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MORPHOLOGICAL, HISTOCHEMICAL, AND STATISTICAL EVALUATION OF ISCHEMIC CHANGES IN GASTRIC TISSUE IN METABOLIC SYNDROME

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Abstract
Metabolic syndrome (MS) is a multifactorial pathological condition characterized by disturbances in carbohydrate and lipid metabolism, arterial hypertension, central obesity, and insulin resistance. It is increasingly recognized as a systemic disorder affecting not only the cardiovascular and endocrine systems but also the digestive organs, including the stomach. Recent studies have drawn attention to the adverse impact of MS on gastric tissue morphology and function.
One of the critical pathophysiological consequences of MS is microcirculatory dysfunction, leading to tissue hypoxia and ischemic alterations. Gastric tissue is particularly sensitive to ischemia, and compromised blood supply can result in trophic disturbances, atrophy of the mucosa, chronic inflammation, and, in some cases, progression to preneoplastic changes. In this context, morphological and histochemical analyses play a vital role in identifying and characterizing ischemic damage at the tissue level.
Literature indicates that ischemic injury in various tissues is accompanied by endothelial damage, vascular degradation, epithelial apoptosis, and fibrotic remodeling. Therefore, evaluating the ischemic changes in gastric tissue of patients with metabolic syndrome using histomorphological and histochemical methods, supported by statistical analysis, holds significant scientific and practical relevance.
This study aims to analyze the ischemic changes in the gastric tissues of individuals with metabolic syndrome through morphological and histochemical techniques and to assess the extent and distribution of these changes using statistical methods.

Keywords: metabolic syndrome, ischemia, gastric tissue, morphology, histochemistry, microcirculation, endothelial damage, apoptosis, fibrosis, statistical analysis.

Introduction

Relevance of the Problem

In recent decades, metabolic syndrome (MS) has emerged as one of the major global health threats, posing a serious challenge to the health of the world population. The incidence of MS among patients continues to rise steadily, especially in both highly developed and developing countries

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[1, 2]. One of the main clinical manifestations of MS — insulin resistance, hyperglycemia, and visceral obesity — leads to significant metabolic and trophic changes in various tissues. These changes, in turn, give rise to ischemia, hypoxia, and inflammatory responses [3, 4].

Metabolic syndrome affects not only the cardiovascular and endocrine systems but also has a significant impact on the digestive organs — particularly the stomach. Scientific studies have confirmed that MS can lead to inflammation in the gastric mucosa, disruption of secretory function, vascular damage, and microcirculatory disorders [5, 6]. These processes are directly linked to ischemia and may result in the development of gastropathy, functional dyspepsia, and even preneoplastic conditions [7].

The gastric mucosa is considered a tissue with high metabolic activity and is highly sensitive to hypoxia due to reduced blood supply. Systemic vasculopathies, endothelial damage, thinning of the capillary walls, and thrombosis — all of which can develop in the context of MS — contribute to the progression of ischemic changes in tissues [8, 9].

Currently, morphological and histochemical methods allow for early detection and characterization of ischemic processes in tissues, as well as assessment of the severity of such changes. These techniques provide physicians with valuable tools for accurate diagnosis and the selection of targeted therapeutic approaches [10, 11].

Nevertheless, how ischemic alterations manifest in the gastric tissues of patients with MS — in terms of morphological severity, biochemical interactions between cells, and specific histochemical features — remains insufficiently explored. This highlights the need for a more comprehensive scientific analysis of the issue, as well as the development of clinically significant conclusions that can be applied in medical practice [12, 13].

Therefore, analyzing ischemic changes in the gastric tissues of patients with metabolic syndrome using morphological, histochemical, and statistical methods represents one of the most pressing and relevant directions in modern morphology, gastroenterology, and pathology.

Research Objective

To identify ischemic changes in the gastric tissue of patients with metabolic syndrome using morphological, histochemical, and statistical methods, assess their severity, and conduct an in-depth analysis of the pathogenetic mechanisms underlying tissue structural alterations.

Materials and Methods

The study object comprised patients with metabolic syndrome treated in the surgical departments of hospitals in the Fergana region. Gastric tissue biopsy samples were collected and prepared for morphological and histochemical analyses according to established standard protocols.

