

Stress-Induced Gastritis: A Comprehensive Literature Review on Etiology and Treatment

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ABSTRACT

Gastritis is a common gastrointestinal disorder with multiple etiologies, including infections, medication use, autoimmune conditions, and psychosocial stress. Among these, stress-induced gastritis remains unrecognized despite its increasing incidence in high-stress environments. This condition is characterized by inflammation of the gastric mucosa resulting from physiological or psychological stressors. The current review offers an in-depth examination of the complex pathogenesis of stress-induced gastritis, highlighting key contributing factors such as disruption of the brain-gut axis, oxidative stress, immune system dysregulation, and microbial dysbiosis. Diagnostic approaches are examined, including clinical evaluation, endoscopic assessment, histopathological examination, and the use of inflammatory and oxidative stress biomarkers to differentiate this condition from other forms of gastritis. Current treatment strategies are reviewed, encompassing pharmacological therapies, such as acid-suppressing agents and mucosal protectants, as well as non-pharmacological interventions including stress management, dietary adjustments, and the use of herbal and probiotic therapies. The review also discusses emerging therapeutic directions, particularly antioxidant supplementation and gut microbiota modulation. These insights collectively advocate for an individualized, integrative approach to treatment aimed at improving clinical outcomes. All in all, the present review concludes by identifying key gaps in existing research and advocating for future studies focused on targeted, evidence-based therapies for stress-induced gastritis.

Keywords: Brain-Gut Axis, Gastrointestinal Inflammation, Microbial Dysbiosis, Oxidative Stress, Stress-Induced Gastritis.

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INTRODUCTION

The human digestive system comprises a coordinated network of organs that facilitate the digestion and absorption of nutrients essential for sustaining bodily functions. Among these organs, stomach plays a central role by executing both mechanical and chemical processes to break down ingested food.^{1,2} A vital aspect of gastric activity is the secretion of Hydrochloric Acid (HCl) by the parietal cells lining the stomach wall. This acidic environment not only facilitates protein digestion but also activates digestive enzymes and serves as a defense mechanism against ingested pathogens. The production of gastric acid is tightly regulated by stimulatory signals such as acetylcholine from the parasympathetic nervous system, histamine acting on H₂ receptors, and the hormone gastrin. On the contrary, prostaglandins and somatostatin act as inhibitory agents to maintain gastric acid

homeostasis.³ However, disruption in this balance, such as excessive acid production or compromised mucosal defense, could often contribute to gastrointestinal disorders such as gastritis, peptic ulcer disease, and gastroesophageal reflux.⁴

Among the various etiological factors, psychological and physiological stress is highly recognized as a significant contributor to gastric disorders, significantly influencing Gastrointestinal (GI) function through neurological and hormonal pathways.^{5,6} The connection between the nervous system and the gastrointestinal tract operates as a two-way communication network, primarily facilitated by the gut-brain axis (Figure 1).⁷ The pathophysiology of stress-induced gastritis often involves the gut-brain axis, which links psychological stress to gastric dysfunction. This axis functions as an intricate signaling network involving the nervous, endocrine, and immune systems, enabling two-way communication between the brain and the GI tract. Psychological stress can significantly impair the function of the gut-brain axis, triggering a cascade of physiological responses that negatively impact gastric health. It often results in elevated gastric acid secretion, diminished blood flow to the stomach lining, and increased production of pro-inflammatory cytokines, factors that collectively weaken the protective lining of the



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stomach and heighten its vulnerability to injury. A key mechanism involves the activation of the Hypothalamic-Pituitary-Adrenal (HPA) axis, which elevates cortisol levels and alters autonomic nervous system activity.^{8,9} These changes not only enhance acid secretion, but also compromise mucosal defences and disrupt normal gastric motility. The resulting effects may include delayed gastric emptying, increased intestinal permeability, and symptoms such as bloating, dyspepsia, and gastric discomfort, all of which contribute to the pathogenesis of stress-induced gastric inflammation.¹⁰

As regards stress-induced gastritis, it is a condition characterized by the inflammation of the stomach lining as a result of physiological or psychological stress.¹¹ It arises in response to physiological or psychological stressors, including severe illness, trauma, burns, or surgical procedures.¹² Stress-induced gastritis is distinct from other forms that may be caused by bacterial infections, such as *Helicobacter pylori*, or the long-lasting use of Non-Steroidal Anti-Inflammatory Drugs (NSAIDs). Instead, it arises when the body's response to stress leads to changes in gastric function and mucosal integrity. The inflammatory process can manifest differently among individuals, ranging from mild irritation to severe erosion of the stomach lining. Understanding the specific mechanisms through which stress impacts the gastric environment is essential for accurate diagnosis and effective management.¹³⁻¹⁶

The incidence of stress-induced gastritis is rising, particularly among individuals experiencing high levels of stress in their daily lives.^{17,18} Modern life presents a range of stressors, such as job-related demands, financial pressures, and personal conflicts, which can significantly impact a person's physical health. When these stressors are persistent or intense, they can disrupt the body's stress regulation mechanisms, contributing to the onset of conditions like gastritis. This strong link between chronic stress and gastric inflammation highlights the need to incorporate stress management into both the prevention and treatment of gastritis.

Stress-induced gastritis can manifest as either an acute or chronic condition, each with distinct characteristics and implications for digestive health.^{19,20} Acute gastritis typically develops suddenly in response to a specific stressful event or period, causing immediate symptoms such as abdominal pain, nausea, and vomiting. Chronic gastritis, on the other hand, develops gradually over time due to prolonged exposure to stressors. Chronic gastritis can lead to more persistent symptoms and may increase the risk of developing complications such as peptic ulcers or even gastric cancer if left unmanaged.^{21,22} Therefore, recognizing the specific form of gastritis is critical for tailoring appropriate treatment interventions and promoting long-term digestive well-being.

