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Allergy as Predisposing Factors of Recurrent Aphthous Stomatitis

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ABSTRACT

Allergy is an abnormality or alteration in the immune system's response to foreign proteins. The clinical presentation of allergic reactions can vary widely, including recurrent aphthous stomatitis in the oral cavity. Therefore, this study aimed to investigate the prevalence of allergy as a potential etiology of recurrent aphthous stomatitis. For the assessment, a descriptive design was used, comprising 50 patients diagnosed with minor-type recurrent aphthous stomatitis who met the inclusion and exclusion criteria. Diagnosis of recurrent aphthous stomatitis was established through the patient's history and clinical examination. Subsequently, blood samples were taken to analyze the levels of IgE and eosinophils. This was followed by a computer-based analysis conducted to assess the prevalence of allergy history along with the levels of IgE and eosinophils. The results showed that 14 patients (28%) had a history of allergies, while laboratory examinations identified 29 patients (58%) with elevated IgE levels, and 13 patients (26%) had high eosinophil levels. This suggested that allergy could play a role in the predisposition of recurrent aphthous stomatitis. Consequently, allergy assessments were recommended for patients with recurrent aphthous stomatitis, using either allergy history or laboratory tests.

Keywords: Allergy, Recurrent Aphthous Stomatitis, Allergy History, IgE, Eosinophil

ABSTRAK

Alergi merupakan kelainan atau perubahan respon sistem imun terhadap protein asing. Secara klinis, reaksi alergi sangat bervariasi. Salah satu manifestasi alergi pada rongga mulut dapat berupa stomatitis aftosa rekuren. Penelitian ini bertujuan untuk mengetahui prevalensi alergi sebagai etiologi stomatitis aftosa rekuren. Alergi diperiksa melalui riwayat alergi pasien dan kadar IgE serta eosinofil. Penelitian ini merupakan penelitian deskriptif. Lima puluh pasien stomatitis aftosa rekuren tipe minor berpartisipasi dalam penelitian ini. Pasien yang dijadikan subjek pada penelitian ini tidak menderita penyakit sistemik, tidak sedang mengonsumsi obat, tidak menggunakan alat ortodontik, dan bersedia menjadi subjek penelitian. Diagnosis stomatitis aftosa rekuren ditegakkan berdasarkan anamnesis dan pemeriksaan klinis. Kadar IgE dan kadar eosinofil dianalisis dengan darah yang diambil dari pasien. Analisis komputer dilakukan untuk menganalisis prevalensi riwayat alergi dan kadar IgE dan eosinofil. Penelitian ini menunjukkan bahwa 14 (28%) pasien mempunyai riwayat alergi. Hasil pemeriksaan laboratorium menunjukkan 29 (58%) pasien mempunyai kadar IgE tinggi, dan hanya 13 (26%) pasien yang mempunyai kadar eosinofil tinggi. Penelitian ini menunjukkan bahwa alergi dapat dianggap sebagai etiologi stomatitis aftosa rekuren. Hal ini dapat dilakukan dengan melakukan pemeriksaan alergi pada pasien stomatitis aftosa rekuren melalui riwayat alergi atau pemeriksaan laboratorium.

Keyword: Alergi, Stomatitis Aftosa Rekuren, Riwayat Alergi, IgE, Eosinofil

1. Introduction

The occurrence of allergy is attributed to the abnormal response of the immune system to foreign proteins. Allergic reactions are triggered by allergens entering the body through inhalation, ingestion, injection, or direct contact with the skin, mucous membranes in the mouth, or eyes [1]. Moreover, these reactions can present with a range of symptoms, varying from mild to severe intensity [2]. When allergic reactions happen, the production of antibodies increases, which is known as immunoglobulin E (IgE). The antibodies bind to specific receptors on mast cells, causing the activation of cells and the release of inflammatory mediators such as leukotrienes, histamine, and eosinophil cationic protein (ECP) from activated eosinophils. This process triggers a lot of allergic symptoms, affecting both the skin and the mucosal surfaces, including the oral mucosa [3,4].

