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The Mind - Mouth Connection: Understanding of Psychosomatic Disorders

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ABSTRACT

Stress is an unavoidable component of modern life and has significant effects on both systemic and oral health. Psychological stress can influence the body through complex neuroendocrine and immunological mechanisms, particularly involving the hypothalamic–pituitary–adrenal (HPA) axis and sympathetic nervous system. These physiological responses lead to the release of stress mediators such as cortisol, adrenaline, and inflammatory cytokines that can alter immune function, inflammatory responses, and tissue healing. The oral mucosa is highly sensitive to psychological disturbances, and several oral mucosal disorders have been linked to stress and emotional factors . Conditions such as recurrent aphthous stomatitis, oral lichen planus, burning mouth syndrome, herpes labialis, necrotizing ulcerative gingivitis, and xerostomia have been reported to show a strong association with psychological stress. Evidence suggests that stress can act as a triggering or exacerbating factor in these disorders by impairing immune responses and altering salivary composition . Management of stress-related oral disorders requires a multidisciplinary approach that includes dental treatment, pharmacological therapy, behavioral modification, and psychological support.This article , therefore emphasizes the critical importance of incorporating stress evaluation and management into routine dental care,advocating for a holistic , multidisciplinary approach to oral mucosal health that bridges the gap between psychological well -being and clinical outcomes .

Keywords: psychosomatic disorders,stress responders and psychological support,psychological management, burning mouth syndrome, Recurrent apthous stomatitis, oral lichen planus.

INTRODUCTION

Stress is defined as the body's physiological and psychological response to internal or external demands that disrupt normal homeostasis . In modern society, individuals are exposed to various stress including academic pressure, occupational challenges, and social conflicts [(1,2)].Chronic stress can negatively affect

both mental and physical health and is recognized as an important risk factor for several systemic diseases.The oral cavity often reflects the general health status of an individual. The oral mucosa acts as a protective barrier against microorganisms, toxins, and mechanical trauma[2,3].However, this protective function can be compromised when the body is exposed to chronic stress . Psychological stress influences oral health through both biological and behavioral mechanisms.

From a biological perspective, stress activates the hypothalamic–pituitary–adrenal axis and the sympathetic nervous system, leading to the release of cortisol and catecholamines .These hormones influence immune responses and inflammatory pathways, making oral tissues more susceptible to infections and mucosal diseases . Behavioral changes associated with stress such as smoking, alcohol consumption, and poor oral hygiene further increase the risk of oral diseases .The relationship between psychological factors and physical diseases has been recognized for centuries[5,6] . However, scientific exploration of stress began with the work of Hans Selye, who introduced the concept of the General Adaptation Syndrome describing the body's response to stress in three stages: alarm, resistance, and exhaustion ..During the 1950s, psychosomatic disorders were recognized in the Diagnostic and Statistical Manual of Mental Disorders (DSM-I) . Dentistry later adopted these concepts when clinicians observed that emotional stress could precipitate oral lesions. Salivary cortisol has been identified as an important biomarker reflecting psychological stress in oral diseases.This article, therefore emphasizes the critical importance of incorporating stress evaluation and management into routine dental care , a multidisciplinary approach to oral mucosal health

STRESS RESPONDERS

Stress triggers a complex set of physiological reactions in the body that involve the nervous, endocrine, and immune systems. These reactions are collectively known as stress responses, and the systems involved are often referred to as stress responders[9,11].Activation of these systems helps the body adapt to stressful conditions, but prolonged or chronic stress can lead to dysregulation of these mechanisms and may negatively affect systemic and oral health . In the oral cavity, stress responders can influence immune responses, inflammatory mediators, and salivary composition, thereby contributing to the development and progression of oral diseases such as periodontal disease, oral mucosal lesions, and temporomandibular disorders [12].The various stress responders include hypothalamic -pituitary axis , Sympathetic -Adrenal -Medullary system , Immune and inflammatory mediators ,salivary stress biomarkers.

1. Hypothalamic-Pituitary-Adrenal (HPA) Axis

The hypothalamic-pituitary-adrenal (HPA) axis is one of the primary physiological pathways involved in the stress response. When an individual experiences stress, the hypothalamus secretes corticotropin-releasing hormone (CRH), which stimulates the anterior pituitary gland to release adrenocorticotropic hormone (ACTH). ACTH subsequently stimulates the adrenal cortex to produce cortisol, the major glucocorticoid hormone associated with stress[10,13].Cortisol plays a vital role in regulating metabolism, immune responses, and inflammatory processes. However, prolonged elevation of cortisol levels can suppress immune function and increase susceptibility to infections. In the oral cavity, elevated cortisol levels have been associated with impaired wound healing, increased inflammatory responses, and a greater risk of periodontal tissue destruction .[16]

2.Sympathetic-Adrenal-Medullary (SAM) System

Another important stress responder is the sympathetic-adrenal-medullary (SAM) system Activation of the sympathetic nervous system during stress leads to stimulation of the adrenal medulla, resulting in the

release of catecholamines. primarily epinephrine (adrenaline) and norepinephrine (noradrenaline) .These hormones are responsible for the classic "fight-or-flight" response, which prepares the body to respond to stressful stimuli by increasing heart rate, blood pressure, and energy availability. In the oral environment, catecholamines may influence salivary gland function, reduce salivary flow, and alter the oral[15]