Diagnosis requires distinguishing stress-induced gastritis from other gastritis types through careful evaluation of patient history

and potential stressors.²³ A thorough clinical assessment should include a detailed account of the patient's lifestyle, including sources of stress, coping mechanisms, and any history of anxiety or depression. Endoscopic examination and biopsy may be necessary to rule out other causes of gastritis, such as *H. pylori* infection or NSAID-induced damage.²⁴ Biomarker analysis, including measurements of inflammatory cytokines and stress hormones, can provide additional support for the diagnosis.²⁵ Differentiating stress-induced gastritis from other forms ensures that patients receive targeted and effective treatment. Understanding the mechanisms and treatments for stress-induced gastritis is crucial for improving patient outcomes. By gaining a deeper insight into how stress affects gastric physiology and the inflammatory processes involved, healthcare professionals can develop more effective strategies for managing this condition. Such insights also support the early identification of individuals at heightened risk for stress-induced gastritis, enabling timely intervention and the implementation of preventive strategies. Improved patient outcomes include reduced symptom severity, enhanced healing of the gastric mucosa, and a decreased risk of complications.

Implementing effective management strategies can significantly reduce the frequency and intensity of symptoms, thereby improving patients' overall quality of life.^{26,27} Stress-induced gastritis can markedly affect daily functioning, and leading to persistent abdominal pain, discomfort, and digestive issues. Targeted interventions, including stress reduction techniques, dietary adjustments, and appropriate pharmacological therapy, can help alleviate these symptoms. As a result, patients often experience improved quality of life, better engagement in daily activities, and enhanced overall well-being.

Accordingly, the present review aims to consolidate current research and provide insights into optimal treatment modalities. The existing literature on stress-induced gastritis is vast and spans multiple disciplines, including gastroenterology, psychology, and nutrition. To aid in conceptual clarity and integration of the multidisciplinary content discussed herein, the authors of the present review provides Table 1 as a synthesized overview of the major mechanisms, diagnostic tools, and therapeutic modalities associated with stress-induced gastritis. All in all, the present review synthesizes this diverse body of research to provide a comprehensive overview of the current understanding of the condition. This review assesses the effectiveness of various treatment strategies, such as lifestyle changes, medications, and natural remedies, with the goal of helping clinicians choose the most suitable and evidence-based options for their patients. Additionally, it highlights existing research gaps and proposes directions for future studies, supporting the continuous advancement of stress-induced gastritis management.

Etiology and Pathophysiology of Stress-Induced Gastritis

The Role of the Gut-Brain Axis

Psychological stress can disrupt normal GI function via the brain-gut axis, a complex bidirectional communication system linking the Central Nervous System (CNS) with the GI tract.²⁸ This sophisticated network integrates neural, hormonal, and immune pathways, enabling continuous interaction and feedback between the brain and gut. Stress initiates a cascade of physiological responses that directly affect the GI system, potentially causing functional and inflammatory disorders such as gastritis. The gut-brain axis involves intricate interplay among the nervous, immune, and endocrine systems.^{29,30} The nervous system component includes the Enteric Nervous System (ENS), often called as the "second brain," which independently regulates GI functions while communicating with the CNS via the vagus nerve.³¹ The immune system is essential for maintaining gut homeostasis, and stress-induced disruption can lead to inflammation. Meanwhile, the endocrine system releases hormones like cortisol that directly impact the gastric mucosa and alter gut motility and secretion. Together, these interactions contribute to the onset and progression of stress-induced gastritis.

Stress hormones, such as cortisol, can directly affect gastric mucosa and promote inflammation.³² Cortisol is a glucocorticoid hormone produced by the adrenal glands in response to stress. Elevated levels of cortisol can have several detrimental effects on the gastric mucosa, including increasing gastric acid secretion, reducing mucosal blood flow, and suppressing the release of protective prostaglandins. These changes compromise the integrity of the gastric lining, making it more vulnerable to damage and inflammation. Additionally, cortisol can modulate the immune response in the gut, promoting the release of pro-inflammatory cytokines that exacerbate gastritis.

Impact of Stress on Gastric Physiology

Stress increases gastric acid production, which can irritate and erode the stomach lining. When the body is under stress, the parasympathetic nervous system, which typically supports digestion and relaxation, may become suppressed, while the sympathetic nervous system becomes more active. This shift can lead to an increase in gastric acid secretion, as the vagus nerve, which stimulates acid production, becomes overactive. The excess acid can overwhelm the stomach's protective mechanisms, causing irritation and erosion of the mucosal lining. It also reduces gastric blood flow, impairing the mucosal defence mechanisms and cellular regeneration. Stress-induced activation of the sympathetic nervous system can lead to vasoconstriction, reducing blood flow to the gastric mucosa. Reduced blood flow impairs the delivery of oxygen and nutrients necessary for cellular repair and regeneration, weakening the mucosal defence mechanisms. This increases the vulnerability of the stomach

lining to damage from gastric acid, pepsin, and other irritants. Additionally, reduced blood flow impairs the secretion of mucus and bicarbonate, key components that safeguard the gastric epithelium.³³

These physiological alterations heighten the risk of developing gastritis and peptic ulcers. The simultaneous increase in gastric acid secretion and decrease in mucosal blood flow creates a harmful environment that promotes gastric inflammation and mucosal injury. Gastritis, marked by inflammation of the stomach lining, can advance to ulcer formation when the damage becomes extensive and persistent. Peptic ulcers are open lesions that may lead to severe pain, bleeding, and other serious complications. Therefore, managing stress and implementing appropriate medical interventions are essential for the prevention and treatment of stress-induced gastric conditions.³⁴

Involvement of Inflammatory Pathways

Stress initiates the activation of inflammatory pathways, leading to the release of pro-inflammatory cytokines such as interleukins and TNF- α , which play a significant role in tissue inflammation and gastrointestinal disturbances.³⁵ Under stressful conditions, immune cells are stimulated to release these cytokines, which are signaling molecules that regulate and propagate inflammatory responses. These mediators exert both local and systemic effects, contributing to the development of various stress-related conditions, including gastritis. While the activation of inflammatory pathways is a natural part of the body's response, persistent or chronic activation can have harmful consequences, particularly damaging the gastric mucosa.³⁶

Vital cytokines such as IL-1 β and TNF- α contribute to gastric mucosal damage and inflammation.³⁷ Interleukin-1 β (IL-1 β) and Tumor Necrosis Factor-alpha (TNF- α) are potent pro-inflammatory cytokines that play a significant role in the development and progression of gastritis.³⁸ These cytokines can enhance the permeability of the gastric mucosa, enabling harmful substances such as acid and digestive enzymes to infiltrate the epithelial barrier and exacerbate tissue damage. They also promote the release of additional inflammatory mediators, establishing a positive feedback loop that intensifies and sustains the inflammatory response. By targeting these key cytokines, therapeutic interventions can help reduce gastric mucosal damage and inflammation.