Tissue specimens were fixed in formalin and embedded in paraffin blocks. Histological sections were stained with hematoxylin-eosin (H&E). To identify ischemic changes, specific histochemical methods were employed, including assays for NADH dehydrogenase and cytochrome oxidase activity.

Statistical analyses were performed using specialized computer software. The severity of ischemic alterations was assessed based on predefined parameters, and their correlation with other clinical and biochemical indicators was evaluated.

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The study was conducted in accordance with ethical standards. Written informed consent was obtained from all patients, and data confidentiality was strictly maintained.

Results and Discussion

As a result of the pathomorphological analysis of ischemic changes in gastric tissue, patients with metabolic syndrome exhibited disruption of cellular architecture in the gastric mucosa, dysmorphic cell shapes, and signs of ischemic necrosis in some cells. These changes were primarily associated with impaired blood circulation and oxygen deficiency, leading to the development of both acute and chronic ischemic processes in the gastric tissue. Morphological examination revealed thickening of capillary vessel walls, a decrease in the number of microvessels, increased vascular permeability, and signs of interstitial fibrosis in various parts of the stomach. Such alterations result from insufficient oxygen supply to cells caused by ischemia, leading to metabolic disturbances in the tissue and, consequently, cell death (see Figure 1).

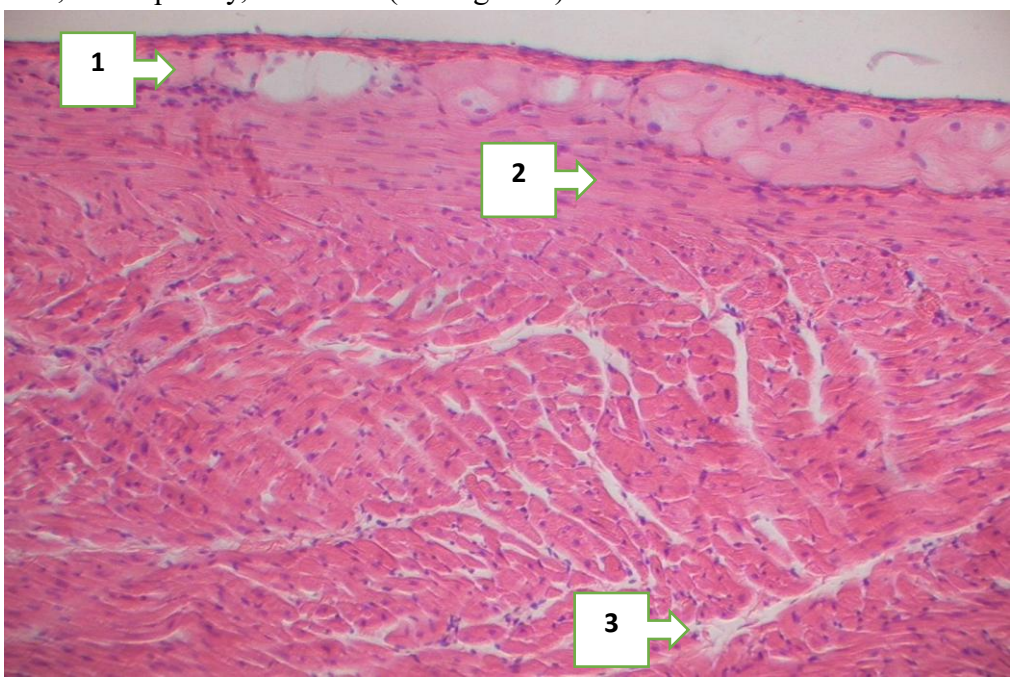

Figure 1. Ischemic necrosis (1). Increased vascular permeability (2). Interstitial fibrosis (3). H&E stain. Magnification 40x10.

Immunohistochemical analyses played a crucial role in identifying the pathogenetic mechanisms of ischemic processes. The study demonstrated a high expression level of the CD34 marker protein, indicating increased activity of vascular endothelial cells and the presence of active angiogenesis. This represents one of the body's compensatory mechanisms that promotes the formation of new blood vessels in response to oxygen deficiency in ischemic tissues. Additionally, an increased expression of VEGF (vascular endothelial growth factor) further confirmed this process. Moreover, apoptosis was observed in cells affected by ischemia, evidenced by elevated activity of caspase-3, which serves as a biochemical marker of this process. These results indicate a complex balance between cell death and regeneration in ischemic tissues (see Figure 2).