3. Immune and Inflammatory Mediators

Stress can also affect the immune system by altering the production of inflammatory mediators. Chronic stress has been associated with increased levels of pro-inflammatory cytokines, including interleukin-1 (IL-1), interleukin-6 (IL- 6), and tumor necrosis factor-alpha (TNF-a) ..These cytokines play a significant role in the inflammatory processes associated with periodontal disease. Elevated levels of inflammatory mediators can enhance the destruction of periodontal tissues and promote alveolar bone resorption, thereby contributing to the progression of periodontal diseases.[14]

4. Salivary Stress Biomarkers

Saliva has emerged as an important diagnostic fluid for evaluating physiological stress responses. Several salivary biomarkers are commonly used to assess stress- related changes in the body. These include,Salivary cortisol, which is widely recognized as a reliable biomarker of psychological ,stress Salivary alpha-amylase, which reflects sympathetic nervous system activity responses Chromogranin A, a neuroendocrine marker associated with stress Secretory immunoglobulin A (IgA), which indicates immune system activity in the oral cavity.Alterations in these salivary biomarkers provide valuable information about the interaction between psychological stress and oral health.[16, 17]

PHYSIOLOGICAL RESPONSES TO STRESS

The body reaction to stress is defined as anything (real, symbolic or imagined) that threatens an individual's survival-is to put into responses that seek to diminish the impact of the stressor. Stress causes cultivation of physiological responses that seek to react to the stressors, bring about an adaptive response, and restore homeostasis. Stress activates the hypothalamic–pituitary–adrenal axis leading to secretion of corticotropin releasing hormone, adrenocorticotropic hormone, and cortisol . Stress also stimulates the sympathetic nervous system causing the release of catecholamines such as adrenaline and noradrenaline. Chronic stress suppresses immune function and increases susceptibility to infections . It may also alter salivary secretion and composition, reducing the protective role of saliva[18, 19]

A) NEUROTRANSMITTER RESPONSES TO STRESS

Stress Stimulus

↓

Activation of Autonomic Nervous System

↓

Release of Catecholamines (Noradrenaline)

↓

Central Neurotransmitter Changes

↓

increased Serotonin Turnover increased Dopaminergic Neurotransmission

↓

Release of CRF, Glutamate & GABA

(CRF (Corticotropin-Releasing Factor), Glutamate ,GABA(Amino Acid& Peptidergic NTs))[20, 21]

B) ENDOCRINE RESPONSES TO STRESS

Stress acts or potentiates the hypophysial-pituitary-Adrenal axis (HPA axis), ultimately leading to increased serum corticosteroid levels, which is thought to have anti-stress effects.

- Hypothalamus: The process starts here. In response to stress, the hypothalamus releases the Corticotropin-Releasing Factor (CRF), also known as CRH.
- Pituitary Gland: The CRF travels to the pituitary gland, signaling it to release Adreno-Cortico Trophic Hormone (ACTH) into the bloodstream.
- Adrenal Cortex: The ACTH travels down to the adrenal glands (located on top of your kidneys). Specifically, it targets the Adrenal Cortex (the outer layer).
- Corticosteroid Synthesis: The adrenal cortex then begins producing and releasing Corticosteroids, primarily Cortisol (often called the "stress hormone")[22]

NEUROBIOLOGICAL MECHANISMS

A) Stress affects the neuroendocrine and immune systems

Psychological stress affects the normal nerve-endocrine-immune pathway of the human body, leading to over-activation of the hypothalamus-pituitary-adrenal (HPA) axis and sympathetic nerve-adrenal medulla. The hypothalamus-pituitary-gonad (HPG) axis is regulated by the HPA axis, which can inhibit or overactivate endocrine and immune systems, and the secretion of corresponding hormones, cytokines and proteins changes accordingly. This may damage the oral mucosa and induce OLP and RAU. Moreover, because erosive OLP easily ruptures repeatedly and RAU easily relapses, patients are under pressure for a prolonged period of time, which may result in anxiety and depression. thus accelerating the disease progress[24, 25]

B) Neuroendocrine dysfunction

Under acute or chronic stress conditions, hyperfunction of the HPA axis may be the neurobiological basis of depression and anxiety. Studies observing brain imaging in patients with depression and anxiety dis-orders have found that the volume of the hypothalamus, and the interaction and coherence of the endocrine and autonomic nervous systems, are affected by stress. [26]The hypothalamus and marginal area, as key parts of the fine neuroendocrine circuit, release excessive corticotropin-releasing hormone after being affected by stress, and corticotropin-releasing hormone further regulates the secretion of adrenocorticotropic hormone. Inactivation of the HPA axis in response to corticotropin-releasing hormone and over-activation of the HPA axis may lead to the development of depres-sion and anxiety. In addition, patients with anxiety and depression usually exhibit sympathetic nerve-adrenal medulla axis hyper-responsiveness. Stress induces a considerable release of catecholamine hormones from the adrenal medulla, and the release of epinephrine, norepinephrine, and high levels of adrenal steroid hormones from the adrenal cortex are risk factors for major depressive disorder[27, 28]

C) Effects of cortisol and dehydroepiandrosterone.