These inflammatory responses intensify the harmful effects of elevated gastric acid secretion and diminished mucosal blood flow. Together, these factors, excess acid, impaired circulation and the release of pro-inflammatory cytokines, create a hostile environment for the gastric mucosa. The resulting inflammation further weakens the integrity of the stomach lining, increasing its vulnerability to acid and other irritants. This sets off a vicious cycle of inflammation, tissue damage, and impaired healing, ultimately contributing to the development and persistence of

chronic gastritis.³⁹ Effectively managing these inflammatory processes is crucial to breaking this cycle and supporting mucosal repair and recovery.

Oxidative Stress and Gastric Mucosal Damage

Generation of Reactive Oxygen Species (ROS)

Stress-induced gastritis is closely associated with elevated production of ROS in the gastric mucosa.⁴⁰ ROS are highly reactive oxygen-containing molecules that can damage essential cellular components. Although these species are normally generated as by-products of cellular metabolism, their levels can increase significantly under conditions of stress and inflammation. In the context of gastritis, excessive ROS generation overwhelms the endogenous antioxidant defence systems, resulting in oxidative stress.⁴¹ These reactive molecules can cause oxidative damage to lipids, proteins, and DNA. Due to their high reactivity, ROS interact with and modify various cellular macromolecules, leading to lipid peroxidation, protein oxidation, and DNA damage (Figure 2). Lipid peroxidation disrupts membrane integrity, protein oxidation impairs enzymatic activity, and DNA damage may result in mutations and cellular dysfunction. These cumulative effects significantly contribute to the pathogenesis of gastritis.⁴² Oxidative stress plays a central role in the initiation and progression of gastritis by promoting inflammation, weakening mucosal defences, and inducing cellular injury. The imbalance between ROS production and antioxidant capacity perpetuates the inflammatory response and compromises the integrity of the gastric mucosa. Therefore, addressing oxidative stress is a critical component in the management of stress-induced gastritis.

Impairment of Antioxidant Defence Mechanisms

Chronic stress significantly impairs the body's natural antioxidant defence systems, reducing their capacity to neutralize ROS.⁴³ The body relies on both enzymatic and non-enzymatic antioxidants to counteract oxidative damage. Key enzymatic antioxidants include Superoxide Dismutase (SOD), catalase, and glutathione peroxidase, while non-enzymatic antioxidants comprise compounds like glutathione, Vitamin C, Vitamin E, and various polyphenols. Under chronic stress, these antioxidant systems become depleted or functionally impaired. In particular, critical enzymes such as SOD and glutathione peroxidase are often down-regulated.⁴⁴ SOD catalyzes the conversion of harmful superoxide radicals into hydrogen peroxide and oxygen, while glutathione peroxidase reduces hydrogen peroxide to water using glutathione as a cofactor. These enzymes are essential for maintaining redox homeostasis and protecting cells from oxidative injury. Chronic stress suppresses their expression and enzymatic activity, weakening the body's ability to manage ROS.⁴⁵

As a result, the imbalance between ROS production and antioxidant capacity leads to oxidative stress, causing the accumulation of ROS that can damage cellular structures and

impair mucosal defence. This oxidative injury compromises the gastric lining, contributing to inflammation and the development of gastritis. Restoring antioxidant balance is critical for reversing mucosal damage and promoting healing.

Consequences of Oxidative Damage

Oxidative stress can initiate both apoptosis and necrosis in gastric epithelial cells. Apoptosis, or programmed cell death, is a controlled mechanism for removing damaged cells, whereas necrosis is a more abrupt and pathological form of cell death resulting from injury. When oxidative stress becomes excessive, it triggers these processes, leading to the loss of protective epithelial cells in the stomach lining.⁴⁶ This cellular loss undermines the structural integrity of the mucosa and fosters the development of gastritis and ulcers.

Moreover, oxidative damage compromises the gastric mucosal barrier, increasing its permeability and promoting inflammation.⁴⁷ The mucosal barrier, composed of mucus and bicarbonate secretions, serves to protect the stomach lining from acid and digestive enzymes. Damage to the cells that maintain this barrier allows irritants such as gastric acid and pepsin to infiltrate and inflame the tissue, exacerbating mucosal injury.

Further, prolonged oxidative stress perpetuates inflammation and tissue damage, potentially leading to chronic gastritis and increasing the risk of gastric ulcer formation.⁴⁸ These ulcers can cause significant discomfort, bleeding, and complications if left untreated. Therefore, addressing oxidative stress through targeted antioxidant therapies and lifestyle changes is essential to prevent disease progression and support mucosal recovery.

Role of Microbial Dysbiosis in Stress-Induced Gastritis

Alterations in Gastric Microbiota Composition

Stress can disrupt the composition and diversity of the gastric microbiota, resulting in dysbiosis, a microbial imbalance that may compromise mucosal integrity and contribute to the development or exacerbation of gastritis.⁴⁹ Gastric microbiota is nothing but a group of microorganisms present in the stomach, which take a pivotal responsibility of maintaining gastric health. Stress disrupts the delicate balance within this microbial community, thereby leading to changes in the types and numbers of bacteria present. This disruption, known as dysbiosis, can have significant implications for gastric function and inflammation. Factors such as changes in gastric acid secretion, immune function, and lifestyle habits contribute to these alterations.

This dysbiosis is often characterized by a decline in beneficial bacterial populations and a concurrent rise in pathogenic species, which can impair mucosal defences and promote inflammation.⁵⁰ Under normal conditions, the gastric microbiota includes a diverse array of beneficial bacteria that support

digestion, immune regulation, and mucosal defence. Stress can lead to a decrease in these beneficial bacteria, reducing their protective effects. At the same time, there may be a proliferation of pathogenic species, including certain bacterial that trigger inflammation and contribute to damage of the gastric mucosa. This shift in microbial composition exacerbates the inflammatory process and contributes to the development of gastritis.

