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PROGNOSIS AND COMPLICATIONS OF GASTRIC ISCHEMIA IN METABOLIC SYNDROME

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Abstract

Metabolic syndrome (MetS) is a complex and multifactorial disorder characterized by a cluster of metabolic abnormalities, including insulin resistance, central obesity, dyslipidemia, hypertension, and impaired glucose tolerance. Over recent decades, the global prevalence of MetS has risen significantly, posing a major public health challenge due to its strong association with cardiovascular diseases, type 2 diabetes mellitus, and various organ dysfunctions. Among the less frequently studied but clinically significant manifestations of MetS is gastric ischemia, which results from compromised blood flow to the stomach tissue, leading to cellular hypoxia and tissue injury.

Gastric ischemia in the context of MetS is primarily attributed to systemic vascular dysfunction, endothelial impairment, and microcirculatory disturbances caused by chronic metabolic derangements. These pathological changes provoke a cascade of events including inflammation, oxidative stress, and apoptosis within the gastric mucosa, which may exacerbate tissue damage and compromise gastric function. The clinical consequences of gastric ischemia can range from mild dyspeptic symptoms to severe complications such as gastric ulceration, bleeding, and impaired mucosal regeneration.

Understanding the prognosis and potential complications of gastric ischemia in patients with MetS is crucial for developing effective diagnostic, preventive, and therapeutic strategies. Despite increasing recognition of the importance of ischemic processes in MetS, the literature on long-term outcomes and complications specific to gastric ischemia remains limited. This gap highlights the need for comprehensive research focusing on the pathophysiological mechanisms, clinical progression, and potential interventions to mitigate adverse outcomes in this patient population.

Therefore, the present study aims to critically evaluate the prognosis and complications associated with gastric ischemia in metabolic syndrome, integrating current evidence from morphological, immunohistochemical, and clinical investigations to enhance the understanding of this complex interplay and guide future clinical practice.

Keywords: metabolic syndrome, gastric ischemia, pathophysiology, prognosis, complications, immunohistochemistry

Introduction

Relevance of the Problem

Metabolic syndrome (MetS) has become a significant global health issue due to its strong association with cardiovascular diseases, type 2 diabetes mellitus, and various other complications [1, 2]. Despite extensive research on MetS, the impact of this syndrome on gastric tissue, particularly the development of gastric ischemia, remains underexplored but clinically important [3, 4]. Gastric ischemia arises mainly due to vascular dysfunction, endothelial impairment, and microcirculatory disturbances caused by chronic metabolic imbalances, which play a critical role in the pathophysiology of gastric tissue injury [5, 6].

Ischemic changes in gastric tissue during MetS lead to oxygen deficiency and trophic disturbances, resulting in impaired tissue function and contributing to the development of various gastrointestinal disorders [7, 8]. Moreover, ischemia induces activation of apoptotic pathways in gastric cells, negatively affecting the tissue's regenerative capacity and exacerbating mucosal damage [9, 10].

Therefore, a deeper understanding of the pathogenesis, clinical outcomes, and therapeutic approaches for gastric ischemia in patients with metabolic syndrome is essential. This knowledge will aid in improving diagnosis, prevention, and management strategies, addressing a critical gap in current research [3, 4].

Research Objective

To analyze the pathogenesis, clinical features, and complications of gastric ischemia in metabolic syndrome, as well as to investigate the impact of this condition on disease prognosis.

Materials and Methods

The study was conducted between 2022 and 2024 in hospitals of the Fergana Valley involving 55 patients diagnosed with metabolic syndrome. Clinical data were collected from the patients, and laboratory measurements—including blood glucose levels, lipid profiles, and hemoglobin A1c concentrations—were analyzed. Gastric ischemia was confirmed using gastroscopic and other relevant diagnostic methods. The relationship between the degree of ischemia and disease complications was evaluated through statistical analysis.