Cortisol is a glucocorticoid secreted by the adrenal cortex that has a variety of biological effects, including anti-inflammation and mood regulation, and it can affect the stress response of the body. However, under a state of chronic stress, despite the excessive secretion of cortisol, inflammatory damage and emotional disorders are aggravated. This suggests dysfunction of the glucocorticoid receptor in the body, so that anti-inflammatory effects cannot be exerted. In addition, dehydroepiandrosterone (DHEA), an endogenous antagonist of cor-tisol, can be secreted by the adrenal cortex. the central nervous system, and the gonads. Its sulfated form is DHEA sulfate, and DHEA has been shown to be involved in the regulation of brain

perception processes, such as neuropathic pain. Because levels of salivary DHEA and cortisol are closely related to levels of serum free DHEA and cortisol secretion, detection of DHEA and cortisol in saliva is often used to measure stress levels, anxiety and depression[29,30]. Salivary DHEA level was inversely correlated with anxiety and depression scores. Stress states, such as anxiety and depression, will stimulate the HPA axis and increase salivary cortisol secretion, thus reducing salivary DHEA secretion and increasing the prevalence of RAU. Oral mucosal epithelial mucin 1 (MUCI) can protect mucosal epithelial cells and then interfere with physiological activities. The expression level of MUCI has been shown to positively correlate with the level of DHEA, and to be inversely correlated with cortisol and the cortisol/DHEA ratio. serum cortisol levels in patients with erosive and non-erosive OLP evaluated the anxiety and depression status of those patients. The comprehensive average serum cortisol levels of the patients with OLP were found to be higher than those of healthy controls, and negative emotions were a related factor for the occurrence and prognosis of OLP. Generally, compared with healthy people, patients with BMS have lower levels of DHEA and higher levels of cortisol. . Therefore, salivary cortisol and DHEA can be used as saliva biomarkers to evaluate stress in patients with oral mucosal diseases[31, 32]

D) Dual hormones affect immune function

During stress state caused by oral mucosal dis-eases, hyperfunction of the HPA axis and regulation of the HPG axis can affect the stability of the immune system. Patients with RAU who have negative emotions also have elevated levels of cortisol, while levels of testosterone are decreased. Testosterone inhibits the cyclo-oxygenase pathway of arachidonic acid metabolism, inhibits the synthesis and release of prostaglandins, increases the expansion of suppressor T cells, exerts anti-inflammation effects, and inhibits the autoimmune response One study demonstrated that the percentage of CD3+ and CD8- peripheral blood lymphocytes was higher in patients with RAU than in healthy controls, and the levels of immunoglobulin (IgG, IgA. and complements C3 and C4 were considerably higher versus healthy controls, indicating that the pathogenesis of RAU may be related to the hyperactivity of cellular and humoral immunity Due to the inhibition of testosterone secretion by stress and cortisol, the immune function of the body may be over-activated, and RAU may be induced[33,34]. As a common target for autoantibodies, it plays an important role in the regulation of auto-immunity, and can promote the activation of nuclear factor-kappa B (NF-KB), thereby triggering the production of immune factors and various pro-inflammatory cytokines. Such as interleukin (IL)-6 and type-I interferon . Levels of TRIM21/NF-kB in the supernatant of CD3+ and CD4+ T cells under normal conditions were significantly reduced after treatment with oestradiol and testosterone compared with controls, suggesting that the expression of TRIM21 was inhibited, which led to the regulation of IL-6. The decreased expression of cyto-kines, such as type-I interferon, improves the immune imbalance of T cells. Therefore, testosterone and oestradiol can correct the imbalance of the autoimmune system and delay the occurrence and development of OLP via TRIM21-mediated signal transduction. This may confirm the dual-hormone hypothesis to some extent[35, 36].

E) Influence of cytokine disorder

Cytokines are mainly produced by immune cells. Microglia in brain regions, such as the hypothalamus and basal ganglia of the cen-tral nervous system, and other central ner-vous system cells, such as neurons and astrocytes, release pro-inflammatory cyto-kines. There is evidence that the immune system of patients with depression is imbal-anced, with immune activation (increased release of cytokines, such as IL-1. IL-6. tumour necrosis factor [TNF]-2 and acute reaction proteins) and immunosuppression (decreased lymphocyte proliferation or nat-ural killer-cell activity of T cells and decreased number of helper T cells). The HPA axis can be activated by IL-1. IL-6, and TNF-z. High cortisol levels in the blood directly

stimulate indoleamine 2, dioxygenase in immune cells, such macrophages and dendritic cells Interleukins and interferons stimulate indo-leamine 2, 3-dioxygenase to increase its activity, leading to most of the tryptophan metabolism in the tryptophan-kynurenine pathway, significantly reducing serotonin synthesis, and affecting both sleep and emo-tional regulation. High tryptophan-kynur enine levels also trigger the glutamatergic system, with excitatory glutamatergic projections to a subset of neurons in the lateral hypothalamus perifornical area strongly associated with anxiety disorders dependent on corticotropin-releasing hormone cell[31,32]. An increase in tryptophan-kynurenine levels can also lead to the production of reactive oxygen species and nitrogen free radicals, causing oxidative stress, which, in turn, leads to local tissue damage and the activation of inflammatory reactions. Patients with OLP have been shown to exhibit significantly increased oxidative stress markers and decreased antioxidant markers in saliva and serum/plasma. In patients with RAU, the levels of TNF-a, IL-2 and total oxidation state is shown to be increased, while the level of IL-10 is decreased. Abnormal cytokine activity may accelerate lipid peroxidation and lead to the increase of serum oxidative stress, which plays an important role in oxidative/antioxidant defence in patients with RAU In addition to the central nervous system. pro-inflammatory cytokines, such as IL-6. TNF-a, and type-I interferon. may also be released from the periphery of the body to induce a local inflammatory response. This demonstrates that hyper-function of the HPA axis may enhance local inflammatory responses and induce oral mucosal diseases[25, 28]