Changes in microbial populations can exacerbate inflammation and impair gastric function.⁵¹ The altered microbial populations can produce metabolites that promote inflammation and disrupt normal gastric function. For example, certain bacteria can increase the production of ammonia, which can damage the gastric mucosa. Dysbiosis can also disrupt the production of Short-Chain Fatty Acids (SCFAs), which play a vital role in maintaining gut health, supporting the integrity of mucosal barrier and reducing inflammation. Collectively, these changes exacerbate the severity of gastritis and compromise the stomach's normal physiological functions.^{52,53}

Impact on Gastric Homeostasis

The gastric microbiota plays a pivotal role in maintaining gastric homeostasis and modulating immune responses. A balanced microbial community supports gastric function by regulating acid secretion, stimulating mucus production, and reinforcing the integrity of the mucosal barrier. Additionally, the microbiota communicates with the immune system, fostering immune tolerance and preventing excessive inflammatory responses to dietary and environmental antigens.^{54,55}

Dysbiosis, an imbalance in the microbial composition disrupts these protective functions. A decline in beneficial microbes and a concurrent rise in pathogenic species contribute to heightened mucosal inflammation and weakened defence mechanisms. This imbalance impairs mucus and bicarbonate secretion, compromising the mucosal barrier and increasing vulnerability to gastric injury.^{56,57}

Moreover, dysbiosis and impaired homeostasis create a self-perpetuating cycle: inflammation exacerbates microbial imbalance, and altered microbiota further fuel inflammation and tissue damage.⁵⁸ Interventions aimed at restoring microbial balance, such as the use of probiotics and tailored dietary strategies, are therefore essential for re-establishing gastric homeostasis and mitigating the progression of stress-induced gastritis.

Contribution to Inflammation and Mucosal Damage

The altered gastric microbiota can produce harmful metabolites that exacerbate inflammation and contribute to mucosal injury. Under normal conditions, beneficial bacteria generate metabolites like Short-Chain Fatty Acids (SCFAs), which promote mucosal health and reduce inflammation.⁵⁹ Nonetheless, dysbiosis favours the proliferation of pathogenic species that secrete

pro-inflammatory compounds capable of disrupting epithelial integrity and triggering cytokine release.⁶⁰

Furthermore, certain bacterial strains produce mucin-degrading enzymes, such as mucinases, which erode the protective mucus layer. The degradation of this layer exposes the gastric epithelium to acidic and enzymatic injury, amplifying inflammation and mucosal damage.^{61,62} Maintaining the integrity of the mucus barrier is thus critical to preventing gastritis.

The combined impact of microbial imbalance, inflammatory metabolite production, and mucus layer degradation intensifies gastritis severity. This hostile gastric environment leads to persistent inflammation, epithelial damage, and impaired gastric function. Addressing these microbial disturbances through targeted therapeutic strategies is vital for controlling stress-induced gastritis and promoting mucosal repair.

DIAGNOSTIC APPROACHES FOR STRESS-INDUCED GASTRITIS

Clinical Evaluation and Patient History

Diagnosis of stress-induced gastritis begins with a comprehensive clinical evaluation, which includes a detailed patient history emphasizing psychological stressors and life style factors. The step includes gathering information about the onset, duration, and severity of symptoms, as well as any associated factors that may be contributing to the condition. A detailed patient history is crucial for identifying potential stressors and differentiating stress-induced gastritis from other forms of gastric inflammation.^{63,64}

Identifying potential stressors and their correlation with symptom onset is crucial. Determining the presence and nature of stressors in the patient's life is a key component of the clinical evaluation. This includes exploring psychological stressors, such as work-related stress, financial difficulties, and relationship problems, as well as physiological stressors, such as chronic illness, sleep deprivation, and poor diet. Establishing the temporal link between stressors and onset of symptoms can offer vital insights into the contribution of stress to the development of gastritis.^{65,66} Evaluating psychological factors like anxiety and depression can support the diagnosis as these conditions are closely associated with stress-induced gastritis. These conditions can exacerbate the body's stress response and contribute to the dysregulation of the brain-gut axis. Screening patients for symptoms of anxiety and depression can provide additional support for the diagnosis of stress-induced gastritis and help guide the selection of appropriate treatment strategies.⁶⁷

Endoscopic Examination and Biopsy

Endoscopy enables direct visualization of the gastric mucosa to assess inflammation and tissue damage. This diagnostic procedure involves inserting a thin, flexible tube equipped with a camera

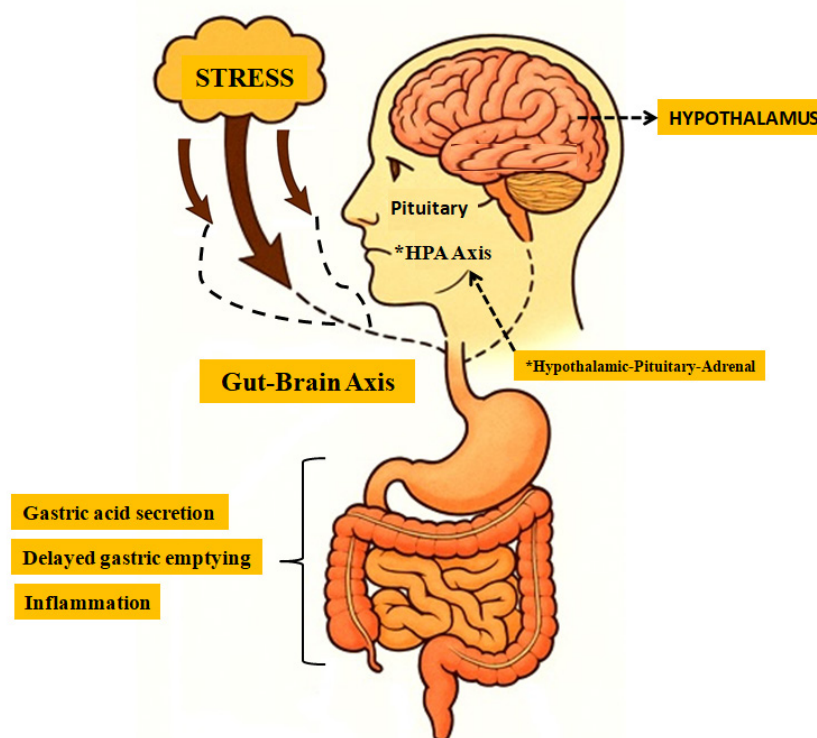


Figure 1: Gut-brain axis - stress induced gastritis.⁷

through the esophagus, stomach and duodenum, allowing the physician to examine the lining for inflammation, erosion, and other abnormalities. Endoscopic examination is a critical tool for evaluating gastritis and distinguishing stress-induced cases from other types.⁶⁸⁻⁷⁰

