2 shows the number of CU events in this study divided into control and case groups. In this study, 50% of the samples belonged to the control group and the other 50% of the samples belonged to the case group.

Table 3. Distribution of Respondent by Backpack Weight

Age	Non-CU	CU	Total
20-30	12 (12.8%)	7 (7.4%)	19
31-40	11 (11.7%)	13 (13.8%)	24
41-50	15 (16.0%)	16 (17.0%)	31
51-60	8 (8.5%)	10 (10.6%)	18
61-70	1 (1.1%)	1 (1.1%)	2
Total	47	47	94

Table 3 shows 19 respondents that 20-30-year-olds diagnosed with non-CU and those diagnosed with CU were 12,8% and 7,4%. Furthermore, 24 respondents that 31-40-year-olds diagnosed with non-CU were 11.7%, and those with CU were 13.8%.

Furthermore, 31 respondents that 41-50-year-olds diagnosed with non-CU were 16%, and those with CU were 17%.

Furthermore, 18 respondents that 51-60 years old diagnosed with non-CU were 8.5%, and those with CU were 10.6%. And finally, 2 respondents that 61–70 years old diagnosed with non-CU and those with CU were both 1.1%.

Table 4. Distribution of Respondent by Shoulder Pain Severity

Smoking Habits	Non-CU	CU	Total
Non-smoking	36 (38.3%)	25 (26.6%)	61
Light smokers	10 (10.6%)	16 (17.0%)	26
Moderate smokers	1 (1.1%)	6 (6.4%)	7
Heavy smokers	0 (0%)	0 (0%)	0
Total	47	47	94

Table 4 Shows the number of respondents who did not smoke and were diagnosed with non-CU was 38.3%, while those with CU were 26.6%. Furthermore, the number of respondents classified as light smokers and diagnosed with non-CU was 10.6%, while CU was 17%. Furthermore, the number of respondents classified as moderate smokers and diagnosed as non-CU was 1.1%, while CU was 6.4%. And finally, there were no samples included in the heavy smoker group.

Table 5. Correlation Test of Contigency Coefficient of Smoking Habit with Chronic Urticaria Incidence

	Value	Approximate Significance
Contingency Coefficient	.262	.031
N of Valid Cases		94

Table 5 shows that the results of the Coefficient of Contigency Correlation test get a p value of 0.031 <0.05, which means H0 is rejected and H1 is accepted. The table

above also shows that the contigency coefficient (C) value was found to be 0.262, which means that there is a weak correlation value between the two variables in this study.

DISCUSSION

Urticaria is a common skin disorder characterized by erythematous, itchy urticarial patches. The exact pathogenesis of urticaria is not yet fully understood. Several theories have been put forward to explain the underlying mechanisms of urticaria development. Although there is still strong evidence about the final events, i.e., release of histamine by mast cells and basophils, what factors cause these cells to degranulate and release various inflammatory mediators, this remains to be elucidated.

In recent years, it has been demonstrated that various biological systems, such as autoimmunity, autoallergy, inflammation, and coagulation, may interact to produce a common underlying mechanism that leads to the symptoms of urticaria. In addition to histamine, the main mediator, tryptase, chymase, proteases, prostaglandin D2, thromboxanes, leukotrienes, and platelet-activating factor are involved in the development of urticaria. [19-24]

The effect of cigarette smoke on mast cell degranulation is still unclear. Few studies have examined the effect of cigarette smoke on mast cell mediators. They showed that cigarette smoke extract increased histamine- and lipopolysaccharide-induced cyclooxygenase-2 (COX-2) expression in endothelial cells, suggesting that cigarette smoke and mast cell mediators may simultaneously enhance inflammatory responses in the vascular wall.[25,26] They further hypothesize that, because cigarette smoke is associated with upregulation of COX-2 and TLR4 expression and mast cell activation, cigarette smoke enhances histamine-mediated upregulation of Toll-like receptor 2 (TLR2)/TLR4 signaling in the endothelium, promoting the progression of atherosclerosis. Smoking is also associated with mast cell activation, as the absolute number of mast cells in the lungs and skin of smokers increases.[27] On the other hand, some studies suggest an inhibitory effect of cigarette smoke on mast cells.[25] In 2013, Givi et al. demonstrated that cigarette smoke medium suppresses the granularity and surface expression of c-kit and the high affinity receptor for IgE (FcεRI) on mast cells.[25]

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cell activation, as the absolute number of mast cells is increased in the lungs and skin of smokers.[27] Meanwhile, some studies have shown an inhibitory effect of cigarette smoke on mast cells.[25] Zibi et al. demonstrated that cigarette smoke medium suppresses the granularity and surface expression of c-kit and high affinity receptor for IgE (FcεRI) on mast cells.[25]

The aim of this study was to investigate the effect of cigarette smoke on mast cell maturation and function. Mast cells have previously shown an inhibitory effect of cigarette smoke medium on bone marrow mast cell degranulation. Although cigarette smoke medium is known to stimulate multiple inflammatory cells via TLR4, the inhibitory effect of cigarette smoke on mast cells is thought to be independent of TLR4 signaling. This study demonstrates the inhibitory development of mast cells and the resulting response to allergic activation by cigarette smoke.[25]

The literature on the association between smoking and urticaria is very limited. Relationship of allergic rhinitis, eczema, and urticaria to sex, age, smoking habits, occupational air exposure, and respiratory symptoms.

However, this study mainly focused on the prevalence of the disease, and the results did not provide meaningful data on the association between smoking and urticaria. The aim of this study was to specifically investigate the epidemiological characteristics of chronic urticaria. Patients were asked about their smoking and drinking habits. Although the results did not provide evidence of the effect of alcohol intake on urticaria, it has been proven that smoking is associated with a significant decrease in the risk of chronic urticaria.[29,30] To our knowledge, our study is the third to investigate smoking habits and the second study to examine the alcohol intake habits of patients with urticaria.[29]

However, of the available evidence, only the suppressive effect of smoking reported by Lapi et al.[27] We demonstrated that smoking increases the risk of urticaria, especially in patients who do not respond to low-dose antihistamines. This finding may explain the role of smoking in non-histamine-associated urticaria but not in histamine-associated urticaria. Urticaria is a highly complex disease with a complex network of interactions between host and environmental factors in the pathogenesis.

Histamine, the main mediator, is primarily responsible for the typical lesions of urticaria. However, the wide variety of clinical syndromes and the presence of antihistamines suggest that other molecules, such as prostaglandins, leukotrienes, cytokines, and chemokines, are also involved in the progression of the disease.[19–24,31]

CONCLUSIONS

In this study, the p value was 0.031 <0.05, which means that is an important correlation between smoking habits and the incidence of chronic urticaria among civitas at the Widya Mandala Surabaya.

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