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Contemporary Updates of Recurrent Aphthous Stomatitis

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ABSTRACT:

Recurrent Aphthous Stomatitis (RAS) is a recurrent, painful condition of the skin or mucous membranes, that commonly occurs after puberty. Minor aphthous ulcers appear on the lips, tongue, and buccal mucosa, though major lesions are more commonly found on non-keratinized areas. This review outlines the current knowledge on the etiology, pathophysiology, and clinical presentation of RAS, emphasizing the aspect of complex and multifactorial nature. A wide range of conducive factors has been recognized, including immune dysregulation, nutritional imbalances, hypersensitivity reactions, certain medications, mechanical trauma, exposure to toxins, poor oral hygiene, hormonal shifts, microbial imbalance, tobacco use, psychological stress, systemic diseases, and allergic tendencies. Furthermore, RAS is particularly common among students, with emotional stress and anxiety playing a significant role in triggering and perpetuating episodes. Environmental exposures, particularly to heavy metals may further aggravate the condition by promoting oxidative stress and disrupting the neuroimmune balance. Relevant literature was identified using PubMed, Scopus, and Web of Science databases with keywords such as "Recurrent Aphthous Stomatitis," "oral ulcers," "stress," "heavy metal exposure," and "oxidative stress." Articles published in peer-reviewed journals over the past 20 years were screened using defined inclusion and exclusion criteria. Key data were extracted on clinical patterns, triggering factors, and environmental links, and the findings were integrated to present a comprehensive, evidence-informed understanding of RAS and its broader public health relevance.

1. Introduction

Aphthous ulcers, frequently denoted as RAS or canker sores, are among the most frequent inflammatory conditions affecting the oral mucosa. In the United States, they represent the leading idiopathic cause of intraoral ulceration and are considered one of the most prevalent mucosal disorders [1,2]. Aphthous ulcers are the most prevalent [3], affecting up to 20 - 25% of the global population and having an estimated point prevalence of 4%-66% worldwide [4-9]. It is a common pathologic condition distinguished by the occurrence of benign and noncontagious oral ulceration on a regular basis. An oral disease that first manifests throughout childhood and adolescence and is characterized by recurrent, tiny, round, or oval mucosal ulcers with constricted edges, erythematous haloes, and grey or yellow floors are the clinical description [3,10-13]. Up to 40% of specific pediatric groups may exhibit a history of RAS, with initial ulcer episodes occurring before the age of five. The likelihood of developing the condition tends to rise with age, and in many cases, ulceration typically emerges during adolescence. Children from higher socioeconomic categories could experience this issue more frequently than kids from lower socioeconomic groups [13].

The ulcers heal in more than two weeks for the severe kind and in 10-14 days for the more prevalent type when there is severe or moderate pain [3,12-15]. Painful ulcerative or erosive lesions with irregular margins were observed on the labial mucosa, hard palate and tongue. These lesions typically emerged within 4 to 7 days, although in one COVID-19 case, they developed three days prior to systemic symptom onset and healed within 5 to 21 days. PCR testing for HSV-1/2 returned negative results in two reported cases [16]. It usually appears on the buccal and labial mucosa, as well as the tongue, in otherwise healthy people. It is less frequent for the extensively keratinized mucosa of the gingiva and palate to be affected [2].

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However, there is also research suggesting a connection to a number of systemic illnesses [12]. Behcet's disease, cyclic neutropenia, recurrent intraoral herpes infections, HIV-related oral ulcers, or gastrointestinal conditions are a few conditions that also produce oral ulcers that could be confused for RAS [2]. Refractory and prone to recurrence oral aphthous ulcers may be signs of underlying inflammatory disorders such as inflammatory bowel disease or celiac disease. Oral lesions may precede gastrointestinal symptoms in 5%–10% of affected individuals.

Hippocrates devised the phrase initially, and Mikulicz and Kummel eventually stated it as "Mikulicz's aphthae.". The Greek word aphthi, which translates as "to set on fire" or "to inflame," is the root of the word aphthae, which is claimed to have been originally used by the philosopher. Hippocrates described the discomfort brought on by a common oral disorder at the time (possibly aphthous stomatitis) [17,18].

