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Morphological Alterations Of Gastric Tissue Under Experimental Stress Conditions

Adxamjanova Saida Saidvaqqos qizi
U. M. Mirsharapov
Tashkent state medical university

Article History	Abstract
Received: 14th February, 2026 Accepted: 11th March, 2026	Stress is a critical physiological factor that significantly affects the structure and function of the gastrointestinal system, particularly the stomach. Experimental models of stress have demonstrated that prolonged exposure to physical or psychological stressors can induce notable morphological and histopathological changes in gastric tissues. The present study aims to investigate the structural alterations of the gastric mucosa under experimentally induced stress conditions and to evaluate the extent of tissue damage at both macroscopic and microscopic levels. In this research, experimental stress was induced using controlled laboratory models designed to simulate acute and chronic stress conditions. Gastric tissue samples were collected and subjected to detailed histological examination using standard staining techniques. Morphological parameters such as epithelial integrity, glandular structure, mucosal thickness, vascular changes, and inflammatory infiltration were systematically analyzed. The findings reveal that stress exposure leads to significant disruption of the gastric mucosal barrier. Observed changes include epithelial desquamation, degeneration of gastric glands, submucosal edema, and increased infiltration of inflammatory cells. Additionally, vascular congestion and microhemorrhages were identified as key features associated with stress-induced damage. These alterations suggest that stress not only compromises the protective mechanisms of the gastric mucosa but also predisposes the tissue to ulcerative processes. Furthermore, the study highlights the role of stress-related hormonal and neurogenic factors in mediating these morphological changes. Elevated levels of glucocorticoids and activation of the autonomic nervous system appear to contribute to reduced mucosal resistance and impaired regenerative capacity.

In conclusion, experimental stress conditions induce profound morphological changes in gastric tissue, emphasizing the importance of stress management in preventing gastrointestinal pathologies. The results of this study provide new insights into the pathogenesis of stress-related gastric disorders and may contribute to the development of targeted therapeutic strategies.

Keywords: Experimental stress, gastric mucosa, morphological changes, histopathology, gastrointestinal system, epithelial damage, inflammation, stress-induced injury, gastric glands, mucosal barrier

Introduction

Stress is an inevitable component of modern life and has become a major factor affecting human health. It is widely acknowledged that both acute and chronic stress can significantly disrupt physiological homeostasis, leading to pathological changes in various organ systems. Among these, the gastrointestinal system is particularly vulnerable due to its close interaction with the neuroendocrine and immune systems. The stomach, as a central organ of digestion, plays a crucial role in maintaining gastrointestinal integrity and is highly sensitive to stress-related stimuli.

The relationship between stress and gastric pathology has been extensively studied, with evidence indicating that stress is a key contributor to the development of gastric mucosal injury, including erosions, hemorrhages, and ulcers. Experimental and clinical studies have demonstrated that stress can impair mucosal defense mechanisms, alter gastric secretion, and disturb microcirculation within the gastric wall. These changes collectively compromise the protective barrier of the gastric mucosa, increasing susceptibility to damage.

From a physiological perspective, stress activates multiple regulatory pathways, most notably the hypothalamic–pituitary–adrenal (HPA) axis and the autonomic nervous system. Activation of these systems results in the release of stress hormones such as glucocorticoids and catecholamines, which exert profound effects on gastric function. Elevated glucocorticoid levels have been shown to inhibit epithelial cell proliferation, reduce mucus and bicarbonate secretion, and delay mucosal healing. Simultaneously, sympathetic nervous system activation can lead to vasoconstriction and reduced gastric blood flow, thereby promoting ischemic conditions within the tissue.

In addition to neuroendocrine mechanisms, stress also influences immune responses within the gastric mucosa. It has been reported that stress can trigger inflammatory processes characterized by increased infiltration of immune cells, including neutrophils and lymphocytes. This inflammatory response further exacerbates tissue injury by releasing cytokines, reactive oxygen species, and

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proteolytic enzymes, all of which contribute to cellular damage and structural disorganization.

Despite the substantial body of research on stress-induced gastric injury, there remains a need for detailed morphological investigations that clearly describe structural alterations at the tissue level under controlled experimental conditions. Many previous studies have focused primarily on biochemical or functional changes, while comprehensive histopathological evaluations are relatively limited. Understanding the precise morphological changes is essential for elucidating the mechanisms underlying stress-related gastric disorders and for developing effective preventive and therapeutic strategies.