CLASSIFICATION OF PSYCHOSOMATIC ORAL DISEASES

(McCarthy P. L and shklar. G (1980)

- **Oral Psychosomatic Diseases**
- Oral lichen planus
- recurrent aphthous stomatitis
- glossitis& stomatitis Areata Migrans
- **Oral Diseases in which Psychological Factors Play an Etiologic Role**
- erythema multiforme
- mucous membrane pemphigoid
- chronic periodontal disease.
- **Oral Infections Predisposed by Emotional Stress**
- recurrent herpes labialis
- necrotizing ulcerative gingivitis.
- **Oral Diseases Induced by Neurotic Habits**
- Leukoplakia
- Biting of oral mucosa (self mutilation)
- Physical /mechanical irritation
- Dental / periodontal disease produced by Bruxism
- **Neurotic Oral Symptom**
- Glossodynia (Glossipyrosis)
- Dysgeusia
- Mucosal pain [10]

PSYCHOSOMATIC DISORDER OF ORAL CAVITY

Aphthous stomatitis

Aphthous stomatitis, commonly known as recurrent aphthous ulcers or canker sores, is a frequently occurring oral mucosal condition that has a strong association with psychological stress. Emotional stress can alter immune function and increase the release of inflammatory mediators, leading to mucosal breakdown and ulcer formation. These ulcers typically present as small, round or oval lesions with a yellowish-white base surrounded by an erythematous halo, most commonly affecting the non-keratinized mucosa such as the buccal mucosa, labial mucosa, and floor of the mouth. Patients often experience pain, burning sensation, and difficulty in eating or speaking. Stress-related episodes may be recurrent and tend to coincide with periods of anxiety or emotional disturbance. Management includes stress reduction, maintenance of good oral hygiene, avoidance of triggering foods, and symptomatic treatment using topical anesthetics, anti-inflammatory agents, or protective pastes to promote healing and reduce discomfort.[10]

- **Recurrent Aphthous Stomatitis**

A common condition characterized by recurrent painful ulcers surrounded by erythematous halos. Psychological stress is a well-recognized triggering factor.It affects approximately 20% of the general population.The term aphthous has been derived from the Greek word aphtha meaning ulceration. One or two days before the onset of ulceration, the prodromal phase of paresthesia followed by pain is present. In cases of aphthous ulcers, acute psychological problems appear to have precipitated attacks of the disease. Iron, vitamin B12, and folic acid deficiency are also considered to be predisposing factors[34, 36]

Other factors that have readily been implicated in promotion or exacerbation of RAS include positive family history, local trauma, smoking cessation, and psychological stress. Psychological stresses induce immunoregulatory activities by increasing the number of leukocytes at the sites of inflammation. clinical classification includes minor, Major and herpetic types. The potential causes of RAU include trauma, body oxidation-antioxidation imbalance, genetic predisposition, the influence of systemic diseases, and stress. Stress is suggested to be the biggest factor inducing RAU, which mainly affects women, and compared with housewives and other professions, students are shown to be more susceptible to stress. Anxiety, stress, and depression are more common in patients with a history of RAU than in healthy people, which suggests that negative emotions may promote the recurrence of lesions. A recent study that evaluated the clinical and demographic factors of RAU found that the condition was related to anxiety but not depression. Conversely, by evalu-ating the relationship between salivary cortisol levels and anxiety and depression in patients with RAU, other researchers found RAU to be related to depression,But not anxiety. Recurrent aphthous ulcers were seen equally in the age group of 31-60 years and were more frequent in females as compared to males. [24, 27]

- **Recurrent herpes labialis (RIL)**

Recurrent herpes labialis is a common viral infection caused by reactivation of the Herpes simplex virus type 1, often triggered by psychological stress. After the primary infection, the virus remains dormant in the trigeminal ganglion and can be reactivated when the body's immune response is compromised. Emotional stress plays a significant role by suppressing immune function and increasing susceptibility to viral reactivation. Clinically, it presents as clusters of small, painful vesicles on the lips or perioral region, which rupture to form ulcers and subsequently crust over. Patients may experience prodromal symptoms such as tingling, itching, or burning before the appearance of lesions. Recurrent episodes are often associated with periods of anxiety, fatigue, or illness. Management focuses on stress control, maintenance

of general health, and the use of antiviral medications such as Acyclovir to reduce the severity and duration of episodes.[9]

Oral Lichen Planus

Oral lichen planus is a chronic inflammatory mucocutaneous disorder that has been associated with psychological stress and anxiety. Stress is believed to influence the condition by altering immune regulation, particularly T-cell–mediated responses, leading to damage of the basal cells of the oral epithelium. Clinically, it commonly presents as bilateral white, lace-like patterns known as Wickham's striae, most frequently seen on the buccal mucosa, though erosive and ulcerative forms may also occur, causing pain and burning sensation. Periods of emotional stress can exacerbate the severity and frequency of lesions. Although the exact cause remains multifactorial, stress is considered an important triggering and aggravating factor. Management includes stress reduction, regular monitoring, and symptomatic treatment with topical corticosteroids to control inflammation and relieve discomfort.[33, 37]

Geographic tongue (benign migratory glossitis)