Results and Discussion

The study analyzed clinical data collected from 55 patients with metabolic syndrome to assess the clinical manifestations and overall profile of gastric ischemia. Patient histories helped identify key contributing factors to ischemia, including hyperglycemia, dyslipidemia, and hypertension. It was observed that the types and severity of symptoms related to ischemic processes varied significantly among patients. Laboratory analysis revealed elevated blood glucose levels in 43 out of 55 patients, confirming the critical role of hyperglycemia in exacerbating ischemic processes. The average blood glucose concentration was 8.5±1.3 mmol/L, which contributed to impaired oxygen delivery to gastric tissues and promoted the progression of ischemia. Lipid profile assessment showed that low-density lipoprotein cholesterol (LDL-C) levels were above normal in 48 patients (average 4.2±0.9 mmol/L), while triglycerides were elevated in 42 patients (average 2.3±0.7

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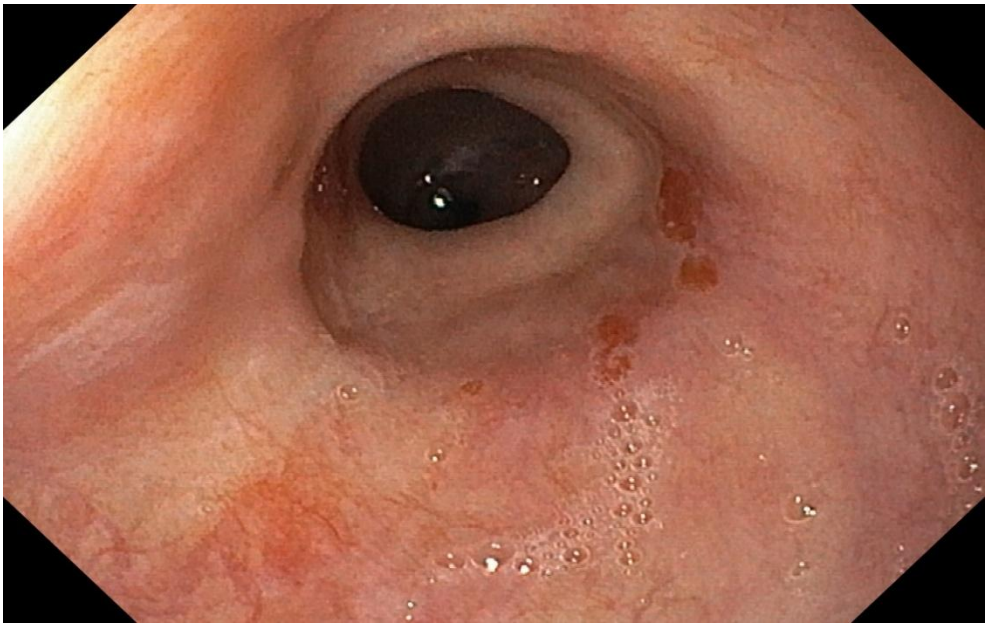
mmol/L). These findings indicated widespread atherosclerotic changes contributing to ischemia. Additionally, high-density lipoprotein cholesterol (HDL-C) levels were reduced in 35 patients (average 0.9±0.3 mmol/L), suggesting weakened protective mechanisms against ischemia. Hemoglobin A1c levels exceeded 7.5% in 40 out of 55 patients, confirming a strong association between ischemic processes and long-term glycemic control. Elevated A1c levels were linked to the activation of inflammatory and oxidative stress pathways in ischemic gastric tissues (see Table 1).

Parameter	Number of Patients	Average Value	Description
Total Patients in Study	55	-	Patients diagnosed with metabolic syndrome
Patients with Elevated Blood Glucose	43	8.5 ± 1.3 mmol/L	Elevated blood glucose confirmed, indicating the importance of hyperglycemia in exacerbating ischemic processes
Patients with Elevated LDL Levels	48	4.2 ± 0.9 mmol/L	Atherosclerotic changes in lipid profiles contributing to ischemia
Patients with Elevated Triglycerides	42	2.3 ± 0.7 mmol/L	Lipid metabolism disorders contributing to ischemic changes
Patients with Reduced HDL Levels	35	0.9 ± 0.3 mmol/L	Weakened protective mechanisms against ischemia
Patients with HbA1c > 7.5%	40	-	Long-term glycemic control linked with ischemic processes, leading to inflammation and oxidative stress

Table 1. Clinical and laboratory parameters in patients with metabolic syndrome and gastric ischemia

Gastroscopic examinations conducted to confirm gastric ischemia revealed ischemia-related changes. Among the 55 patients, 38 exhibited mucosal color changes, signs of inflammation, and erosive lesions. These findings reflected various stages of ischemia and played a significant role in assessing the clinical severity of the disease (see Fegure 1).