Experimental models of stress provide a valuable tool for studying these processes in a controlled environment. Such models allow for the systematic assessment of structural changes in gastric tissue and enable the identification of key pathological features associated with different types and durations of stress exposure. Morphological analysis, particularly through histological examination, offers direct insight into tissue integrity, cellular organization, and pathological alterations.

Therefore, the present study aims to investigate the morphological changes in gastric tissue under experimentally induced stress conditions. By analyzing histological features such as epithelial integrity, glandular structure, mucosal thickness, vascular alterations, and inflammatory infiltration, this study seeks to provide a comprehensive understanding of the structural impact of stress on the stomach. The findings are expected to contribute to the growing body of knowledge on stress-related gastrointestinal pathology and to support future research in this field.

Materials and Methods

This study was designed as a controlled experimental investigation aimed at evaluating morphological changes in gastric tissue under conditions of induced stress. Adult male Wistar rats (Rattus norvegicus), weighing 180–220 g and aged 8–10 weeks, were selected due to their established suitability for stress-related gastrointestinal research. The animals were obtained from a certified laboratory facility and acclimatized for one week prior to the experiment under standard laboratory conditions, including a controlled temperature of 22 ± 2°C, relative humidity of 50–60%, and a 12-hour light/dark cycle. During the entire experimental period, animals were provided with standard pellet diet and water ad libitum, and their health status was continuously monitored to ensure stability and exclude external influencing factors. After acclimatization, the animals were randomly divided into two groups: a control group, which was maintained under normal conditions without exposure to stress, and a stress group, which was subjected to experimentally induced stress protocols.

Experimental stress was induced using a standardized immobilization (restraint) method, widely recognized for its ability to reproduce both physical and psychological stress responses. In this procedure, animals were placed individually in restraining devices that limited movement without causing direct physical harm. The stress protocol consisted of daily immobilization for three hours over a continuous period of ten days, a duration sufficient to activate neuroendocrine stress pathways, including the hypothalamic–pituitary–adrenal axis and the sympathetic nervous system. These physiological responses are associated with increased secretion of glucocorticoids and catecholamines, which are known to influence gastric mucosal integrity. Animals in the control group were handled similarly each day to eliminate handling bias but were not exposed to immobilization stress.

At the conclusion of the experimental period, all animals were fasted for twelve hours with free access to water to ensure uniform gastric conditions prior to tissue collection. Euthanasia was performed humanely using an overdose of intraperitoneal sodium pentobarbital in accordance with established ethical guidelines. Following euthanasia, the abdominal cavity was opened through a midline incision, and the stomach was carefully excised. The organ was immediately examined macroscopically for visible pathological changes, including hemorrhagic spots, erosions, or ulcerative lesions. Subsequently, the stomach was opened along the greater curvature and gently rinsed with cold physiological saline to remove residual contents. Tissue samples were obtained from the glandular region of the stomach, as this area is particularly sensitive to stress-induced damage.

The collected tissue specimens were fixed in 10% neutral buffered formalin for 24–48 hours to preserve morphological integrity. Following fixation, samples were processed using standard histological techniques, including dehydration through graded alcohol series, clearing in xylene, and embedding in paraffin wax. Paraffin blocks were sectioned at a thickness of 4–5 µm using a rotary microtome, and the sections were mounted on glass slides. For morphological evaluation, the sections were stained with hematoxylin and eosin, allowing detailed visualization of cellular and tissue structures. Histological examination was performed under a light microscope, focusing on key parameters such as epithelial integrity, organization of gastric glands, mucosal thickness, presence of submucosal edema, vascular alterations, and degree of inflammatory cell infiltration. The observed morphological changes in the stress group were systematically compared with those in the control group to assess the extent and severity of stress-induced gastric damage. Semi-quantitative evaluation methods were applied where appropriate to enhance the objectivity of the analysis, ensuring that the findings were both reliable and scientifically robust.

Results

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The histological and morphological evaluation of gastric tissues demonstrated significant differences between the control and stress-exposed groups. In the control group, the gastric mucosa exhibited normal histoarchitecture, characterized by an intact epithelial layer, well-organized gastric glands, and uniform mucosal thickness. No evidence of edema, vascular abnormalities, or inflammatory cell infiltration was observed, indicating preserved structural integrity.