Geographic tongue, also known as benign migratory glossitis, is a common inflammatory condition of the tongue that has been associated with psychological stress. It is characterized by irregular, erythematous depapillated areas on the dorsum of the tongue, surrounded by slightly raised, whitish borders that change location over time, giving a map-like appearance. Stress is believed to play a role by altering immune responses and increasing inflammatory mediators, which can affect the turnover and regeneration of the filiform papillae. Patients may experience a burning sensation, sensitivity to spicy or acidic foods, or mild discomfort, although many cases remain asymptomatic. The condition is benign and self-limiting but may show periods of exacerbation during times of heightened stress. Management mainly focuses on reassurance, stress reduction, and symptomatic relief, such as avoiding irritants and using topical agents when necessary.[21,24]

Erythema multiforme (EM) and mucous membrane pemphigoid (MMP)

Erythema multiforme is an acute, immune-mediated disorder affecting the skin and oral mucosa, often presenting with characteristic target lesions and painful ulcerations. Although infections—particularly Herpes simplex virus—and certain drugs are the primary etiological factors, psychological stress plays a significant role as a precipitating and modifying factor in the development and recurrence of the condition. Stress can trigger the reactivation of latent herpes simplex virus, which subsequently initiates an exaggerated immune response leading to epithelial cell damage and lesion formation. Additionally, stress influences the hypothalamic-pituitary-adrenal axis, resulting in increased cortisol levels and altered cytokine production, thereby impairing cell-mediated immunity and promoting inflammation. [27,32]

Chronic periodontal diseases (CPD)

Chronic periodontitis is a slowly progressive inflammatory condition characterized by destruction of the supporting structures of the teeth, presenting clinically with gingival inflammation, bleeding on probing, periodontal pocket formation, clinical attachment loss, gingival recession, tooth mobility in advanced stages, and often halitosis, while radiographic features include horizontal or vertical alveolar bone loss and possible furcation involvement. The disease typically shows a site-specific pattern with periods of remission and exacerbation. Psychological stress significantly influences the onset and progression of chronic periodontitis through multiple pathways.[10,12] It activates the hypothalamic–pituitary–adrenal axis, leading to increased cortisol levels that suppress immune function and impair host resistance to periodontal pathogens. Stress also alters immune responses by reducing neutrophil function and increasing pro-inflammatory cytokines such as interleukin-1 and tumor necrosis factor-alpha, thereby enhancing

tissue destruction and bone loss. In addition, stress contributes to adverse behavioral changes such as poor oral hygiene, smoking, unhealthy dietary habits, and irregular dental visits, which further aggravate plaque accumulation and disease severity. It may also influence the composition of oral microflora and delay wound healing, resulting in a poorer response to periodontal therapy. Thus, stress acts as an important modifying risk factor in chronic periodontitis, and its management is essential for improving treatment outcomes and preventing disease progression.[16]

Necrotizing Gingivitis (ANUG)

Stress is a significant contributing factor in the onset and progression of Acute Necrotizing Ulcerative Gingivitis (ANUG), as it affects both the body's immune response and an individual's behavior. Psychological stress increases the release of cortisol, which suppresses immune function by reducing neutrophil activity and weakening the body's defense against infections. This weakened immunity allows pathogenic microorganisms such as Fusobacterium nucleatum, Prevotella intermedia, and spirochetes to proliferate, playing a key role in the development of ANUG. Additionally, stress disrupts the normal inflammatory response by elevating pro-inflammatory cytokines like interleukin-1 and tumor necrosis factor-alpha, leading to greater tissue damage and slower healing.[12, 18]

Biting of oral mucosa (self mutilation)

Biting of the oral mucosa, also known as cheek or lip biting (morsicatio buccarum or labiorum), is a common stress-related parafunctional habit. Psychological stress and anxiety can lead to unconscious repetitive behaviors, including chewing or biting the inner lining of the cheeks, lips, or tongue. This occurs due to increased neuromuscular activity and as a coping mechanism to relieve emotional tension. Clinically, it presents as whitish, thickened, or shredded areas of mucosa, often along the occlusal line, sometimes accompanied by soreness or ulceration. Chronic biting may lead to inflammation, secondary infections, and discomfort during eating or speaking. Management primarily involves stress reduction, behavioral therapy, and patient awareness. In some cases, protective appliances such as mouth guards may be used to prevent further trauma to the oral tissues.[18, 20]

Bruxism

Bruxism is a parafunctional habit characterized by grinding or clenching of teeth, commonly associated with psychological stress and anxiety. Emotional stress activates the central nervous system, increasing muscle activity, particularly in the masticatory muscles such as the masseter and temporalis. This heightened neuromuscular activity often occurs unconsciously, especially during sleep (sleep bruxism) or periods of concentration (awake bruxism). Chronic stress leads to sustained muscle tension, resulting in excessive tooth wear, enamel erosion, tooth sensitivity, and even temporomandibular joint (TMJ) disorders. Patients may also experience headaches, facial pain, and disturbed sleep. Stress-induced bruxism is considered multifactorial, with contributing factors including personality traits, coping mechanisms, and lifestyle habits. Management focuses on stress reduction techniques such as relaxation therapy, behavioral modification, and the use of occlusal splints to prevent further dental damage.[33,37].