During endoscopy, biopsy samples can be collected from the gastric mucosa for histological analysis. These samples are examined microscopically to identify histological changes such as inflammation, oedema, and epithelial injury.⁷¹ Histological evaluation helps confirm gastritis, exclude conditions like *H. pylori* infection or gastric cancer, and contributes to differentiating stress-induced gastritis from other forms.⁷² Although endoscopic and histological findings provide important insights into gastritis severity, they cannot definitively identify stress-induced cases. However, the absence of *H. pylori* infection combined with specific histological features may indicate a stress-related cause. Accurate diagnosis and management require integrating these findings with the patient's clinical history and additional diagnostic tests.

Biomarker Analysis

Measuring biomarkers, such as inflammatory cytokines (IL-1 β , TNF- α) and oxidative stress markers (ROS, MDA), can aid in diagnosing stress-induced gastritis.⁷³ Biomarkers serve as measurable indicators of biological processes. In this context, they provide objective evidence of inflammation, oxidative stress, and related pathophysiological changes.⁷⁴

Quantifying inflammatory cytokines like IL-1 β and TNF- α helps assess the level of gastric mucosal inflammation. Likewise, evaluating oxidative stress markers such as ROS and MDA offers insights into the extent of oxidative damage.^{75,76} In addition, assessing antioxidant enzyme levels, including Superoxide Dismutase (SOD) and glutathione peroxidase, provides a broader understanding of oxidative stress status. Low activity of these enzymes may reflect compromised antioxidant defences and increased vulnerability to oxidative injury.⁷⁷

Biomarkers offer objective, quantifiable data that complement subjective clinical assessments. They assist in determining disease severity, monitoring therapeutic responses, and identifying patients at higher risk of developing stress-induced gastritis, thereby guiding preventive strategies. Although biomarker analysis does not replace clinical evaluation or endoscopy, it provides valuable supplementary information for diagnosis and management.

Lifestyle Modifications for Managing Stress-Induced Gastritis

Stress Management Techniques

Incorporating stress reduction strategies such as meditation, yoga, and deep breathing exercises can significantly alleviate symptoms of stress-induced gastritis by addressing stress at its core. Meditation involves focusing the mind on a specific object, thought, or activity to achieve mental clarity and emotional calm. Yoga blends physical postures, breathing exercises, and

meditation to foster relaxation and decrease stress levels. Deep breathing techniques, like diaphragmatic breathing, activate the parasympathetic nervous system, which helps counterbalance the body's stress response.⁷⁸⁻⁸⁰

Cognitive-Behavioural Therapy (CBT) is another effective tool that assists individuals in managing stress by identifying and altering negative thought patterns and behaviours. Through CBT, people develop coping skills to handle stressors more effectively, reducing the physical and psychological impact of stress. This therapy targets the psychological roots of stress, offering lasting relief from symptoms associated with stress-induced gastritis.^{81,82}

Together, these approaches help regulate the brain-gut axis, the complex communication system between the brain and gastrointestinal tract. By lowering stress hormone levels and promoting relaxation, they restore balance in this axis, leading to reduced gastric acid secretion, improved blood flow to the gastric mucosa, and decreased inflammation. These physiological changes contribute to the relief of gastritis symptoms.

Dietary Adjustments

Modifying the diet is a key strategy in managing stress-induced gastritis. Avoiding foods that trigger gastric irritation such as spicy, acidic, and fatty foods can help soothe the stomach lining. Spicy foods contain capsaicin, which can increase acid production and irritate the gastric mucosa. Acidic foods like citrus fruits and tomatoes can directly inflame the stomach lining. Fatty foods tend to slow gastric emptying, prolonging acid exposure and discomfort. Steering clear of these foods can significantly reduce irritation and ease symptoms.^{83,84}

Eating smaller, more frequent meals also helps by regulating gastric acid production. Large meals can prompt excessive acid secretion, worsening gastritis symptoms. Frequent smaller meals maintain steadier acid levels and prevent overproduction, while also supporting stable blood sugar levels, which can lessen stress and enhance overall well-being.⁸⁵

Moreover, including anti-inflammatory foods, which are rich in omega-3 fatty acids and antioxidants further supports healing. Omega-3s, found in fatty fish, flaxseeds, and walnuts, have strong anti-inflammatory effects. Antioxidants present in fruits, vegetables, and whole grains neutralize free radicals, protecting the gastric mucosa from oxidative damage. A diet rich in these nutrients promotes mucosal healing and helps reduce symptom severity.^{86,87}

Regular Physical Activity

Engaging in regular exercise is an effective way to lower stress and improve overall health. Physical activity, especially aquatic exercise, helps reduce stress hormone levels, enhance mood, and promote relaxation. It also improves sleep quality, which is often disrupted by stress. By lowering stress and boosting well-being,

exercise can ease symptoms of stress-induced gastritis.^{88,89} Exercise triggers the release of endorphins, the natural mood enhancers and pain relievers produced by the body. These chemicals help reduce stress and improve emotional well-being, which is particularly helpful for those whose gastritis symptoms worsen with stress or mood disturbances.^{90,91} Additionally, regular physical activity supports digestive health by enhancing gastric motility and reducing constipation. It also has systemic anti-inflammatory effects, including in the gastric mucosa. By promoting healthy digestion and reducing inflammation, exercise plays a valuable role in managing stress-induced gastritis.^{92,93}

Pharmacological Treatments for Stress-Induced Gastritis

Acid-Suppressing Medications

Proton Pump Inhibitors (PPIs), for instance Omeprazole reduces gastric acid production, promoting healing of the gastric mucosa. PPIs function by inhibiting the enzyme responsible for gastric acid production in the stomach. By lowering stomach acid levels, PPIs help alleviate gastritis symptoms and support the healing of the gastric mucosa. PPIs are among the most commonly prescribed medications for gastritis and peptic ulcers.^{94,95}

H2 receptor antagonists such as ranitidine also decrease acid secretion, though they are generally less potent than PPIs.⁹⁶ H2 receptor antagonists (or H2 blockers), are another set of agents help in reducing gastric acid secretion. They act by blocking the action of histamine on histamine H2 receptors in the stomach, that stimulates acid production. While H2 receptor antagonists are effective at reducing acid secretion, they are generally less potent than PPIs and may not provide as much symptom relief.^{97,98}

These medications provide symptomatic relief and prevent further mucosal damage. By reducing gastric acid secretion, PPIs and H2 receptor antagonists can provide significant symptomatic relief from gastritis, including heartburn, abdominal pain, and nausea. These medications also help protect the gastric mucosa from further damage by decreasing its exposure to acid and other irritants. This protective effect promotes healing and lowers the risk of complications like peptic ulcers.

