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# ETIOLOGY OF MALABSORPTION SYNDROME AND PATHOMORPHOLOGICAL CHANGES IN THE INTESTINE

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***Annotation.*** *Malabsorption syndrome is a multifactorial clinical condition characterized by impaired digestion and absorption of nutrients in the small intestine. The disorder may arise from congenital enzyme deficiencies, chronic inflammatory diseases, pancreatic and hepatobiliary dysfunction, infectious agents, or structural intestinal abnormalities.*

*Pathomorphological changes, such as villous atrophy, crypt hyperplasia, and inflammatory infiltration of the mucosa, are central to its development and progression.*

*Clinically, malabsorption presents with both gastrointestinal symptoms, including chronic diarrhea, abdominal discomfort, and steatorrhea, as well as systemic manifestations such as anemia, bone demineralization, growth retardation, and neurological deficits. Diagnosis requires an integrated approach, combining biochemical, radiological, endoscopic, and histopathological evaluations.*

***Keywords:*** *Malabsorption syndrome, Intraluminal digestion, Mucosal absorption, Postmucosal transport, Villous atrophy, Crypt hyperplasia, Inflammatory infiltration, Steatorrhea, Hypoproteinemia, Osteomalacia.*

# ЭТИОЛОГИЯ СИНДРОМА МАЛЬАБСОРБЦИИ И ПАТОМОРФОЛОГИЧЕСКИЕ ИЗМЕНЕНИЯ КИШЕЧНИКА

***Аннотация.*** *Синдром мальабсорбции – это многофакторное клиническое состояние, характеризующееся нарушением переваривания и всасывания питательных веществ в тонком кишечнике. Заболевание может быть обусловлено врожденной ферментной недостаточностью, хроническими воспалительными заболеваниями, дисфункцией поджелудочной железы и гепатобилиарной системы, инфекционными агентами или структурными аномалиями кишечника. Патоморфологические изменения, такие как атрофия ворсинок, гиперплазия крипт и воспалительная инфильтрация слизистой оболочки, играют ключевую роль в его развитии и прогрессировании.*

*Клинически мальабсорбция проявляется как желудочно-кишечными симптомами, включая хроническую диарею, дискомфорт в животе и стеаторею, так и системными проявлениями, такими как анемия, деминерализация костей, задержка роста и неврологические нарушения. Диагностика требует комплексного подхода, сочетающего биохимические, рентгенологические, эндоскопические и гистопатологические исследования.*

***Ключевые слова:*** *Синдром Мальабсорбции, Внутрипросветное Пищеварение, Слизистая Абсорбция, Постмукозный Транспорт, Атрофия Ворсинок, Гиперплазия Крипт, Воспалительная Инфильтрация, Стеаторея, Гипопротеинемия, Остеомаляция.*

## Introduction

Malabsorption syndrome is a pathological condition characterized by impaired digestion and absorption of nutrients in the small intestine, leading to deficiencies of proteins, fats,

carbohydrates, vitamins, and minerals. This disorder is not an independent disease but rather a complex clinical manifestation that develops as a result of various gastrointestinal and systemic pathologies. The etiology of malabsorption syndrome is diverse and includes congenital enzymatic defects, chronic inflammatory bowel diseases, pancreatic insufficiency, celiac disease, infections, and surgical resections of the intestine. The pathogenesis of malabsorption is closely related to both functional and structural changes in the small intestine. Particularly, morphological alterations in the intestinal mucosa such as villous atrophy, crypt hyperplasia, inflammatory cell infiltration, and fibrosis play a crucial role in the impairment of absorption processes. These pathological changes disrupt the surface area available for nutrient absorption, alter enzymatic activity, and cause secondary systemic complications, including malnutrition, anemia, osteoporosis, and growth retardation in children. Understanding the mechanisms of malabsorption syndrome and its associated intestinal pathomorphological changes is essential for timely diagnosis, effective treatment, and prevention of complications. Therefore, this topic holds great importance in gastroenterology and internal medicine, requiring further research and clinical attention.