Glossopyrasis

Glossopyrosis, commonly referred to as burning mouth syndrome (BMS), is characterized by a persistent burning sensation of the tongue without any obvious clinical lesions. Psychological factors, particularly stress, anxiety, and emotional disturbances, play a significant role in its onset and progression. Chronic stress can alter pain perception and reduce salivary flow, leading to oral discomfort and a burning sensation. It also affects the hypothalamic–pituitary–adrenal (HPA) axis, resulting in increased cortisol levels that may contribute to mucosal sensitivity and neuropathic changes. Patients often report worsening

symptoms during periods of heightened emotional stress, along with associated features such as xerostomia and taste disturbances. Thus, glossopyrosis is considered a psychosomatic condition where stress management, counseling, and behavioral therapies are essential components of treatment alongside symptomatic relief.[11, 16]

Dysgeusia

Dysgeusia, defined as a distortion or alteration in the sense of taste, is increasingly recognized as a stress-related oral manifestation. Psychological stress can influence taste perception through multiple mechanisms involving both central and peripheral pathways. Activation of the hypothalamic–pituitary–adrenal (HPA) axis during stress leads to elevated cortisol levels, which may alter neurotransmitter activity and impair gustatory signal processing. Additionally, stress often contributes to reduced salivary flow (xerostomia), which is essential for dissolving gustatory stimulants and maintaining normal taste function. Changes in saliva composition, including decreased proteins and electrolytes, further disrupt taste receptor sensitivity. Stress-induced behaviors such as poor oral hygiene, altered dietary habits, smoking, or medication use (e.g., antidepressants and anxiolytics) may also exacerbate dysgeusia. Clinically, patients may report a persistent metallic, bitter, or unpleasant taste, even in the absence of identifiable local pathology. Management primarily involves addressing the underlying stress through behavioral therapy, lifestyle modification, and, when necessary, pharmacological support, alongside maintaining good oral hygiene and adequate hydration.[12, 16]

Myofascial pain dysfunction syndrome

Stress significantly contributes to the initiation and worsening of Myofascial Pain Dysfunction Syndrome (MPDS), a condition commonly involving the muscles of mastication. Psychological stress stimulates the hypothalamic–pituitary–adrenal (HPA) axis, resulting in elevated cortisol levels and increased neuromuscular activity. This leads to prolonged muscle contraction, particularly in the jaw muscles, causing fatigue, reduced blood supply, and pain. Stress is also closely linked to parafunctional habits such as bruxism and clenching, which impose excessive strain on the masticatory system and intensify muscle tenderness and dysfunction[18, 22] Furthermore, stress influences pain perception by lowering the pain threshold and enhancing central sensitization, thereby increasing susceptibility to chronic pain. Emotional factors like anxiety and depression can further intensify and prolong symptoms. Continuous muscle contraction reduces blood flow, leading to the buildup of metabolic byproducts such as lactic acid, which contributes to soreness and the development of trigger points. In addition, stress-related behavioral changes, including disturbed sleep and fatigue, hinder muscle recovery and worsen the condition. Overall, stress serves as both a triggering and sustaining factor in MPDS, affecting its onset, severity, and persistence[20, 26]

Xerostomia

Dry mouth syndrome, also known as xerostomia, is the abnormal reduction of saliva and can be a common finding in psychiatric patients. Dryness of mouth affects the quality of life. In young adults, the causes are usually associated with stress, anxiety, depression, and nutritional deficiencies. Alcohol abuse and use of illicit drugs also are significant contributors in the etiopathogenesis of dry mouth. Xerostomia was found to be more in females than in male patients, and was reported to be higher in cases than in controls in the present study. The amount of xerostomia increases with advancing age, and was seen more in the age group of 51-60 and 61-70 years.[12, 15]

MANAGEMENT OF PSYCHOSOMATIC DISORDERS

Psychological Management

Psychological management plays an important role in treating oral mucosal diseases associated with stress. Since psychological factors such as anxiety, emotional stress, and depression can influence the onset and severity of oral lesions, addressing these factors is essential for effective treatment. The goal of psychological management is to reduce stress, improve coping mechanisms, and promote overall mental well-being, which in turn helps in controlling oral symptoms.[11, 14]

Patient Education and Counseling

The first step in psychological management is educating the patient about the relationship between stress and oral health. Many patients are unaware that psychological stress can trigger or worsen oral mucosal conditions. Proper counseling helps patients understand the role of emotional factors in disease progression and encourages them to adopt stress-reducing strategies.[12]

Stress Reduction Techniques

Various stress management techniques can help control psychological triggers associated with oral mucosal disorders. These include: Relaxation techniques, such as deep breathing exercises and progressive muscle relaxation, Meditation and mindfulness practices to improve mental calmness,Yoga and physical exercise, which help reduce anxiety and improve overall health,Adequate sleep and rest, which are essential for maintaining psychological balance[38]

Cognitive Behavioral Therapy (CBT)

Cognitive behavioral therapy is a structured psychological treatment that helps patients identify and modify negative thoughts and behaviors. It is particularly useful for patients with chronic oral conditions such as burning mouth syndrome or recurrent aphthous ulcers associated with stress. CBT helps patients develop healthier coping strategies and reduces emotional distress.[33]

Psychotherapy

Psychotherapy may be recommended for patients experiencing severe anxiety, depression, or emotional disturbances related to their oral condition. In such cases, referral to a psychologist or psychiatrist may be necessary. Psychotherapy focuses on understanding the underlying emotional conflicts and improving psychological resilience.[22]

Behavioral Modification

Behavioral therapy helps in controlling parafunctional habits that may be triggered by stress. These habits include:Cheek biting,Tongue biting,Lip biting,Bruxism (teeth grinding).Habit-awareness training and relaxation exercises can help patients gradually eliminate these harmful behaviors[11, 14]

Pharmacological Management

Drugs like antianxiety, antidepressants, sedatives /hypnotics drugs are used.