Mucosal Protective Agents

Sucralfate creates a protective coating over the ulcerated mucosa, shielding it from acid and digestive enzymes. Sucralfate is a medication that works by creating a protective barrier over the ulcerated mucosa in the stomach. This barrier shields the ulcer from acid, enzymes, and other irritants, allowing it to heal. Sucralfate also promotes the production of prostaglandins, which plays a key role in protecting the gastric mucosa.^{99,100}

Bismuth subsalicylate possesses anti-inflammatory and antimicrobial properties, providing additional protection. Bismuth subsalicylate is another medication that can provide

protection to the gastric mucosa. The anti-inflammatory properties help reduce pain and inflammation. Additionally, it has antimicrobial property, that helps preventing the stomach infection. Bismuth subsalicylate is used for treating gastritis, peptic ulcers, and other digestive disorders.¹⁰¹⁻¹⁰³ These agents enhance mucosal defence mechanisms and support healing. Mucosal protective agents work by enhancing the natural defence mechanisms of the gastric mucosa and supporting the healing process. By creating a protective barrier, enhancing prostaglandin production, and reducing inflammation, these agents help restore the stomach lining's integrity and relieve gastritis symptoms.

Anti-inflammatory Drugs

These drugs may be used to reduce inflammation and alleviate pain associated with gastritis.¹⁰⁴ However, they must be used cautiously, as some anti-inflammatory medications can worsen gastritis. The decision to prescribe anti-inflammatory drugs should be personalized, carefully balancing potential benefits against risks. NSAIDs are commonly used to relieve pain and inflammation, should generally be avoided in patients with gastritis because they can irritate the gastric mucosa and increase the risk of gastritis and peptic ulcers.^{105,106} Therefore, NSAIDs should only be used under specific medical guidance only. Alternately, selective COX-2 inhibitors, a subclass of NSAIDs that selectively block the COX-2 enzyme involved in inflammation, may be considered with caution. These agents are thought to cause less gastric irritation than traditional NSAIDs, but they still carry some risk. Therefore, their use in patients with gastritis should be carefully evaluated, weighing the potential benefits against possible adverse effects.^{107,108}

Natural and Herbal Remedies for Stress-Induced Gastritis

Cinnamomum cassia

Cinnamomum cassia, commonly known as Chinese cinnamon, is derived from the bark of the *C. cassia* tree and has been used in traditional medicine for centuries due to its wide-ranging health benefits, including the alleviation of gastrointestinal disorders.^{109,110} The above spice exhibits notable anti-inflammatory and gastroprotective effects, making it a promising natural remedy for managing stress-induced gastritis. The active compound cinnamaldehyde plays a key role by modulating the activity of nuclear factor-kappa B (NF-κB), a crucial transcription factor involved in inflammatory responses.¹¹¹ By inhibiting NF-κB, cinnamaldehyde reduces the production of pro-inflammatory cytokines, thereby alleviating inflammation within the gastric mucosa.^{112,113}

Studies have demonstrated that *C. cassia* extract protects against gastric ulcers caused by various factors, including stress, ethanol, and NSAIDs.^{114,115} Its gastroprotective effects are attributed to the enhancement of mucosal defence mechanisms, reduction in

gastric acid secretion, and inhibition of harmful bacterial growth. These properties highlight *C. cassia* as a valuable natural option for the prevention and treatment of stress-induced gastritis.

Quercus infectoria

Quercus infectoria Olivier galls, produced by wasp infestations on the Aleppo oak tree, possess strong antioxidant and anti-inflammatory properties, making them beneficial in treating gastritis.¹¹⁶ These galls have been used since ages for their various therapeutic properties, including its potential to treat inflammatory conditions. The antioxidant and anti-inflammatory properties of *Q. infectoria* galls are a promising natural remedy for managing gastritis. The extract ameliorates changes in gastric pH and protects against ethanol-induced ulceration.^{117,118} Studies have shown that extracts from *Q. infectoria* galls can ameliorate changes in gastric pH and protect against ethanol-induced ulceration in rats. The extract has been found to reduce gastric acidity, increase mucus production, and inhibit the formation of ulcers. These findings suggest that *Q. infectoria* galls may have gastro protective effects that can help prevent and treat gastritis.

Its effects are comparable to those of Omeprazole, a common medication for gastritis. Research has indicated that the gastro-protective effects of *Quercus infectoria* gall extract are comparable to those of Omeprazole, a commonly prescribed medication for gastritis.¹¹⁷ This suggests that *Q. infectoria* galls may be a natural alternative to Omeprazole for subjects intending to manage gastritis symptoms.¹¹⁹

Other Herbal Remedies

Certain herbal formulas, such as Yeokwisan (YWS), have shown potential in relieving functional dyspepsia symptoms and protecting against gastritis. YWS is a standardized herbal formula used in traditional Korean medicine to relieve symptoms of functional dyspepsia. YWS shows anti-inflammatory and gastro-protective properties, making it a potential remedy for managing gastritis.¹²⁰ Prickly pear cactus (*Opuntia ficus indica*) fruit juice can protect against stress-induced gastric lesions, providing a natural source of antioxidants and anti-inflammatory compounds. This cactus fruit juice contains betanin, an antioxidant that offers protection from oxidative damage in the gastric mucosa. Studies have shown that prickly pear cactus fruit juice can protect against stress-induced gastric lesions.¹²¹

These remedies often work by reducing inflammation, enhancing mucosal defence, and providing antioxidant support, addressing multiple aspects of the pathogenesis of stress-induced gastritis. By reducing inflammation, enhancing mucosal defence, and providing antioxidant support, these remedies alleviate symptoms and heals individuals with stress-induced gastritis. They can be incorporated into a comprehensive treatment plan alongside lifestyle modifications and additional therapies.