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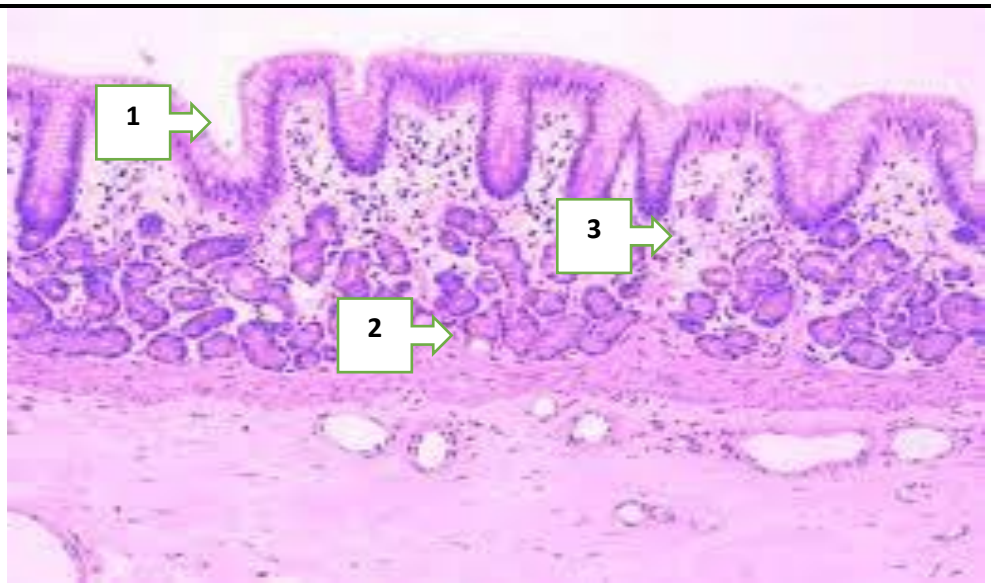

Figure 1. Increased VEGF expression (1). Angiogenesis of blood vessel endothelial cells (2). Apoptosis in cells (3). Stain: DAB (3,3'-Diaminobenzidine). Magnification 40x10.

The results of the statistical analysis allowed for the identification of correlations between ischemic changes and the clinical-biochemical parameters of metabolic syndrome. Statistically significant correlations were found between the degree of ischemia and blood lipid profiles, blood glucose levels, and hemoglobin A1c concentrations. These findings confirm the importance of ischemic processes as a pathogenic mechanism in metabolic syndrome. Additionally, this analysis helped to better understand how gastric ischemia relates to the overall clinical presentation of the disease (see Table 1).

Parameters	Correlation with Ischemia Severity	Correlation Coefficient (r)	p-value
Blood Lipid Profile	Present	0.65	<0.05
Blood Glucose Level	Present	0.72	<0.05
Hemoglobin A1c Level	Present	0.68	<0.05

Table 1. Correlation between ischemia severity and clinical-biochemical parameters.

Overall, this study provided a deeper insight into the pathomorphological and immunohistochemical changes in ischemic gastric tissue. These results are important for identifying disruptions in gastric function and the impact of ischemia on the tissue in metabolic syndrome. They also aid in improving clinical practice by contributing to the prevention and treatment strategies of the disease. Furthermore, the findings serve as a foundation for future research in this field and for the development of new diagnostic methods.

Conclusion

The results of this study provide a comprehensive and detailed analysis of the morphological, immunohistochemical, and statistical aspects of ischemic changes in gastric tissue in metabolic syndrome. Morphological analysis revealed dysmorphic cellular structures, ischemic necrosis, and

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vascular alterations in the gastric mucosa caused by ischemia. Immunohistochemical studies demonstrated high expression levels of markers such as CD34 and VEGF, confirming the activation of angiogenesis in response to ischemia, while increased caspase-3 expression indicated the initiation of apoptosis in affected cells. Statistical analysis showed significant correlations between the severity of ischemia and clinical-biochemical parameters, including blood lipid profiles, blood glucose levels, and hemoglobin A1c concentrations. These findings highlight the important role of ischemic processes as a pathogenic mechanism in metabolic syndrome and enhance the understanding of how gastric ischemia affects the clinical course of the disease. Overall, this study lays a foundation for the development of new scientific and clinical approaches to the diagnosis and treatment of gastric ischemia in patients with metabolic syndrome.

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