In contrast, the stress-exposed group showed marked pathological alterations. The surface epithelium exhibited clear signs of desquamation and degeneration, reflecting disruption of the mucosal barrier. Gastric glands appeared disorganized, with areas of dilation and partial structural destruction. Submucosal edema was consistently observed, contributing to tissue swelling and distortion of normal architecture. In addition, vascular congestion and focal microhemorrhages were evident, indicating impaired blood circulation. A significant increase in inflammatory cell infiltration, predominantly neutrophils and lymphocytes, was also noted.

To quantitatively summarize these findings, the main morphological parameters were evaluated and compared between the groups, as presented in **Table 1**.

Table 1. Morphological changes in gastric tissue under experimental stress

Morphological Parameter	Control Group	Stress Group
Epithelial integrity	Normal	Disrupted / Desquamation
Gastric gland structure	Organized	Degenerated / Disorganized
Mucosal thickness	Normal	Reduced
Submucosal edema	Absent	Present
Vascular congestion	Absent	Moderate to severe
Microhemorrhages	Absent	Present
Inflammatory infiltration	Minimal	Marked

The table clearly demonstrates that all evaluated pathological features were significantly more pronounced in the stress group compared to the control group, confirming the damaging effect of stress on gastric tissue morphology.

Representative histological findings are illustrated in **Figure 1**, which provides visual confirmation of the observed structural differences between groups.

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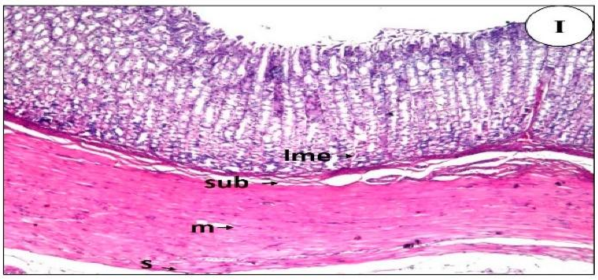


Figure 3. I. Showing a normal histological structure of the lining mucosal epithelium (lme) of the stomach with underlying glands and submucosa (sub), muscularis propria (m), and serosa (s) in the control rats group without any degenerative or necrotic changes. H&E × 16.

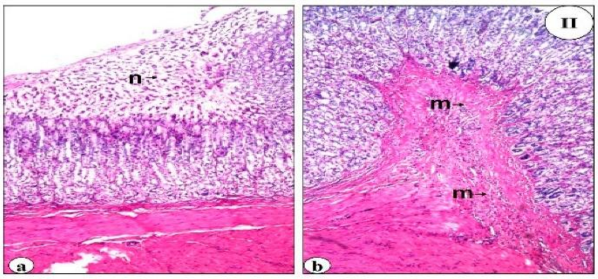


Figure 3. II-a. Showing focal necrosis (n) and desquamation of the lining mucosa of rat stomach, II-b: showing focal inflammatory cells infiltration (m) in submucosa and muscularis in indomethacin treated group (H&E × 16).

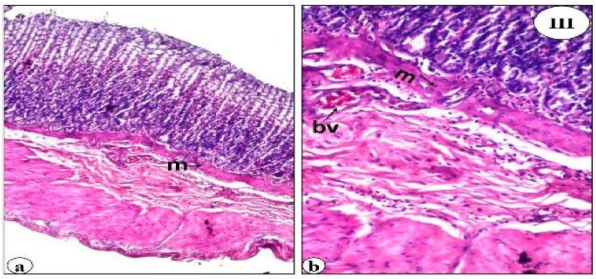


Figure 3. III-a. Showing mild congestion in blood vessels with few focal inflammatory cells infiltration (m) in submucosa (H&E ×16), III-b: indicating the magnification (×40) of Figure III-a to identify the few focal inflammatory cells infiltration in submucosa; also mild congestion of blood vessels (bv) in submucosa were observed in SP-treated group (H&E × 16).

