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**МОРФОФУНКЦИОНАЛЬНЫЕ ХАРАКТЕРИСТИКИ
РЕМОДЕЛИРОВАНИЯ ЭПИТЕЛИАЛЬНОЙ ТКАНИ ПОД
ВЛИЯНИЕМ ПАТОГЕННЫХ МИКРООРГАНИЗМОВ:
ГИСТОЛОГИЧЕСКАЯ И МИКРОБИОЛОГИЧЕСКАЯ
ПЕРСПЕКТИВА**

Аннотация: Эпителиальные ткани служат основным барьером между организмом-хозяином и патогенными микроорганизмами, играя важнейшую роль как в защитной функции, так и в иммуномодуляции. В данной статье рассматриваются гистологические изменения, наблюдаемые в эпителиальных клетках после воздействия патогенных бактерий и вирусов, включая нарушение структуры межклеточных контактов, перестройку цитоскелета и морфологические изменения ядра. Анализ 124 гистологических препаратов от пациентов с подтвержденными инфекциями слизистых оболочек выявил отчетливые паттерны ремоделирования эпителия, в том числе нарушение стратификации, гиперплазию бокаловидных клеток и деградацию базальной мембраны.

Ключевые слова: гистология, эпителиальная ткань, патогенные микроорганизмы, морфофункциональные изменения, ремоделирование ткани

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**MORPHOFUNCTIONAL CHARACTERISTICS OF EPITHELIAL
TISSUE REMODELING UNDER THE INFLUENCE OF PATHOGENIC**

MICROORGANISMS: A HISTOLOGICAL AND MICROBIOLOGICAL PERSPECTIVE

Abstract: Epithelial tissues serve as the primary interface between the host organism and pathogenic microorganisms, playing a critical role in both barrier defense and immune modulation. This article examines the histological alterations observed in epithelial cells following exposure to pathogenic bacteria and viruses, including structural disorganization of intercellular junctions, cytoskeletal rearrangement, and nuclear morphological changes. Analysis of 124 histological specimens from patients with confirmed mucosal infections revealed distinct patterns of epithelial remodeling, including stratification disruption, goblet cell hyperplasia, and basement membrane degradation.

Keywords: histology, epithelial tissue, pathogenic microorganisms, morphofunctional changes, tissue remodeling

INTRODUCTION

Epithelial tissues represent one of the most widely distributed and functionally diverse tissue types in the human organism. Lining the external surfaces of the body as well as the internal luminal surfaces of organs, epithelia form indispensable mechanical and biochemical barriers against environmental challenges, including pathogenic microorganisms. The structural integrity of these barriers is maintained through a complex system of intercellular junctions — tight junctions, adherens junctions, desmosomes, and gap junctions — which collectively regulate paracellular permeability, mechanotransduction, and cellular signaling [1].

Pathogenic bacteria and viruses have evolved sophisticated mechanisms to subvert epithelial defenses, including the secretion of proteolytic enzymes that degrade extracellular matrix components, expression of adhesins that promote colonization, and production of toxins that disrupt cytoskeletal architecture [2].

Despite extensive research on individual pathogen-epithelium interactions, comparative analysis of histological remodeling patterns across different classes of pathogens — particularly in the context of mucosal infection — remains insufficiently characterized.

Understanding the tissue-level consequences of these interactions is essential for developing targeted diagnostic criteria and therapeutic interventions that preserve or restore epithelial barrier function [3].

MATERIALS AND METHODS

The study was conducted on the basis of the Department of Histology and Biology, Fergana Medical Institute of Public Health, Fergana, Uzbekistan, during the period 2022–2024. A total of 124 histological specimens were collected from patients (mean age 34.7 ± 11.2 years; 58 male, 66 female) with clinically and microbiologically confirmed mucosal infections. Biopsy material was obtained from the respiratory (n=42), gastrointestinal (n=51), and urogenital (n=31) mucosae.

Tissue specimens were fixed in 10% buffered formalin for 24 hours, dehydrated in ascending alcohol concentrations, cleared in xylene, and embedded in paraffin. Sections of 5–7 μm thickness were prepared using a Leica RM2255 rotary microtome. Standard hematoxylin and eosin (H&E) staining was applied to all specimens for general morphological assessment. Periodic acid-Schiff (PAS) staining was employed to evaluate goblet cell secretory activity and basement membrane integrity. Immunohistochemical detection of E-cadherin (clone 36, 1:200, Dako) and ZO-1 (polyclonal, 1:150, Abcam) was performed to assess adherens junction and tight junction status, respectively [4].

RESULTS AND DISCUSSION

Histological examination of all 124 specimens revealed identifiable epithelial alterations compared to control tissue sections obtained from non-infected individuals. The nature and severity of these alterations varied

significantly across the three pathogen categories ($p < 0.01$ for all principal morphometric parameters).

In specimens infected with Gram-positive bacteria (predominantly *Staphylococcus aureus* and *Streptococcus pyogenes*), H&E sections demonstrated marked neutrophilic infiltration of the lamina propria, focal erosions of the surface epithelium, and pronounced intracellular edema. Mean epithelial layer thickness was significantly reduced ($148.3 \pm 22.6 \mu\text{m}$ vs. $212.4 \pm 18.1 \mu\text{m}$ in controls; $p < 0.001$). Immunohistochemical labeling for E-cadherin showed focal discontinuities at the lateral membrane domains, consistent with disruption of adherens junctions. Goblet cell density was moderately increased ($18.7 \pm 4.3 \text{ cells/mm}^2$) relative to controls ($11.2 \pm 2.8 \text{ cells/mm}^2$), suggesting a compensatory secretory response [5].

RNA viral infections (including influenza A, respiratory syncytial virus, and norovirus) produced a distinct pattern of epithelial remodeling characterized predominantly by intracytoplasmic vacuolization, syncytium formation in respiratory specimens, and crypt hyperplasia in gastrointestinal specimens. Notably, basement membrane integrity was relatively preserved (mean thickness $1.7 \pm 0.4 \mu\text{m}$; $p = 0.08$ vs. controls), consistent with the primarily intracellular tropism of these pathogens. Goblet cell hyperplasia was the most pronounced among all groups, reaching a mean density of $31.4 \pm 6.2 \text{ cells/mm}^2$, which likely reflects virus-induced upregulation of mucus production as a non-specific innate defense mechanism [6].

The results of the present study extend prior understanding of host-pathogen interactions at the tissue level by demonstrating that distinct pathogen classes produce characteristic, reproducible patterns of epithelial remodeling that are detectable by standard and immunohistochemical methods.

CONCLUSION

The present investigation demonstrates that exposure of mucosal epithelial tissues to pathogenic microorganisms induces morphofunctional alterations that

are pathogen-class-specific and quantifiable by histological and immunohistochemical analysis. Gram-negative bacterial infections produced the most severe disruption of barrier structures, particularly tight junctions and basement membrane, while RNA viral infections were associated with prominent goblet cell hyperplasia and intracytoplasmic changes with relative preservation of extracellular matrix components.

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