**Main part**

Malabsorption syndrome is a pathological state resulting from impaired digestion and absorption of nutrients in the small intestine. Normally, intestinal villi and microvilli ensure efficient uptake of proteins, fats, carbohydrates, vitamins, and minerals, but in this condition, that function is disturbed. It is not regarded as a single disease entity but rather as a clinical manifestation of various disorders. Patients may present with symptoms ranging from mild nutritional deficiency to severe systemic complications. The syndrome may occur acutely or persist as a chronic disorder. Its general characteristics include weight loss, weakness, anemia, and biochemical disturbances. Clinicians categorize malabsorption into intraluminal, mucosal, and postmucosal types, depending on the primary mechanism involved. This classification reflects the complexity of the disorder and emphasizes the importance of comprehensive evaluation. In essence, malabsorption syndrome represents a multifactorial condition requiring accurate diagnosis.

The causes of malabsorption syndrome are highly diverse and multifactorial. Congenital etiologies include enzyme deficiencies such as lactase deficiency and rare genetic disorders affecting transport systems. Acquired causes are more common and include chronic pancreatitis, liver diseases, and celiac disease. Infections, particularly parasitic infestations like Giardia lamblia, can also impair absorption. Surgical resection of large segments of the intestine, known as short bowel syndrome, is another well-documented cause. Autoimmune processes, such as those observed in inflammatory bowel disease, contribute to mucosal injury and subsequent malabsorption. Other causes include bile acid deficiency, lymphatic obstruction, and radiation enteropathy. Each of these mechanisms ultimately interferes with nutrient absorption. The multifactorial nature of these causes necessitates individualized diagnostic approaches.

The pathogenesis of malabsorption involves a complex interplay between defective digestion and impaired absorption. Intraluminal mechanisms include reduced enzyme activity and bile salt deficiency, leading to incomplete breakdown of food. Mucosal mechanisms involve damage to the epithelial lining, such as villous atrophy, crypt hyperplasia, or epithelial

inflammation. Postmucosal mechanisms occur when absorbed nutrients cannot be transported due to lymphatic obstruction or vascular compromise. Pathogenesis is further complicated by alterations in the gut microbiota, which may exacerbate inflammation. These processes reduce the absorptive surface area and impair enzyme activity, resulting in inadequate nutrient uptake.

Over time, persistent malabsorption causes secondary systemic effects such as malnutrition, anemia, and metabolic bone disease. Understanding the pathogenesis provides a framework for targeted therapeutic strategies.

Pathomorphological alterations in the small intestine are central to malabsorption syndrome. The most characteristic feature is villous atrophy, which dramatically reduces absorptive surface area. Crypt hyperplasia is often observed, indicating compensatory epithelial regeneration. Inflammatory cell infiltration in the lamina propria contributes to tissue damage and functional impairment. Fibrosis and scarring of the mucosa may develop in chronic cases, further restricting nutrient absorption. In celiac disease, these changes are particularly prominent, with complete flattening of villi. Infectious causes may produce mucosal erosions, edema, and epithelial destruction. Pancreatic insufficiency, though primarily extraluminal, also leads to secondary intestinal mucosal changes due to maldigestion. These histopathological findings are crucial for establishing diagnosis and guiding therapy.

Clinical manifestations of malabsorption syndrome are highly variable but generally reflect nutrient deficiencies. Gastrointestinal symptoms include chronic diarrhea, bloating, abdominal pain, and steatorrhea. Systemic manifestations are more serious, including anemia from iron, folate, or vitamin B12 deficiency. Fat-soluble vitamin deficiencies lead to osteomalacia, rickets, and coagulopathy. Protein deficiency results in muscle wasting, edema, and hypoalbuminemia. Children may present with growth retardation, delayed puberty, and developmental delays. Neurological symptoms, such as peripheral neuropathy, are associated with vitamin B12 or thiamine deficiency. In severe cases, patients develop cachexia and profound metabolic disturbances. The multisystemic nature of clinical manifestations underscores the need for comprehensive evaluation and treatment.