Clinically, the clinical features of an allergic reaction vary greatly, ranging from rashes on the skin in the form of macules, papules, vesicles, swelling, and rashes on the oral mucosa in the form of perioral dermatitis, erythema, ulceration, lichenoid reactions, gingivitis, and cheilitis [1, 3]. Allergic reactions in the oral cavity include recurrent aphthous stomatitis, lichenoid reactions, Behçet's disease, Reiter's disease, sarcoidosis, and Wegener's granulomatosis [4,5]. Among these disorders, recurrent aphthous stomatitis is the most common oral condition characterized by recurring painful sores on the non-keratinized mucosa, with a prevalence of 5-66% at an average occurrence rate of 20% [6].

A history of exposure to allergen is needed for the diagnosis of oral allergy, associated clinical signs and symptoms, including documentation of disease's progression after elimination. Various tests also help the identification of the process [5] such as examining biomarkers related to allergy, including evaluating IgE and ECP levels. Previous studies have shown that the levels of IgE and ECP rise during allergic reaction, serving as potential biomarkers for allergy [4].

Based on the background above, this study aimed to investigate the prevalence of allergic reactions as a potential predisposing factor of recurrent aphthous stomatitis. To evaluate the presence of allergies, patients underwent a detailed analysis of their allergy history, measurement of IgE levels, and measurement of eosinophil levels.

2. Materials and Methods

This descriptive study was conducted at the Oral Medicine Installation of Universitas Sumatera Utara Dental Hospital and Prodia Clinical Laboratory in Medan, using a cross-sectional method. Specifically, diagnosis of recurrent aphthous stomatitis and comprehensive review of medical history were performed at the Oral Medicine Installation, while the levels of IgE and eosinophil tests were conducted at Prodia Clinical Laboratory.

A total of 50 patients diagnosed with recurrent aphthous stomatitis were selected through a non-probability purposive sampling. Each patient who met the established criteria was included in the study. The inclusion criteria required that participants had a minor form of recurrent aphthous stomatitis, must not have had systemic disease or taken any medication, and must not have had orthodontic appliances. They also showed a willingness to participate. Subsequently, a comprehensive review of the medical history and a clinical examination were required for the diagnosis of recurrent aphthous stomatitis. A thorough anamnesis was conducted to assess the frequency of recurrent episodes and obtain information regarding any previous allergy of patient. This was followed by clinical evaluation to assess the ulcers associated with recurrent aphthous stomatitis. Blood samples were collected from all patients to measure levels of IgE and eosinophils, which were then analyzed by specialized laboratory professionals.

The information obtained was examined and evaluated to determine the prevalence of allergy history and the levels of IgE as well as eosinophils. This study followed ethical guidelines from national and international sources and received approval from the Research Ethics Committee of Universitas Sumatera Utara, No. 730/KEP/US/2021. Informed consent was obtained from each patient, ensuring full awareness of the procedures and implications included in their treatment.

3. Results

This study included a group of 50 patients who were diagnosed with recurrent aphthous stomatitis. The results showed that a significant majority, 37 patients (74%), were in the late adolescent age group, comprising ages 17 to 25 years. A total of five patients (10%) were categorized as early adolescents (ages 12-16). Additionally, four patients (8%) fell into both the early adulthood (ages 26-35) and late adulthood (ages 36-45) age groups. The study also showed a predominance of female patients, with 35 females (70%) compared to 15 males (30%). Table 1 shows an overview of how patients are distributed according to their age and sex.

Table 1. Age and sex distribution of patients.

Age	Female		Male		Total	
	N	%	N	%	N	%
12 – 16 years	3	6	2	4	5	10
17 – 25 years	26	52	11	22	37	74
26 – 35 years	2	4	2	4	4	8
36 – 45 years	4	8	0	0	4	8
Total	35	70	15	30	50	100

Table 2 shows that 14 patients (28%) in the total study group have reported a prior history of allergy. In comparison, a significantly larger group of 36 patients (72%) do not have any observable history of allergic conditions.

Table 2. Prevalence of allergy history in recurrent aphthous stomatitis patients.

Allergy History	Frequency (n)	Percent (%)
Yes	14	28
No	36	72
Total	50	100

Table 3 shows intriguing results regarding the prevalence of IgE levels among patients suffering from recurrent aphthous stomatitis. The data showed that 29 (58%) out of 50 patients had elevated IgE levels.

Table 3. Prevalence of IgE levels in recurrent aphthous stomatitis patients.

IgE level	Frequency (n)	Percent (%)
Normal	21	42
High	29	58
Total	50	100

Table 4 shows eosinophil level in individuals with recurrent aphthous stomatitis. The results showed that a mere 13 (40%) patients had elevated eosinophil levels.