There have been numerous hypotheses proposed on the etiology of RAS. Although the exact cause of RAS is still unspecified [7]. Stress, allergies, infection, genetic susceptibility, trauma, and dietary inadequacies are examples of impulsive causes even these occur due to climate burden too [19]. A number of research have discovered a link between RAS and stress levels [1]. There are no statistics available yet on how climate change is affecting dental health. Through the same factors impacting air quality, chronic pulmonary disease is connected to climate change and has a high correlation with periodontal disease.

The prevalence of the condition has been shown to be directly influenced by a number of factors, including genetics, exposure to specific foods, hypersensitivity, medications, hormones, heredity, quitting smoking, nutritional deficiencies, and hormonal imbalance [6,7,20]. In addition, Immunological, microbiological, and systemic diseases also play a pathogenic role [6,20].

In silico functional investigations, it is shown that T cell modulation is involved in the etiology of mouth ulcers. [21] One of the genetic risk factors that may affect a person's tendency for recurrent aphthous stomatitis is the wide variety of DNA polymorphisms found throughout the human genome [22]. New drugs are frequently put through trials in an effort to diminish pain and functional impairment. The phrase "complex aphthosis" was first used by Jorizzo and colleagues to describe patients without BD who had three or more virtually constantly present oral aphthae [23].

2. Search strategy:

Relevant literature for this narrative review was identified through structured searches conducted in PubMed, Scopus, and Web of Science using targeted keywords such as “Recurrent Aphthous Stomatitis,” “oral ulcers,” “stress,” “heavy metal exposure,” and “oxidative stress.” Studies published in peer-reviewed journals over the past two decades were screened using predefined inclusion and exclusion criteria, prioritizing those that addressed the etiology, clinical features, and environmental influences of RAS. Eligible articles were selected based on thematic relevance and methodological clarity.

3. Etiology

Recurrent aphthous stomatitis (RAS) is a multifactorial condition characterized by idiopathic, cell- mediated immune activation that leads to localized epithelial breakdown and ulcer formation. The lesions are non-contagious and typically arise independent of acute infectious processes. A wide array of endogenous and exogenous factors contributes to disease onset, with both genetic predisposition and environmental exposures playing pivotal roles [1,9,20,22,24-27].

Genetic susceptibility is strongly implicated, as evidenced by familial clustering and twin studies, where a positive history is documented in up to 46% of cases. This predisposition may modulate immune responses, particularly in individuals with autoimmune conditions such as celiac disease, Behçet’s disease, and inflammatory bowel disorders. These conditions often share immunopathogenic mechanisms that contribute to mucosal vulnerability [8,13,22,28].

Nutritional deficiencies—especially of hematinic factors such as folic acid, iron, vitamin B12, and zinc— are frequently observed in RAS patients. These deficits may impair oxygen delivery to the oral mucosa, resulting in progressive atrophy due to sustained tissue hypoxia. Approximately 20% of RAS cases are attributed to hematinic insufficiency, which may arise secondary to malabsorption syndromes or autoimmune enteropathies like pernicious anemia [12,21,22,24,29-31]. Microbial and allergic triggers also play a

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significant role. Alterations in the oral microbiome, along with viral infections (e.g., HSV-1/2, Epstein-Barr virus, cytomegalovirus), have been associated with ulcerative episodes. Dietary allergens such as chocolate, nuts, gluten, and food additives, as well as hypersensitivity to toothpaste ingredients and sulfhydryl-containing medications, may provoke immune responses targeting epithelial junctions [10,17,19,24]. Notably, tobacco use—both smoking and smokeless forms—appears inversely associated with RAS prevalence, though the underlying mechanism remains unclear.

Psychological stress is a recognized modulatory factor, particularly among students and individuals under academic pressure. Stress may precipitate parafunctional habits like lip or cheek biting, leading to mechanical trauma and mucosal injury. Elevated salivary cortisol and reactive oxygen species during stress episodes can transiently impair immune regulation, facilitating lesion development. However, stress is considered a precipitating rather than a primary etiological factor, given the inconsistent correlation between stress intensity and episode severity [1,20,25,32,33].