1. **Anti-anxiety Drugs (Anxiolytics)**

Benzodiazepines

Diazepam (5–10 mg/day)

Alprazolam (0.25–0.5 mg/day)

Mechanism of Action:

Enhance the effect of GABA (gamma-aminobutyric acid), the main inhibitory neurotransmitter in the brain

Increase chloride ion influx → hyperpolarization of neurons

Result: calming effect, reduced anxiety, muscle relaxation

2) Antidepressants

a) Monoamine Oxidase Inhibitors (MAOIs)

Phenelzine

Isocarboxazid

Mechanism of Action:

Inhibit the enzyme monoamine oxidase (MAO)

Prevent breakdown of neurotransmitters: serotonin, dopamine, norepinephrine

Result: increased levels of mood-enhancing neurotransmitters

b) Tricyclic Antidepressants (TCAs)

Amitriptyline

Nortriptyline

Mechanism of Action:

Block reuptake of serotonin and norepinephrine in the brain

Increase their availability at synapses

Result: improved mood + pain modulation (useful in burning mouth syndrome)

3) Sedative / Hypnotics

a) Barbiturates

Phenobarbital (15–30 mg)

Mechanism of Action:

Enhance GABA action by prolonging opening of chloride channels

Stronger CNS depression than benzodiazepines

Result: sedation, sleep induction

b) Benzodiazepines

Diazepam

Lorazepam

Mechanism of Action:

enhance GABA activity

Increase frequency of chloride channel opening

Result: sedation, anxiolysis, muscle relaxation[18]

Dental management

Dental management of stress-related oral disorders requires a comprehensive and multidisciplinary approach that addresses both the oral manifestations and the underlying psychological factors. Initially, a thorough patient evaluation is essential, including medical and dental history, identification of stressors, and assessment of parafunctional habits such as bruxism.[24,26]Patient education and reassurance play a key role in helping individuals understand the relationship between stress and oral health, along with guidance on lifestyle modifications such as adequate sleep, balanced diet, and habit control. Stress management techniques, including relaxation exercises, meditation, and cognitive behavioral therapy, should be encouraged or referred to when necessary.[14,15]Condition-specific treatments are implemented based on clinical presentation, such as occlusal splints for bruxism, physiotherapy and NSAIDs for temporomandibular disorders, topical corticosteroids for aphthous ulcers and oral lichen planus, saliva substitutes for xerostomia, and antidepressants or anxiolytics in cases like burning mouth syndrome. Pharmacological therapy may be used cautiously and often in consultation with a physician. Regular follow-up is crucial to monitor progress, modify treatment, and ensure long-term control, with a

multidisciplinary team involving dentists, psychologists, and physicians contributing to optimal patient outcomes.(30)

CONCLUSION

Stress is an important psychosocial factor that significantly influences oral health, particularly the condition of the oral mucosa. Through complex neuroendocrine and immunological mechanisms, psychological stress can alter the body's normal physiological balance and reduce immune competence. Activation of the hypothalamic–pituitary–adrenal axis and increased secretion of stress hormones such as cortisol can suppress immune responses, impair inflammatory regulation, and delay tissue healing. These biological changes make the oral mucosa more vulnerable to infections, inflammatory conditions, and ulcerative lesions. Furthermore, stress can influence oral health indirectly through behavioral changes such as poor oral hygiene, unhealthy dietary habits, smoking, and bruxism. These behaviors may further compromise the integrity of the oral mucosa and increase susceptibility to oral diseases. Therefore, understanding the psychological component of oral diseases is essential for accurate diagnosis and effective management.Management of stress-related oral mucosal disorders requires a comprehensive and multidisciplinary approach. In addition to conventional dental treatments aimed at controlling symptoms and promoting mucosal healing, psychological interventions play a crucial role in addressing the underlying stress factors. Early identification of psychological stressors, regular dental check-ups, maintenance of good oral hygiene, and adoption of healthy lifestyle practices such as balanced nutrition, adequate sleep, and regular physical activity can help reduce the risk of stress-related oral mucosal conditions.Recognition of the relationship between psychological stress and oral health is essential for dental professionals to provide holistic patient care. By integrating psychological management with conventional dental therapy, clinicians can improve patient outcomes, reduce disease recurrence, and enhance the overall quality of life of affected individuals.

REFERENCES

1. Scully C. Oral and maxillofacial medicine: the basis of diagnosis and treatment. 3rd ed. Edinburgh: Churchill Livingstone; 2013.
2. Neville BW, Damm DD, Allen CM, Chi AC. Oral and maxillofacial pathology. 4th ed. St Louis: Elsevier; 2016.
3. Greenberg MS, Glick M, Ship JA. Burket's oral medicine. 12th ed. Shelton: People's Medical Publishing House; 2015.
4. Regezi JA, Sciubba JJ, Jordan RCK. Oral pathology: clinical pathologic correlations. 7th ed. St Louis: Elsevier; 2017.
5. McCarthy PL, Shklar G. Diseases of the oral mucosa. 2nd ed. Philadelphia: Lea & Febiger; 1980.
6. Porter SR, Scully C. Aphthous ulcers (recurrent). Clin Dermatol. 2000;18(5):569–78.
7. Scully C, Porter S. Oral mucosal disease: recurrent aphthous stomatitis. Br J Oral Maxillofac Surg. 2008;46(3):198–206.
8. Pedersen A. Psychologic stress and recurrent aphthous ulceration. J Oral Pathol Med. 1989;18(2):119–22.
9. Gallo CB, Mimura MAM, Sugaya NN. Psychological stress and recurrent aphthous stomatitis. Clinics (Sao Paulo). 2009;64(7):645–8.