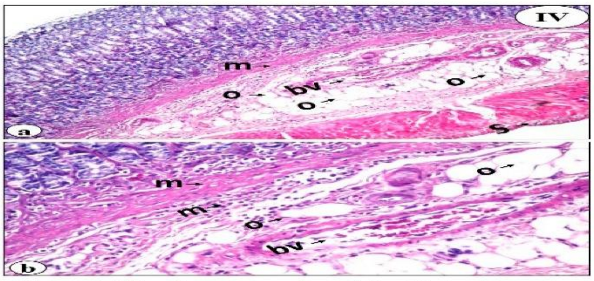
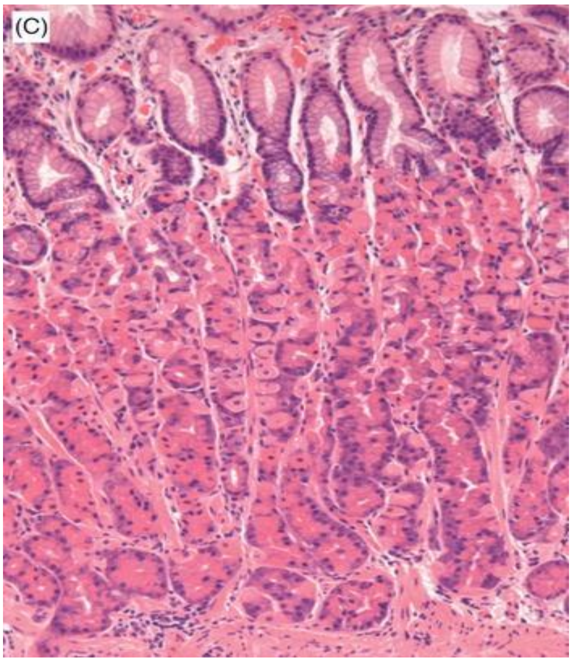
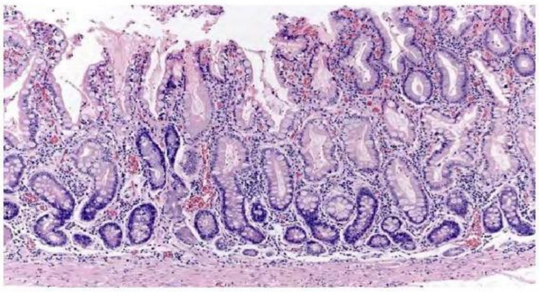
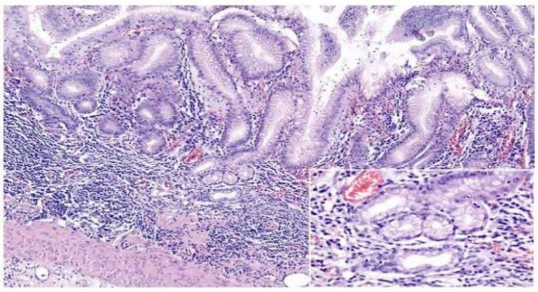


Figure 3. IV-a. Showing oedema (o) with focal inflammatory cells infiltration (m) and congestion of blood vessels (bv) in submucosa, IV-b: showing the magnification (×40) of Figure IVa to identify oedema; focal inflammatory cells infiltration with congestion of blood vessels in submucosa and oedema in muscularis (O) in KF-treated group (H&E × 16).





(a)



(b)

Figure 1. Representative histological sections of gastric mucosa stained with hematoxylin and eosin (H&E). The normal control tissue shows intact epithelial

lining and well-organized gastric glands, while stress-exposed tissue demonstrates epithelial desquamation, glandular degeneration, submucosal edema, vascular congestion, and inflammatory infiltration.

Conclusion

In conclusion, the findings of the present study demonstrate that experimental stress exerts a profound negative impact on gastric tissue morphology. The observed structural alterations, including epithelial disruption, glandular degeneration, submucosal edema, vascular congestion, and inflammatory infiltration, indicate a clear breakdown of the gastric mucosal defense system under stress conditions. These changes not only compromise the integrity of the gastric lining but also create a favorable environment for the development of more severe pathological conditions, such as erosions and ulcerative lesions.

The results highlight the critical role of neuroendocrine and inflammatory mechanisms in mediating stress-induced gastric damage. Activation of the hypothalamic–pituitary–adrenal axis, increased secretion of stress hormones, impaired microcirculation, and enhanced inflammatory responses collectively contribute to the progression of morphological injury. Importantly, this study provides detailed histopathological evidence supporting the concept that stress is a key etiological factor in gastrointestinal disorders.

From a clinical and scientific perspective, these findings emphasize the importance of early prevention and effective management of stress to protect gastric health. Furthermore, the study contributes to a deeper understanding of the structural basis of stress-related gastric diseases and may serve as a foundation for future research aimed at developing targeted therapeutic interventions.

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