Pharmacological agents such as NSAIDs and nicorandil have been reported to induce RAS-like lesions. Although multivitamin supplementation has been investigated for prophylactic benefit, current evidence does not support a significant reduction in episode frequency or duration [9,13,34]. (Table 1)

Table 1. Overview of predisposing causes and clinical triggers of RAS

CAUSES	DESCRIPTION
Genetic predisposition	Inherited tendency for RAS.
Trauma and injury	Tissue damage and physical trauma.
Immune system dysfunction	Abnormal immune response. Inflammation plays a role in RAS.
Stress	Psychological or emotional stress. Parafunctional habits like lip or cheek biting to cause harm to the oral soft tissues.
Nutritional deficiencies	Lack of vital vitamins especially A, B Complex, C, D, and E, folate, iron and zinc or minerals.
Food sensitivity	Allergies or sensitivities to certain foods. Certain foods (such as chocolate, gluten, cow milk, preservatives, nuts, and food colouring agents, citrus fruits, strawberries)
Hormonal factors	Hormonal changes or imbalance. Menstrual cycles or menopause or hormonal fluctuations during pregnancy.
Microbial infections	Bacterial infections (Helicobacter pylori, Streptococcus oralis) and viral infections (Herpes simplex virus, adenoviruses, cytomegalovirus, varicella-zoster virus,).
Medicines	Nicorandil or Nonsteroidal anti-inflammatory medicines (NSAIDs)
Environmental stressors	Climate change adversely affects mental well-being, manifesting as acute psychological trauma from natural disasters or persistent emotional strain driven by eco-anxiety and grief over environmental loss.
Chemical Irritants	Allergy to toothpaste and germs. Mouthwashes containing alcohol Acidic foods and beverages.
Systemic illnesses	Ulcerative colitis, Crohn's disease, Celiac disease, and AIDS.

4. Pathophysiology

The pathogenesis of RAS involves a complex cascade of immunoinflammatory events triggered in genetically predisposed individuals. Upon exposure to specific stimuli—whether nutritional, microbial, mechanical, or psychological—a localized immune response is initiated, targeting the oral mucosa and culminating in ulcer formation [8,13,16,21,22,35,36]. (Figure 1)

Journal of Chemical Health Risks
www.jchr.org
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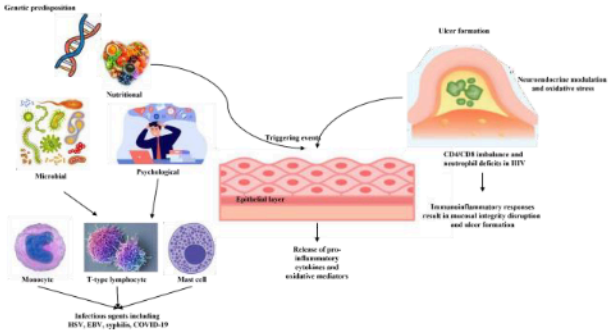

Figure 1: *Multifactorial Pathogenesis of Recurrent Aphthous Stomatitis (RAS). The complex interplay of genetic predisposition, nutritional deficiencies, psychological stress, microbial factors, and systemic infections in the development of oral ulcers. Triggering events initiate immune cell infiltration beneath the epithelial layer—primarily monocytes, T-type lymphocytes, mast cells, and plasma cells—leading to the release of pro-inflammatory cytokines and soluble mediators. In HIV-positive individuals, CD4/CD8 imbalance and neutrophil deficits further compromise mucosal integrity. Neuroendocrine modulation and oxidative stress amplify inflammation, culminating in ulcer formation.*

Histopathological analysis reveals a dynamic infiltration of immune cells across disease stages. In the early pre-ulcerative phase, monocytes and predominantly T-type lymphocytes accumulate beneath the basal epithelial layer, accompanied by isolated mast cells and plasma cells. As the lesion progresses, polynuclear leukocytes dominate the ulcer center, with infiltration intensity correlating to clinical severity [22,37]. Triggering events stimulate the release of pro-inflammatory cytokines and oxidative mediators, which disrupt epithelial integrity and delay tissue repair. Psychological stress further amplifies this response by increasing leukocyte counts and altering immune regulatory activity. Elevated salivary cortisol and reactive oxygen species are believed to contribute to mucosal inflammation and lesion persistence [1,20,32,33].