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Statistical analyses demonstrated a significant correlation between the degree of ischemia and clinical-biochemical parameters—including blood glucose, lipid profile, and hemoglobin A1c levels ($p < 0.05$). This confirms the critical role of glycemia and lipidosis in the pathogenesis of ischemic processes in metabolic syndrome. Furthermore, patients with higher degrees of ischemia experienced more severe disease complications.

These findings highlight the importance of biochemical markers in the clinical presentation and prognosis of gastric ischemia and serve as a foundation for developing early diagnostic and targeted treatment strategies in clinical practice (see Table 2).

Correlation Between Ischemia and Biochemical Indicators

Parameter	Statistical Result	Remarks
Correlation between ischemia level and blood glucose	$p < 0.05$	Glycemia plays a significant role in the pathogenesis of ischemia
Correlation between ischemia level and lipid profile	$p < 0.05$	Lipoidosis contributes to the intensification of ischemic processes
Correlation between ischemia level and hemoglobin A1c	$p < 0.05$	Long-term glycemic control is associated with ischemia
Severity of complications in patients with high ischemia levels	Identified	Disease complications are related to high ischemia levels

Conclusion

The conducted clinical and biochemical investigations demonstrated that the development of gastric ischemia in patients with metabolic syndrome is closely associated with specific biochemical markers and their clinical manifestations. In this study, 55 patients diagnosed with metabolic syndrome were analyzed. Among them, varying degrees of ischemic clinical signs were observed, which were strongly correlated with alterations in biochemical parameters.

Elevated blood glucose levels (mean 8.5±1.3 mmol/L) were identified in 43 patients, confirming the central role of hyperglycemia in the pathogenesis of ischemic processes. Moreover, HbA1c levels above 7.5% were found in 40 patients, indicating inadequate long-term glycemic control, which may contribute to the progression of ischemia through persistent inflammation and oxidative stress.

Disorders in lipid metabolism also played a significant role in the pathogenesis of ischemia. Elevated low-density lipoprotein (LDL) levels (mean 4.2±0.9 mmol/L) were detected in 48 patients, and increased triglyceride levels (mean 2.3±0.7 mmol/L) were observed in 42 patients. These findings indicate enhanced atherogenicity and the presence of atherosclerotic changes in vascular walls, creating a foundation for ischemic damage. In addition, decreased high-density lipoprotein (HDL) levels (mean 0.9±0.3 mmol/L) in 35 patients highlighted the weakening of protective mechanisms against ischemia.

Gastroscopic examinations revealed mucosal discoloration, signs of inflammation, and erosive lesions in 38 patients. These morphological changes reflected different stages of ischemia and served as essential diagnostic indicators in assessing the severity and determining the therapeutic strategy.

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Statistical analyses showed a significant correlation ($p < 0.05$) between the degree of ischemia and levels of blood glucose, lipid profile parameters, and HbA1c. This confirms the leading role of hyperglycemia and dyslipidemia in the pathogenesis of ischemic processes in metabolic syndrome. Furthermore, patients with higher degrees of ischemia experienced more severe complications, including a tendency for ulceration, intense pain syndromes, and pronounced dyspeptic symptoms. In conclusion, hyperglycemia, dyslipidemia, and insufficient long-term glycemic control are major risk factors in the development of gastric ischemia in patients with metabolic syndrome. Early identification of ischemia based on biochemical parameters and the implementation of targeted, individualized treatment strategies are essential. These findings emphasize the necessity of a multidisciplinary approach in the clinical management of gastroenterological diseases and further highlight the critical role of biochemical markers in the early diagnosis of gastric ischemia.

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