Table 4. Prevalence of eosinophil levels in recurrent aphthous stomatitis patients.

Eosinophil level	Frequency (n)	Percent (%)
Low	17	34
Normal	20	40
High	13	26
Total	50	100

4. Discussion

Recurrent aphthous stomatitis is identified by the occurrence of repeated ulcers in the mouth's soft tissue without any additional signs of disease [7], which typically appears during childhood or adolescence. This condition is commonly found in people during their 20s, and recurrent tends to diminish as age increases [8]. Approximately 80% of people with recurrent aphthous stomatitis initially developed this condition before age 30 [9]. In this study, the highest age group of patients with recurrent aphthous stomatitis was in late adolescence (17-25 years), comprising 37 (74%). Similarly, Xu et al. [10] showed that 73% of patients with recurrent stomatitis were within the second decade of life (18-29 years). Rajmane et al. [11] in the study about recurrent aphthous stomatitis prevalence, also showed that the age group of 20–29 years (56.9%) was mainly affected and decreased with age. Regarding age, recurrent aphthous stomatitis prevalence showed a pattern that was lower in the first decade but peaked in the second and third before a lower prevalence [12].

Related to sex, recurrent aphthous stomatitis is common among females [13]. This study showed more females (70%) with recurrent aphthous stomatitis. Kounoupis et al. [14] reported that the highest recurrent aphthous stomatitis incidence in their study was found in females with 36 patients (60%). Diaz et al. [15] also found more frequent distribution among female patients (67.4%). This high prevalence could be attributed to hormonal imbalance, such as menstruation. Recurrent aphthous stomatitis is primarily associated with the secretory phase of menstruation, the luteal phase, and decreases in its incidence throughout gestation [16]. The high occurrence among females can also be attributed to females seeking medical care more frequently [17].

Allergy is hypersensitivity reaction initiated by suspected immunologic mechanisms that are proven, either IgE-mediated or non-IgE-mediated [18]. These reactions can be observed as a vesicle, papule, patch, ulceration, or swelling in a specific area. The symptoms experienced will vary depending on which part of the body is affected [1,2]. Allergy mechanisms in the skin and mucosa are not different. Generally, there are four types of allergic responses, namely I, II, III, and IV [3]. The most frequent form is type I hypersensitivity, which is distinguished by the release of IgE by plasma cells. Type II hypersensitivity reactions occur infrequently and typically require 2 to 24 hours to manifest. This allergy form causes the process of recognizing and targeting particles for phagocytosis. It also leads to red blood cells clumping (agglutination), breaking down the cell membrane, and ending with cell death. The pathogenesis of type III hypersensitivity reactions includes IgG and IgM antibodies binding with soluble proteins, leading to the formation of immune complexes that can manifest in tissues. The process can trigger complement activation, inflammation, the flow of neutrophils, and the release of contents from mast cells (degranulation). This hypersensitivity occurs within a few days or possibly even weeks and is usually treated using anti-inflammatory drugs and corticosteroids. Type IV hypersensitivity reactions are cell-mediated and not dependent on antibodies. As the second most prevalent form, type IV hypersensitivity reaction requires at least 48 hours to manifest. This type of allergic reaction is the most frequently observed in the mouth, but type I is occasionally included, with types II and III showing possible tendency [3,19,20].

There is a wide range of clinical manifestations of oral and perioral diseases that may be linked to allergy, including conditions like lichenoid reactions, cheilitis, perioral dermatitis, contact stomatitis, recurrent aphthous stomatitis, etc. [1,21]. The exact cause of recurrent aphthous stomatitis is unclear, and the pathogenesis is unclear. However, it is known that allergy has a significant impact on the occurrence of recurrent aphthous stomatitis, suggesting the potential benefits of evaluation [4]. The diagnostic procedure of allergy is categorized into clinical and laboratory. The clinical diagnostic procedure includes a skin prick test, biomarker testing (IgE, eosinophil levels in blood or sputum, the fractional exhaled nitric oxide (FeNO) level, level of serum interleukin (IL)-5, and level of (IL)-13), and molecular allergy testing. Among the group of clinical methods, the clinical history should include specific information regarding the progression of clinical characteristics, symptoms, the time between contact with allergen and the onset of the signs, as well as the order in which symptoms manifest [22-24]. In this study, anamnesis was performed to evaluate allergy history, and the results show that 14 patients (28%) have allergy history. These results were in line with Rodríguez-Archilla et al. (2018), where among 200 patients with recurrent aphthous stomatitis patients, only 17% had allergy history [25]. Similarly, Almoznino [5] reported that eight patients (16.3%) had allergy history, and some showed multiple allergens.