Infectious agents such as HSV-1/2, Epstein-Barr virus, and other systemic pathogens (e.g., tuberculosis, COVID-19, syphilis) may directly or indirectly contribute to ulcerative lesions. In HIV-positive individuals, disrupted CD4/CD8 lymphocyte ratios and neutrophil deficits mirror immune profiles observed in autoimmune conditions, reinforcing the role of immunological dysregulation in RAS pathophysiology [10,22].

Neuroendocrine modulation and oxidative stress are increasingly recognized as central to disease progression. Stress-induced immune shifts, coupled with genetic and nutritional vulnerabilities, create a permissive environment for recurrent mucosal injury. Although RAS is generally self- limiting, frequent recurrences can significantly impair quality of life, affecting speech, eating, and emotional well-being.

5. Clinical features

RAS typically emerges after puberty and affects approximately 5–25% of individuals, with peak prevalence between ages 10 and 40 [11]. Minor aphthous ulcers are significantly more common than Herpetiform ulcers and Major aphthous ulcers [38]. About 10% of RAS patients present with Major RAS, a more severe clinical variant. Herpetiform RAS, though less frequent, is considered the most serious form due to its clustered presentation and recurrence [11]. Women may predominate across both adult and pediatric cohorts, and children from higher socioeconomic backgrounds may be disproportionately affected [11,13]. Additionally, HSV-1 lesions are frequently observed on keratinized oral mucosae [4,5].

Minor aphthae commonly affect the lips, tongue, and buccal mucosa, while Major RAS lesions are found on the lips, soft palate, labial mucosa, and tonsillar fauces. Although rare on the gingiva, palate, or posterior tongue, RAS lesions are more prevalent on non-keratinized surfaces, particularly the labial and buccal mucosa and the floor of the mouth [4]. RAS lesions persist for up to six weeks [10,11,24]. Regardless of the size of the RAS lesion, the pain is often disproportionately severe relative to the extent of the ulceration [10].

The three main clinical variants of RAS are Major, Minor, and Herpetiform. They are small, round, or oval in shape, aphthae typically have an erythematous halo and a grey-white pseudo membrane [10]. Major RAS oral ulceration lesions range in size from 1 cm to 3 cm, they are bigger and create ulcers that are deeper. Herpetiform RAS is distinguished by clusters of tiny, numerous, 1 to 3 mm lesions and some of them may combine to form bigger, more erratic ulcerations. RAS is a challenging condition to manage [11]. Oral lesions are most prevalent and

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reoccur most frequently in herpetiform RAS [11]. Lesions larger than 10 mm in diameter and capable of leaving scars are present in 10% of patients [39].

The majority of persons with RAS experience few ulcers with a width of less than 10 mm that goes away without leaving any scars within 7 to 10 days. Although there are no recognized causes, localized physical trauma can lead to ulcers in susceptible individuals [34]. Major RAS will typically leave scars and take two to six weeks to recover [4]. Individuals may feel a prickling or burning sensation in the mucosa 24 to 48 hours before the onset of a small aphthous ulcer. Herpetiform RAS usually recover without leaving scars in less than a month [11]. In extreme circumstances, mouth opening may be restricted as a result of repetitive scarring processes [4]. RAS patients may exhibit increased thrombosis of the feeding arterioles supplying the oral epithelial cells, potentially contributing to localized ischemia and ulcer formation [12,22]. Although there is some uncertainty regarding the pattern of intraoral ulcers in PFAPA, the lesions are typically characterized as few to several in number, non-clustered, shallow, and approximately 5 mm in diameter, with spontaneous healing observed within 5 to 10 days [12].

6. CONCLUSION

RAS remains a perplexing and multifactorial condition influenced by genetic, immunological, environmental, and psychosocial factors. Despite its high prevalence and recurrent nature, the precise pathophysiological mechanisms driving RAS remain insufficiently grasped. Emerging evidence suggests that oxidative stress, heavy metal exposure, and immune dysregulation play significant roles in disease onset and progression. While conventional treatment strategies focus on symptomatic relief, the integration of targeted therapies addressing immune modulation and nutritional deficits may provide a more comprehensive approach to management. Given the impact of RAS on quality of life, further research is essential to explore novel therapeutic interventions, establish standardized diagnostic criteria, and assess the long-term effects of environmental triggers. A multidisciplinary approach combining clinical, molecular, and epidemiological perspectives will be crucial in advancing our understanding and improving patient outcomes.

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