Emerging Therapies and Future Directions

Probiotics and Gut Microbiota Modulation

Probiotics offer a promising strategy for managing stress-induced gastritis by restoring a healthy gut microbiota, reducing inflammation, and improving gastric function. Probiotics are live microorganisms that, when consumed in adequate amounts, provide health benefits to the host. They help reestablish the balance of gut microbiota, which can be disrupted by stress and other factors.^{122,123} Specific probiotic strains, particularly those belonging to *Lactobacillus* and *Bifidobacterium*, have demonstrated beneficial effects in stress-induced gastritis by providing targeted support to restore microbial balance and reduce inflammation. These strains exhibit anti-inflammatory properties and protect the gastric mucosa from injury, while also enhancing digestion and nutrient absorption.^{124,125} For severe cases of dysbiosis, Faecal Microbiota Transplantation (FMT) is an emerging treatment modality that involves transferring fecal material from a healthy donor to a recipient to restore gut microbiota balance. This technique has shown promise in treating various gastrointestinal disorders, including inflammatory bowel disease.¹²⁶⁻¹²⁸

Antioxidant Therapies

Antioxidant supplementation, including agents like vitamin E and α -lipoic acid, can reduce oxidative stress, protect the gastric mucosa from damage, and promote healing.¹²⁹ Antioxidants function by neutralizing free radicals, which otherwise damage cellular components and exacerbate inflammation. Vitamin E

mitigates oxidative stress and inflammation by modulating the Nrf2 and NF- κ B signaling pathways, thus providing a targeted approach to reduce inflammation and enhance mucosal repair.¹³⁰ Nrf2 is a transcription factor regulating antioxidant gene expression, while NF- κ B controls pro-inflammatory gene expression. By influencing these pathways, vitamin E effectively diminishes oxidative damage and inflammation.¹³¹ α -Lipoic acid activates the Nrf2/HO-1 pathway, which suppresses pro-inflammatory cytokine production and protects the gastric mucosa. HO-1 is an enzyme critical for cellular defence against oxidative injury. Activation of the Nrf2/HO-1 axis by α -lipoic acid therefore contributes to reduced inflammation and mucosal protection.¹³²

Novel Drug Targets

Ongoing research into the molecular mechanisms underlying stress-induced gastritis holds promise for identifying new drug targets, which could lead to the development of more effective and precise therapies. Understanding these molecular pathways is essential for pinpointing specific therapeutic targets, potentially resulting in treatments with improved efficacy and fewer adverse effects.¹³³ Both inflammatory pathways and the brain-gut axis are pivotal in the pathogenesis of stress-induced gastritis. Targeting these systems may offer innovative treatment options, providing new avenues to manage the condition and improve patient outcomes.^{134,135} However, these potential targets require further validation through preclinical and clinical studies. Continued research efforts are crucial to translate these findings into effective therapies for stress-induced gastritis.

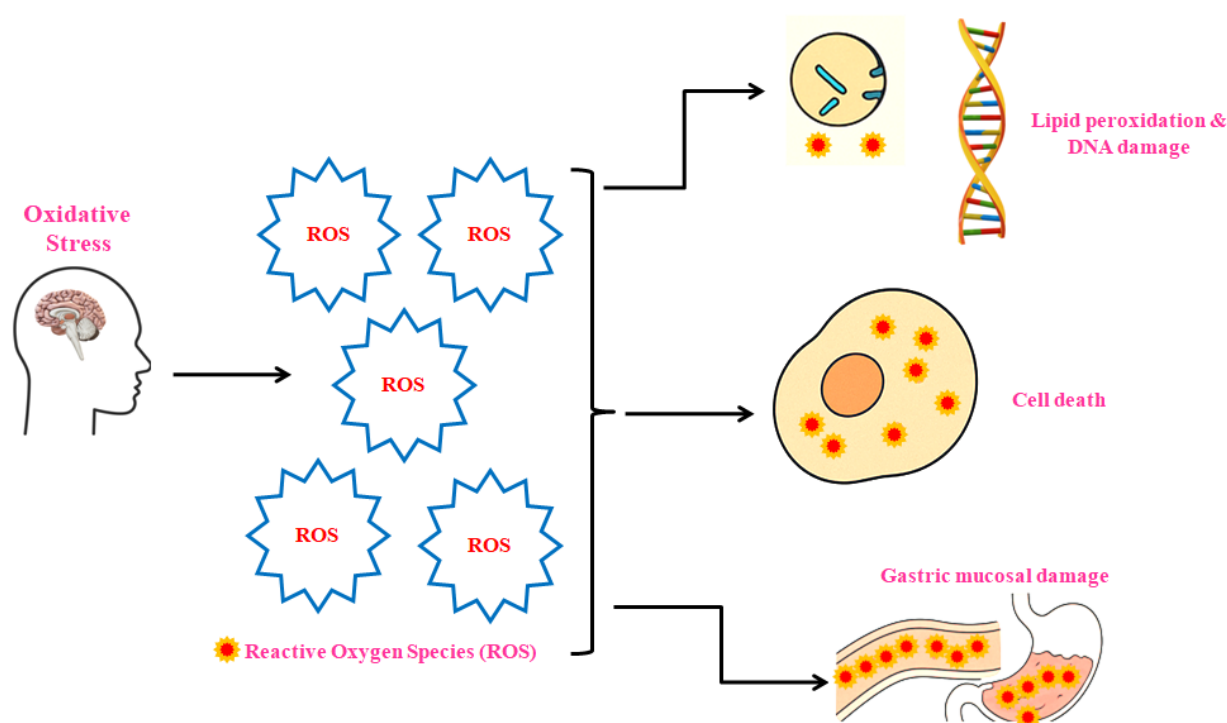


Figure 2: Impact of oxidative stress.⁴⁰

Table 1: Summary of key aspects in stress-induced gastritis.