Laboratory diagnostic of allergy was conducted by examining IgE and eosinophil levels. IgE is one of the five classes of immunoglobulins in the body, with a significant role in causing allergy. After initial contact, individuals with allergy become sensitized by generating allergen-specific IgE. This binds to receptors on mast cells in tissues and basophils in circulation, causing acute allergy symptoms [1, 2], which is high in 29 (58%) patients. Almoznino et al. [5] also found that patients with recurrent aphthous stomatitis had elevated levels. Other studies about the relationship between increased IgE serum and xerostomia with recurrent aphthous stomatitis have shown elevated levels in patients [26]. A report on the Kashmiri population in 2017 showed that 42% of patients with recurrent aphthous stomatitis reported high levels beyond normal [27].

Allergic reactions can lead to the activation of additional cells, like eosinophils [28]. Eosinophils can control immune and inflammatory reactions in a specific area, and their accumulation in the bloodstream and tissue is correlated with allergy [29]. This study found that most patients with recurrent aphthous stomatitis have a normal eosinophil level (40%). The result might happen due to eosinophils high rates of spontaneous apoptosis. After being discharged from the bone marrow because of stimulation from IL-5, eosinophils proceed to move through the bloodstream and migrate to various tissues [30]. The lifespan of eosinophils in the blood is 8 to 18 hours. As the developing eosinophil goes through differentiation and maturation, the ability to reproduce diminishes [31]. This study is among the few investigations that examined the level of IgE and eosinophil in individuals suffering from recurrent aphthous stomatitis. However, because of the absence of similar reports and variations in methods, comparing the results was not possible, which was a limitation of this study.

Allergy leads to a higher production of IgE-antibodies by B-lymphocytes, responsible for allergic inflammation and the least common type of antibodies. Subsequently, IgE Fe receptors on mast cells become activated, releasing biogenic mediators that cause the signs and symptoms of allergy [28]. Farhad-Mollashahi et al. [4] conducted a study about the levels of IgE and ECP in saliva among patients both with and without recurrent aphthous stomatitis. The results showed a significant difference in IgE and ECP values. In patients with recurrent aphthous stomatitis, IgE and ECP levels were recorded at 1.11 ± 0.65 IU/ml and 26.93 ± 6.95 ng/ml, respectively, while in those without the condition had 0.73 ± 0.39 IU/ml and 21.97 ± 6.72 ng/ml. Therefore, patients who experience recurrent aphthous stomatitis have higher salivary IgE and ECP levels.

5. Conclusion

In conclusion, this study showed that patients with recurrent aphthous stomatitis had allergy history, high levels of IgE, and eosinophil. Therefore, dentists should understand the predisposition factor of recurrent aphthous stomatitis, such as allergy. The results study showed that allergy could potentially contribute to recurrent aphthous stomatitis. Moreover, future studies should clarify the importance and role of other allergy biomarkers and their relationship in causing recurrent aphthous stomatitis.

6. Acknowledgments

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7. Conflict of Interest

The authors do not have any conflict of interest with the parties included in this study.

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Appendix

Appendix 1. Age and sex distribution of patients (Table 1).

Subject	Age (Years)	Sex
Subject 1	23	Female
Subject 2	21	Female
Subject 3	20	Male
Subject 4	24	Female
Subject 5	17	Female
Subject 6	22	Male
Subject 7	33	Male
Subject 8	17	Male
Subject 9	2	Female
Subject 10	23	Female
Subject 11	15	Male
Subject 12	25	Female
Subject 13	16	Female
Subject 14	24	Female
Subject 15	21	Female
Subject 16	20	Female
Subject 17	23	Female
Subject 18	23	Female
Subject 19	21	Female

