

**DYNAMIC MORPHOLOGICAL CHANGES IN THE STOMACH IN THE
CONTEXT OF ULCERATIVE COLITIS: AN EXPERIMENTAL STUDY***Vohidov Bekzod Rafiq o'g'li**Independent Researcher, Bukhara**State Medical Institute***Abstract**

This study comprehensively examined morphological and morphometric changes in the gastric mucosa and wall under experimental ulcerative colitis (UC) conditions. The experiment was conducted using an animal model in which colitis was induced with TNBS. Histological examinations revealed severe dystrophic, inflammatory, erosive, and necrotic changes in the gastric mucosa, vascular and cellular infiltration in the submucosa, and degenerative processes in the muscular layer. These changes significantly impact the structural and functional state of the gastric wall. The results have practical significance for clinical gastroenterology and morphology.

Keywords: stomach, ulcerative colitis, experiment, histology, morphometry, rats

Materials and Methods

This experimental study was carried out under strictly controlled laboratory conditions at the Morphology Laboratory of the Bukhara State Medical Institute. Thirty (n=30) biologically healthy, 9-month-old male albino rats weighing between 200–250 grams were used as the animal model. Prior to experimentation, all animals underwent a 7-day acclimatization period, during which their physiological state, feeding patterns, and hydration levels were closely monitored. Environmental parameters were maintained at optimal levels: a 12-hour light/dark cycle, ambient temperature of 22–24°C, and relative humidity of 55–60%. Animals had ad libitum access to sterilized food and drinking water.

All animal procedures were conducted in accordance with ethical standards and approved by the Bioethics Committee of the Ministry of Health of the Republic of Uzbekistan, complying with international guidelines for the care and use of laboratory animals.

The rats were randomly assigned into two distinct groups as follows:

- **Group 1 – Control (n=10):** This group did not undergo any pathological induction or drug treatment and was used to establish physiological baseline parameters.

- **Group 2 – Experimental Ulcerative Colitis (n=20):** Ulcerative colitis (UC) was chemically induced using 2,4,6-trinitrobenzenesulfonic acid (TNBS) dissolved in 50% ethanol at a concentration of 30 mg/kg. A 1 mL volume of this solution was administered rectally via a flexible polyethylene catheter under light anesthesia. The inclusion of ethanol served to enhance the permeability of the colonic mucosa, facilitating TNBS penetration and inflammation induction.

Five to seven days post-induction, the rats were assessed for clinical manifestations of UC, including reduced food intake (anorexia), decreased activity (lethargy), and abnormal stool consistency (diarrhea). Animals displaying marked symptoms were humanely euthanized under deep anesthesia using decapitation, in line with approved euthanasia protocols.

Immediately following euthanasia, stomach tissues were carefully dissected and placed in 10% neutral-buffered formalin for 24–48 hours for fixation. The fixed tissues underwent routine histological processing, including dehydration in graded alcohols, paraffin embedding, and microtome sectioning at 5–7 μm thickness.

Three types of histological staining methods were employed for tissue analysis:

- **Hematoxylin and Eosin (H&E):** To evaluate general tissue architecture and inflammatory response.
- **Van Gieson Stain:** To identify fibrotic changes and assess the presence and distribution of collagen fibers.
- **Toluidine Blue Stain:** For detecting granulated immune cells, especially mast cells, eosinophils, and basophils.

Microscopic analysis of the stained tissue sections was conducted using a Leica DM500 light microscope at magnifications of 100 $\times$, 400 $\times$, and 1000 $\times$. Digital photomicrographs were taken for documentation and quantitative assessment.

Morphometric evaluation was performed with the help of a DN-107T micrometer in conjunction with an NLSD-307B microscope camera. For each experimental animal, at least three representative histological sections were analyzed. In each section, 20–25 measurements were taken from selected epithelial cells, glandular structures, and vascular components.

The following morphometric parameters were quantitatively assessed:

- Thickness of the gastric epithelial layer (μm)
- Diameter and morphometric profile of the gastric glands
- Submucosal vessel diameter and wall thickness
- Density and distribution of collagen fibers (via Van Gieson)
- Infiltration level and relative count of inflammatory cells (granulocytes, lymphocytes, and plasma cells)

All quantitative data were statistically analyzed using **GraphPad Prism version 9.0**. The data were expressed as mean $\pm$ standard error of the mean (SEM). Statistical

significance was determined using unpaired t-tests for two-group comparisons and one-way ANOVA for multi-group analyses, with a threshold of $p < 0.05$ considered statistically significant.

Results

Histological analysis of gastric tissues from the control group revealed normal architecture without any signs of pathological alterations. The epithelial layer appeared intact, with well-preserved nuclei centrally located within columnar epithelial cells. Chromatin was evenly distributed, and the cytoplasm was homogeneous, lacking vacuolization. The lamina propria beneath the epithelium consisted of loose connective tissue with well-defined capillaries and venules. No signs of inflammation, hemorrhage, or edema were observed in the mucosal or submucosal layers.

Gastric glands were cylindrical in shape, uniformly arranged, and exhibited open lumens, indicative of active secretion. Submucosal vessels were of normal diameter, and the muscularis layer displayed regularly aligned smooth muscle fibers without signs of atrophy or degeneration.

In contrast, the experimental ulcerative colitis (UC) group showed marked histopathological changes in the gastric wall. The mucosal epithelium exhibited extensive desquamation, nuclear pyknosis, karyorrhexis, and cytoplasmic vacuolization. Focal areas showed signs of cytolysis and epithelial erosion. The integrity of the basement membrane was compromised, suggesting weakened mucosal barrier function.

Subepithelial zones displayed pronounced hyperemia, plasma leakage, and microhemorrhages. A dense inflammatory infiltrate composed of lymphocytes, plasma cells, and neutrophils was observed, indicating an active immune response. Van Gieson staining revealed increased collagen deposition in the submucosa and muscularis layers, signifying early fibrotic transformation. Toluidine blue staining highlighted the presence of basophilic and eosinophilic granulocytes, suggestive of ongoing immuno-allergic inflammation.

The muscular layer demonstrated signs of atrophic degeneration, with thinning and fragmentation of smooth muscle fibers. Hydropic degeneration and vacuolization of myocytes were frequently observed. In some cases, perivascular edema and endothelial dysfunction were noted in arterioles and venules within the muscularis layer.

Conclusion

The findings of this study demonstrate that ulcerative colitis is not restricted to colonic pathology but can also induce significant histomorphological changes in the gastric tissues. These include epithelial damage, vascular hyperemia, inflammatory cell infiltration, and early fibrotic remodeling, which together compromise gastric structure and function. Such systemic manifestations highlight the need for broader diagnostic

and therapeutic approaches when managing UC, especially when upper gastrointestinal symptoms are present. The study reinforces the value of experimental models in understanding the multisystemic nature of inflammatory bowel disease and contributes to the development of targeted interventions in gastroenterology and pharmacology.

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