Diagnosis of malabsorption syndrome requires a stepwise and multidisciplinary approach. Laboratory tests are essential for detecting anemia, hypoproteinemia, electrolyte imbalances, and vitamin deficiencies. Stool analysis provides evidence of steatorrhea and undigested food particles. Imaging studies, including abdominal ultrasound, CT, and MRI, can reveal structural abnormalities. Endoscopic procedures with biopsy of the small intestine are critical for assessing villous atrophy, crypt hyperplasia, and inflammatory infiltrates. Breath tests are useful for detecting carbohydrate malabsorption and bacterial overgrowth. Pancreatic function tests help identify exocrine insufficiency. Radiological contrast studies can demonstrate intestinal shortening or structural lesions. Genetic testing may be indicated for congenital enzyme deficiencies. Accurate diagnosis requires integration of clinical, biochemical, radiological, and histological data.

Treatment of malabsorption syndrome is based on correcting the underlying cause and addressing nutritional deficiencies. Conservative management includes dietary modification, such as a gluten-free diet in celiac disease or lactose restriction in lactase deficiency. Pancreatic enzyme replacement is used in exocrine pancreatic insufficiency.

Vitamin and mineral supplementation is crucial, particularly fat-soluble vitamins, iron, folate, and B12. Infections require specific antimicrobial therapy. Anti-inflammatory or immunosuppressive drugs are indicated in autoimmune causes such as Crohn's disease. In cases of lymphatic obstruction, surgical or interventional procedures may be necessary. Nutritional support, including enteral or parenteral nutrition, is used in severe cases. Regular monitoring ensures prevention of long-term complications. An individualized, multidisciplinary approach is essential for optimal management.

Prevention of malabsorption syndrome primarily depends on early recognition and treatment of underlying diseases. Public health measures, such as controlling parasitic infections and improving nutrition, reduce incidence. Patient education on dietary management plays a key role, particularly for chronic conditions. Prognosis varies depending on the etiology: celiac disease and lactose intolerance generally have excellent outcomes with dietary therapy, whereas short bowel syndrome or chronic pancreatitis may carry a poorer prognosis. Delayed diagnosis often results in severe malnutrition, growth failure, and long-term complications. Clinically, malabsorption remains significant due to its systemic impact and potential for misdiagnosis. Continued research into pathophysiology and novel treatments is essential for improving patient outcomes.

**Conclusion**

Malabsorption syndrome represents a multifactorial clinical condition with profound implications for patient health and quality of life. It arises from a wide range of congenital and acquired causes, including enzymatic defects, chronic inflammatory diseases, pancreatic insufficiency, intestinal resections, and infections. The underlying pathogenesis involves disturbances in intraluminal digestion, mucosal absorption, and postmucosal transport, all of which culminate in inadequate nutrient uptake. Pathomorphological changes, particularly villous atrophy, crypt hyperplasia, and inflammatory infiltration of the mucosa, are hallmarks of the disorder and play a central role in its progression. Clinically, the syndrome manifests through both gastrointestinal and systemic features, ranging from diarrhea and steatorrhea to anemia, bone disease, growth retardation, and neurological deficits. Diagnosis requires a comprehensive approach, integrating biochemical, radiological, endoscopic, and histological findings. Treatment strategies must be tailored to the underlying etiology, with emphasis on dietary modification, enzyme replacement, vitamin and mineral supplementation, and management of complications. Ultimately, early recognition and targeted management of malabsorption syndrome are crucial for preventing long-term systemic consequences. Its significance in gastroenterology and internal medicine highlights the necessity of continued research into its mechanisms, diagnostic innovations, and therapeutic options. Through a multidisciplinary approach, patient outcomes can be substantially improved, ensuring better quality of life and reduction of morbidity.

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