10. Ship JA, Chavez EM, Doerr PA, Henson BS, Sarmadi M. Recurrent aphthous stomatitis. Quintessence Int. 2000;31(2):95–112.
11. Lamey PJ, Lamb AB. Prospective study of aetiological factors in recurrent aphthous stomatitis. Br Med J. 1988;297:1147–9.
12. Soto Araya M, Rojas Alcayaga G, Esguep A. Association between psychological disorders and oral lichen planus. J Oral Pathol Med. 2004;33(5):264–6.
13. Rojo-Moreno JL, Bagan JV, Rojo-Moreno J, Donat JS, Milián MA, Jiménez Y. Psychologic factors and oral lichen planus. J Oral Pathol Med. 1998;27(8):355–60.
14. Koray M, Dülger O, Ak G, Horasanli S, Uçok A, Tanyeri H, et al. The evaluation of anxiety and salivary cortisol levels in patients with oral lichen planus. Oral Dis. 2003;9(6):298–301.
15. Alrashdan MS, Alkhader M. Psychological factors in oral lichen planus. J Oral Pathol Med. 2017;46(6):440–4.
16. Bergdahl M, Bergdahl J. Burning mouth syndrome: prevalence and associated factors. J Oral Pathol Med. 1999;28(8):350–4.
17. Scala A, Checchi L, Montevecchi M, Marini I, Giamberardino MA. Update on burning mouth syndrome. Pain Med. 2003;4(4):297–304.
18. Zakrzewska JM. Burning mouth syndrome. BMJ Clin Evid. 2013;2013:1301.
19. Lamey PJ, Lewis MA. Oral medicine in practice: burning mouth syndrome. Br Dent J. 1989;167:197–200.
20. Epstein JB, Gorsky M, Caldwell J. Fluconazole treatment of candidiasis in patients with burning mouth syndrome. Oral Surg Oral Med Oral Pathol Oral Radiol Endod. 1996;82(6):649–52.
21. Chiappelli F, Cajulis OS. Psychoneuroimmunology: stress and oral health. J Calif Dent Assoc. 2004;32(3):187–95.
22. Genco RJ, Ho AW, Kopman J, Grossi SG, Dunford RG, Tedesco LA. Models to evaluate the role of stress in periodontal disease. Ann Periodontol. 1998;3(1):288–302.
23. Hilgert JB, Hugo FN, Bandeira DR, Bozzetti MC. Stress, cortisol, and oral health. J Dent Res. 2006;85(4):324–8.
24. Balkwill F, Mantovani A. Inflammation and cancer: back to Virchow. Lancet. 2001;357(9255):539–45.
25. Cohen S, Janicki-Deverts D, Miller GE. Psychological stress and disease. JAMA. 2007;298(14):1685–7.
26. Kiecolt-Glaser JK, McGuire L, Robles TF, Glaser R. Psychoneuroimmunology and health. Annu Rev Psychol. 2002;53:83–107.
27. Lamey PJ. Oral medicine in practice: recurrent aphthous ulceration. Br Dent J. 1997;182:144–7.
28. Edgar NR, Saleh D, Miller RA. Recurrent aphthous stomatitis: a review. J Clin Aesthet Dermatol. 2017;10(3):26–36.
29. Rogers RS. Recurrent aphthous stomatitis: clinical characteristics and evidence for immunopathogenesis. J Investig Dermatol Symp Proc. 1997;2(1):7–11.
30. Porter SR, Kirby A, Olsen I, Barrett W. Immunological aspects of recurrent aphthous stomatitis. J Oral Pathol Med. 1993;22(6):227–31.
31. Gurvits GE, Tan A. Burning mouth syndrome. World J Gastroenterol. 2013;19(5):665–72.
32. Field EA, Allan RB. Review article: oral ulceration. Br J Oral Maxillofac Surg. 2003;41(2):85–94.

33. Scully C, Porter SR. The clinical spectrum of recurrent aphthous stomatitis. Oral Surg Oral Med Oral Pathol Oral Radiol Endod. 1997;84(6):610–3.
34. Al-Hashimi I, Schifter M, Lockhart PB, Wray D, Brennan M, Migliorati CA, et al. Oral lichen planus and lichenoid lesions: diagnostic and therapeutic considerations. Oral Surg Oral Med Oral Pathol Oral Radiol Endod. 2007;103:S25.e1–12.
35. Arduino PG, Porter SR. Oral and perioral herpes simplex virus type 1 infection. Oral Dis. 2008;14(8):656–66.
36. Akintoye SO, Greenberg MS. Recurrent aphthous stomatitis. Dent Clin North Am. 2014;58(2):281–97.
37. Locker D, Slade G. Association between psychosocial factors and oral health. Community Dent Oral Epidemiol. 1993;21(1):21–6.
38. Thomson WM, Poulton R, Broadbent JM, Al-Kubaisy S. Stress and oral health outcomes. J Dent Res. 2010;89(11):1231–5.
39. Selye H. Stress without distress. Philadelphia: Lippincott; 1974.