Key Aspects	Key Components	Mechanism / Role	Clinical Relevance / Intervention
Etiology	Psychological & physiological stress	Activates the HPA axis, leads to increased cortisol levels	Triggers gastric dysfunction through the brain-gut axis
Pathophysiology	Brain-Gut Axis	Vagus nerve and HPA axis dysregulation	Increases gastric acid secretion, reduces mucosal blood flow and mucosal defense, and enhances GI permeability
	Oxidative stress	Overproduction of Reactive Oxygen Species (ROS) coupled with a reduction in antioxidant enzymes, such as Superoxide Dismutase (SOD) and Glutathione Peroxidase (GPx)	Leads to gastric mucosal injury, apoptosis, and necrosis
	Inflammation	Overproduction of pro-inflammatory cytokines, for instance, Interleukin-1-Beta (IL-1 β) and Tumor Necrosis Factor-Alpha (TNF- α); Nuclear Factor kappa B (NF- κ B) activation	Sustains chronic inflammation and damages the gastric lining
	Microbial dysbiosis	Reduction of beneficial gut microbes alongside an overgrowth of harmful pathogens	Disrupts mucosal integrity, promotes inflammation, and impairs immune regulation
Diagnosis	Clinical history	Evaluation of lifestyle factors, psychological and physiological stressors	Helps identify the stress-related origin of symptoms
	Endoscopy & Biopsy	Direct visualization of gastric mucosa and histological assessment	Confirms inflammation and excludes other causes like H. pylori infection
	Biomarkers	Measurement of cytokines (IL-1 β , TNF- α), oxidative stress markers (Reactive Oxygen Species (ROS), Malondialdehyde (MDA), and antioxidant enzyme levels SOD, GPx)	Offers objective evidence supporting diagnosis and helps monitor treatment response
Treatment	Pharmacological	Proton pump inhibitors, H2 blockers, sucralfate, bismuth compounds	Reduce gastric acid, protect mucosa, and promote healing
	Anti-inflammatory agents	Selective COX-2 inhibitors (used cautiously)	May relieve symptoms; NSAIDs are contraindicated
	Lifestyle modifications	Stress reduction techniques (e.g., yoga, Cognitive Behavioral Therapy (CBT)), dietary changes, regular physical activity	Alleviate stress impact, enhance digestive health, and improve quality of life
	Natural remedies	<i>Cinnamomum cassia</i> , <i>Quercus infectoria</i> , Yeokwisan formula	Provide anti-inflammatory and antioxidant support; enhance mucosal defense

Key Aspects	Key Components	Mechanism / Role	Clinical Relevance / Intervention
Emerging Therapies	Probiotics	Specific strains of <i>Lactobacillus</i> and <i>Bifidobacterium</i>	Restore microbial balance and reduce gastric inflammation
	Antioxidants	Vitamin E, α -lipoic acid	Modulate Nrf2/HO-1 and NF- κ B pathways; reduce oxidative stress
	Novel targets	Brain-gut axis modulators, cytokine inhibitors	Represents future personalized treatment strategies

CONCLUSION

Stress-induced gastritis is a multifaceted condition involving the brain-gut axis, oxidative stress, and microbial dysbiosis, all contributing to its pathogenesis. The complex interplay among these factors highlights the importance of a comprehensive management approach. A clear understanding of each component is essential for developing effective treatments. Options such as lifestyle modifications, pharmacological therapies, and natural remedies can help manage symptoms and support healing. Lifestyle changes, including stress reduction techniques and dietary adjustments, can lessen the body's stress response. Pharmacological agents, like acid suppressants and mucosal protectants, aid in symptom relief and mucosal repair. Additionally, natural remedies such as herbal formulations and antioxidant supplements offer supplementary benefits. Emerging therapies like probiotics and antioxidant supplementation show promise by restoring gut microbiota balance and reducing oxidative damage, potentially improving patient outcomes.

In clinical practice, it is vital for clinicians to recognize stress as a primary cause of gastritis, particularly in patients experiencing high levels of stress. Accurate diagnosis and effective management depend on acknowledging the role of stress in disease development. Clinicians should evaluate patients for stress-related factors and incorporate stress management into treatment plans. A holistic approach that combines lifestyle changes, medications, and natural remedies provides the most comprehensive care. This approach addresses the multiple contributing factors, stress, inflammation, and microbial imbalance, for ensuring better support for patients. Personalized treatment strategies tailored to each patient's unique circumstances, including psychological support, are crucial for achieving optimal results.

Looking ahead, further research is necessary to fully unravel the molecular mechanisms underlying stress-induced gastritis, laying the groundwork for targeted, more effective therapies with fewer side effects. Clinical trials are needed to validate emerging treatments such as probiotics and antioxidant supplementation, providing evidence-based guidance for their use. These studies should focus on symptom reduction, mucosal healing, and quality-of-life improvements. Moreover, understanding the long-term effects of various treatments on gastric health remains critical. Existing data on the sustained safety and efficacy of

pharmacological and non-pharmacological interventions is limited. Future research should evaluate how these therapies influence gastric function, mucosal inflammation, and microbial balance over extended periods. Such insights are essential for developing safe, effective, and personalized therapies that not only relieve symptoms but also maintain long-term gastrointestinal health.

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ABBREVIATIONS

GI: Gastrointestinal; **CNS:** Central Nervous System; **ENS:** Enteric Nervous System; **HPA:** Hypothalamic-Pituitary-Adrenal; **IL-1 β :** Interleukin-1 beta; **TNF- α :** Tumor Necrosis Factor-alpha; **ROS:** Reactive Oxygen Species; **MDA:** Malondialdehyde; **SOD:** Superoxide Dismutase; **GPx:** Glutathione Peroxidase; **NSAIDs:** Non-Steroidal Anti-Inflammatory Drugs; **SCFAs:** Short-Chain Fatty Acids; **FMT:** Faecal Microbiota Transplantation; **PPIs:** Proton Pump Inhibitors; **CBT:** Cognitive-Behavioural Therapy; **NF- κ B:** Nuclear Factor kappa-light-chain-enhancer of activated B cells; **Nrf2:** Nuclear Factor Erythroid 2-Related Factor 2; **HO-1:** Heme Oxygenase-1; **GERD:** Gastroesophageal Reflux Disease.

CONFLICT OF INTEREST

The authors declare that there are no conflict of interest.

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