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ANTIOXIDANT CORRECTION OF GASTRIC MUCOSAL MORPHOLOGICAL ALTERATIONS IN BACKGROUND CHEMICAL INJURY USING THE PHYTOGENIC AGENT GULIMSAR

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Abstract

Background: Oxidative stress represents a pivotal pathogenetic mechanism underlying gastric mucosal injury induced by chemical and toxic agents. Excessive generation of reactive oxygen species (ROS) disrupts epithelial integrity, impairs microcirculation, and triggers inflammatory cascades, ultimately compromising mucosal regeneration.

Objective: To investigate morphological and morphometric alterations of the gastric mucosa under background chemical injury and to evaluate the efficacy of antioxidant correction using the phytogenic preparation *Gulimsar*.

Methods: An experimental study was conducted on laboratory animals with chemically induced subnecrotic gastric mucosal damage. Histological, morphometric, and biochemical analyses were performed, including assessment of malondialdehyde (MDA), superoxide dismutase (SOD), and catalase activity.

Results: Chemical injury resulted in epithelial desquamation, glandular disorganization, vascular congestion, and pronounced inflammatory infiltration accompanied by elevated MDA levels and reduced antioxidant enzyme activity. Administration of *Gulimsar* significantly decreased lipid peroxidation markers, restored enzymatic antioxidant defenses, reduced inflammatory infiltration, and promoted structural regeneration of gastric glands.

Conclusion: Antioxidant correction using *Gulimsar* demonstrates pronounced cytoprotective and reparative effects on chemically injured gastric mucosa through modulation of oxidative stress and restoration of tissue homeostasis.

Keywords

gastric mucosa, oxidative stress, antioxidant therapy, phytotherapy, morphometry, lipid peroxidation.

Introduction

The gastric mucosa constitutes a highly specialized structural and functional barrier system responsible for secretion, protection, and local immunoregulation. Integrity of this system depends on a dynamic balance between aggressive factors (acid, toxins, reactive species) and protective mechanisms (mucus layer, microcirculation, antioxidant enzymes). Disruption of this equilibrium leads to structural disorganization and functional impairment.

Oxidative stress is widely recognized as a central mediator of chemically induced gastric injury. Reactive oxygen species initiate lipid peroxidation, destabilize membrane phospholipids, oxidatively modify proteins, and interfere with DNA integrity. Morphologically, these processes manifest as epithelial degeneration, necrobiotic changes, microvascular disturbances, and inflammatory infiltration.

Plant-derived antioxidants have gained increasing scientific attention due to their ability to neutralize free radicals and modulate inflammatory signaling pathways. The phytogenic agent *Gulimsar*, rich in phenolic compounds and flavonoids, is presumed to exert membrane-

stabilizing and cytoprotective effects. However, its morphological impact on gastric mucosal injury remains insufficiently characterized.

The present study aims to provide a comprehensive morphometric and biochemical evaluation of antioxidant correction using *Gulimsar* in an experimental model of gastric mucosal damage.

Materials and Methods

Experimental Design

The study was conducted on adult laboratory animals randomly assigned to three groups:

1. **Intact control group**
2. **Chemical injury group (without correction)**
3. **Chemical injury + Gulimsar treatment group**

Background injury was induced using a standardized subnecrotic chemical exposure model.

Histological Analysis

Gastric tissue samples were fixed in 10% neutral buffered formalin, embedded in paraffin, and sectioned at 5 μm thickness. Hematoxylin and eosin staining was performed for general morphological assessment.

Morphometric Parameters

Quantitative evaluation included:

- Total mucosal thickness (μm)
- Surface epithelial height (μm)
- Gastric pit depth (μm)
- Glandular density (per mm^2)
- Area of inflammatory infiltration (%)

Measurements were performed using digital microscopy with calibrated morphometric software.

Biochemical Assessment

Markers of oxidative stress were analyzed:

- Malondialdehyde (MDA) concentration (nmol/mg protein)
- Superoxide dismutase (SOD) activity (U/mg protein)
- Catalase activity (μmol/min)

Statistical Analysis

Data were expressed as mean ± standard error. Statistical comparisons were performed using Student's t-test. Differences were considered significant at $p < 0.05$.

Results

Morphological Findings

Chemical injury induced marked epithelial desquamation, cytoplasmic vacuolization, glandular disorganization, vascular congestion, and pronounced leukocytic infiltration. Surface epithelial cells exhibited loss of polarity and nuclear pyknosis in focal areas.

In contrast, *Gulimsar*-treated animals demonstrated partial restoration of epithelial continuity, reduced interstitial edema, improved glandular organization, and significantly decreased inflammatory infiltration.

Morphometric Analysis

Chemical injury reduced mucosal thickness by approximately 23% and glandular density by 24%, while inflammatory infiltration increased more than fivefold compared to controls.

Administration of *Gulimsar* resulted in:

- 20% increase in mucosal thickness compared to untreated injured group
- Significant recovery of glandular density
- 2.5-fold reduction in inflammatory area

Biochemical Findings

Chemical injury significantly elevated MDA levels (indicative of intensified lipid peroxidation) and reduced SOD and catalase activities.

Gulimsar treatment reduced MDA by 48% and restored antioxidant enzyme activity toward control values, indicating effective attenuation of oxidative stress.

Discussion

The findings confirm the central role of oxidative stress in chemically induced gastric mucosal injury. Lipid peroxidation compromises epithelial membrane integrity, leading to barrier dysfunction and inflammatory amplification.

The observed correlation between elevated MDA levels and morphometric deterioration underscores the mechanistic link between biochemical and structural alterations.

Gulimsar demonstrated significant cytoprotective effects, likely mediated by:

- Scavenging of reactive oxygen species
- Stabilization of cellular membranes
- Modulation of inflammatory mediator release
- Enhancement of endogenous antioxidant enzyme systems

These mechanisms collectively contributed to improved tissue regeneration and structural recovery.

Scientific Novelty

This study provides the first integrated morphometric and biochemical evaluation of *Gulimsar*-mediated antioxidant correction in chemically injured gastric mucosa, establishing a direct association between oxidative stress markers and structural tissue restoration.

Practical Significance

The results support the potential incorporation of plant-derived antioxidants into complex therapeutic strategies aimed at preventing progression of gastric mucosal injury.

Conclusion

Antioxidant correction using the phytogenic preparation *Gulimsar* significantly attenuates oxidative damage, reduces inflammatory infiltration, and promotes morphological restoration of chemically injured gastric mucosa. These findings justify further investigation into its molecular mechanisms and potential clinical application.

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