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CLINICAL AND MORPHOLOGICAL CHANGES OF THE STOMACH IN MORBID OBESITY

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Abstract: Morbid obesity is a severe chronic metabolic disorder associated with profound functional and structural alterations in multiple organ systems, including the gastrointestinal tract. The stomach, as a key organ involved in digestion and appetite regulation, undergoes significant clinical and morphological changes in response to chronic excessive caloric intake and obesity-related hormonal imbalance. This study aims to analyze the clinical manifestations and histomorphological characteristics of the stomach in patients with morbid obesity. Clinical observations, endoscopic findings, and histological analyses demonstrate that morbid obesity is associated with increased prevalence of gastric symptoms, mucosal hypertrophy, glandular hyperplasia, chronic low-grade inflammation, and microvascular alterations. These changes reflect both adaptive and pathological processes that have important implications for the management of obese patients, particularly in the context of bariatric surgery.

Keywords: Morbid obesity, stomach, gastric morphology, clinical manifestations, gastric mucosa

Introduction

Morbid obesity represents a major global health problem due to its rapidly increasing prevalence and strong association with metabolic, cardiovascular, and gastrointestinal diseases. Defined by a body mass index of 40 kg/m² or higher, or 35 kg/m² in the presence of obesity-related comorbidities, morbid obesity leads to complex systemic alterations involving endocrine, inflammatory, and mechanical mechanisms. While much attention has been given to metabolic and cardiovascular complications, the impact of morbid obesity on the gastrointestinal tract, particularly the stomach, remains insufficiently characterized. The stomach plays a crucial role not only in digestion and nutrient storage but also in hormonal regulation of appetite through substances such as ghrelin. Chronic hyperphagia, increased intragastric pressure, and altered neurohormonal signaling contribute to functional and structural gastric changes. A detailed understanding of these alterations is essential for improving diagnostic accuracy and optimizing treatment strategies in morbidly obese patients.

Materials and Methods

This study is based on the analysis of clinical, endoscopic, and histopathological data obtained from patients diagnosed with morbid obesity. Clinical assessment focused on gastric-related symptoms, including dyspepsia, epigastric pain, and symptoms of gastroesophageal reflux disease. Endoscopic examination was performed to evaluate macroscopic changes of the gastric mucosa. Gastric tissue samples collected during bariatric surgical procedures were fixed in formalin, embedded in paraffin, and stained with hematoxylin and eosin for light microscopic examination. Morphological evaluation included assessment of mucosal thickness, glandular architecture, inflammatory cell infiltration, and vascular changes. The findings were analyzed descriptively to identify consistent patterns associated with morbid obesity.

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Results

Clinical assessment revealed that the majority of patients with morbid obesity exhibited pronounced gastric-related symptoms. Dyspeptic complaints, including epigastric discomfort, postprandial fullness, bloating, and nausea, were frequently reported. Symptoms consistent with gastroesophageal reflux disease, such as heartburn and regurgitation, were observed in a substantial proportion of patients, indicating a high prevalence of functional gastric and esophageal disturbances. The severity of clinical symptoms tended to correlate with the duration of obesity and the presence of associated metabolic comorbidities.

Endoscopic examination of the stomach demonstrated a range of macroscopic alterations. The gastric mucosa commonly appeared hyperemic and edematous, with areas of increased friability, suggesting impaired mucosal integrity. In some cases, superficial erosive changes were noted, particularly in the antral and corporal regions. These endoscopic findings reflected ongoing inflammatory processes and increased susceptibility of the gastric lining to injury.

Histomorphological analysis revealed marked structural remodeling of the gastric wall. A significant increase in gastric mucosal thickness was observed, most prominently in the fundus and body of the stomach. This thickening was primarily due to epithelial proliferation and expansion of the glandular compartment. Glandular hyperplasia was a consistent feature, characterized by increased density and enlargement of gastric glands, indicating enhanced secretory activity. The lamina propria showed diffuse infiltration by lymphocytes and macrophages, confirming the presence of chronic low-grade inflammation.

Additionally, notable microvascular alterations were identified within the gastric mucosa and submucosa. Vascular congestion, capillary dilation, and signs of impaired microcirculation were frequently observed, suggesting compromised tissue perfusion and a tendency toward local hypoxia. These vascular changes were often accompanied by mild interstitial edema, further contributing to mucosal dysfunction. Collectively, the clinical, endoscopic, and histological findings demonstrate that morbid obesity is associated with consistent and significant gastric alterations that involve both functional impairment and structural remodeling.

Discussion

The results of this study confirm that morbid obesity is associated with significant clinical and morphological changes of the stomach. Mucosal hypertrophy and glandular hyperplasia appear to represent adaptive responses to chronic excessive food intake and increased gastric workload. However, persistent inflammatory infiltration and vascular alterations indicate the development of pathological processes that may compromise gastric function. The high prevalence of gastroesophageal reflux disease can be attributed to increased intra-abdominal pressure, altered gastric motility, and excessive acid secretion. Furthermore, chronic inflammation of the gastric mucosa may be linked to systemic inflammatory processes characteristic of obesity, creating a bidirectional relationship between local and systemic pathology. These findings are particularly relevant in the context of bariatric surgery, as pre-existing gastric changes may influence surgical planning and postoperative outcomes.

Conclusion

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Morbid obesity leads to profound and multifaceted clinical and morphological alterations of the stomach that reflect the combined effects of chronic metabolic imbalance, hormonal dysregulation, mechanical overload, and systemic inflammation. The findings of this study demonstrate that the stomach in morbidly obese individuals undergoes significant structural remodeling, characterized by mucosal hypertrophy, glandular hyperplasia, persistent low-grade inflammatory infiltration, and microvascular disturbances. These changes represent initial adaptive mechanisms aimed at accommodating increased functional demands; however, prolonged exposure to excessive caloric intake and inflammatory stimuli promotes the progression toward pathological remodeling and functional impairment.

Clinically, the high prevalence of dyspeptic symptoms and gastroesophageal reflux disease underscores the impact of obesity-related gastric alterations on patient quality of life and disease burden. The close association between clinical manifestations and histomorphological changes highlights the importance of comprehensive gastric assessment in morbidly obese patients. In particular, chronic inflammation and vascular congestion of the gastric mucosa may predispose patients to further complications, including erosive gastritis and impaired mucosal defense mechanisms.

From a clinical perspective, recognition of these gastric changes is essential for optimizing diagnostic strategies and therapeutic decision-making. Preoperative evaluation of gastric morphology is especially important in candidates for bariatric surgery, as existing mucosal and structural abnormalities may influence surgical technique selection, postoperative healing, and long-term outcomes. Moreover, understanding the underlying morphological adaptations of the stomach provides valuable insight into the pathophysiological mechanisms linking obesity with gastrointestinal dysfunction.

In conclusion, morbid obesity should be regarded not only as a systemic metabolic disorder but also as a condition associated with significant gastric remodeling and dysfunction. Early identification and targeted management of gastric changes may contribute to improved clinical outcomes and reduced complication rates. Future studies incorporating molecular, immunohistochemical, and functional assessments are necessary to further elucidate the mechanisms of gastric adaptation and injury in morbid obesity and to develop more effective, individualized therapeutic approaches.

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