Subject	Age (Years)	Sex
Subject 20	22	Female
Subject 21	24	Female
Subject 22	20	Male
Subject 23	28	Female
Subject 24	30	Female
Subject 25	23	Male
Subject 26	22	Female
Subject 27	27	Male
Subject 28	22	Female
Subject 29	16	Male
Subject 30	44	Female
Subject 31	44	Female
Subject 32	19	Male
Subject 33	23	Male
Subject 34	16	Female
Subject 35	36	Female
Subject 36	17	Male
Subject 37	22	Female
Subject 38	14	Female
Subject 39	17	Female
Subject 40	40	Female
Subject 41	25	Female
Subject 42	23	Male
Subject 43	23	Female
Subject 44	21	Female
Subject 45	17	Female
Subject 46	24	Female
Subject 47	23	Female
Subject 48	23	Male
Subject 49	24	Female
Subject 50	24	Male

Appendix 2. Allergy history (Table 2).

Subject	Allergy
Subject 1	No
Subject 2	No
Subject 3	Yes
Subject 4	No
Subject 5	Yes
Subject 6	Yes
Subject 7	No
Subject 8	No
Subject 9	No
Subject 10	No
Subject 11	No
Subject 12	No
Subject 13	No
Subject 14	No
Subject 15	Yes
Subject 16	No
Subject 17	No
Subject 18	Yes
Subject 19	No
Subject 20	No
Subject 21	Yes
Subject 22	No
Subject 23	No
Subject 24	No
Subject 25	No
Subject 26	Yes
Subject 27	Yes
Subject 28	Yes
Subject 29	Yes

Subject	Allergy
Subject 30	No
Subject 31	No
Subject 32	No
Subject 33	No
Subject 34	No
Subject 35	No
Subject 36	No
Subject 37	Yes
Subject 38	No
Subject 39	No
Subject 40	No
Subject 41	No
Subject 42	Yes
Subject 43	No
Subject 44	Yes
Subject 45	No
Subject 46	No
Subject 47	Yes
Subject 48	No
Subject 49	No
Subject 50	No

Appendix 3. IgE levels (Table 3).

Subject	IgE
Subject 1	High
Subject 2	Normal
Subject 3	High
Subject 4	Normal
Subject 5	High
Subject 6	High
Subject 7	High
Subject 8	Normal
Subject 9	High
Subject 10	Normal
Subject 11	Normal
Subject 12	Normal
Subject 13	High
Subject 14	Normal
Subject 15	Normal
Subject 16	Normal
Subject 17	High
Subject 18	High
Subject 19	Normal
Subject 20	Normal
Subject 21	High
Subject 22	Normal
Subject 23	High
Subject 24	Normal
Subject 25	Normal
Subject 26	High
Subject 27	High
Subject 28	High

Subject	IgE
Subject 29	High
Subject 30	Normal
Subject 31	Normal
Subject 32	High
Subject 33	High
Subject 34	High
Subject 35	Normal
Subject 36	High
Subject 37	Normal
Subject 38	High
Subject 39	High
Subject 40	High
Subject 41	Normal
Subject 42	High
Subject 43	High
Subject 44	High
Subject 45	High
Subject 46	Normal
Subject 47	High
Subject 48	High
Subject 49	High
Subject 50	Normal

Appendix 4. Eosinophil levels (Table 4)

Subject	**Eosinofil**
Subject 1	High
Subject 2	Normal
Subject 3	Normal
Subject 4	Low
Subject 5	High
Subject 6	Low
Subject 7	Normal
Subject 8	Normal
Subject 9	Normal
Subject 10	Low
Subject 11	Normal
Subject 12	Normal
Subject 13	High
Subject 14	Normal
Subject 15	High
Subject 16	Normal
Subject 17	High
Subject 18	Normal
Subject 19	Low
Subject 20	Normal
Subject 21	Low
Subject 22	Normal
Subject 23	High
Subject 24	Normal
Subject 25	Low
Subject 26	Low
Subject 27	High
Subject 28	High
Subject 29	High
Subject 30	Low
Subject 31	Low
Subject 32	High

Subject	Eosinofil
Subject 33	Normal
Subject 34	Normal
Subject 35	High
Subject 36	Normal
Subject 37	Low
Subject 38	Normal
Subject 39	High
Subject 40	Low
Subject 41	High
Subject 42	Normal
Subject 43	Low
Subject 44	Low
Subject 45	Normal
Subject 46	Low
Subject 47	Low
Subject 48	Normal
Subject 49	Low
Subject 50	Low