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MORPHOFUNCTIONAL CHANGES IN THE SMALL INTESTINE IN CHILDREN WITH PNEUMONIA

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Abstract
Pneumonia remains one of the leading causes of childhood mortality. Respiratory system damage activates systemic response reactions in the organism and negatively affects distant organs, including the morphofunctional state of the small intestine. This article analyzes structural and functional changes occurring in the small intestinal mucosa in children with pneumonia, including villous atrophy, enterocyte damage, microcirculatory disorders, and pathogenetic mechanisms of inflammatory processes. The study demonstrates the systemic effect of pneumonia on the gastrointestinal tract, substantiating the necessity of a comprehensive approach in pediatric practice.

Keywords: Pneumonia, small intestine, morphology, villi, enterocytes, microcirculation, hypoxia, endotoxicosis, inflammation, mucosa, pathophysiology, children, gastrointestinal, structural, functional.

Introduction

Pneumonia represents one of the principal infectious diseases contributing to elevated morbidity and mortality rates among children under five years of age. The inflammatory process developing in pulmonary tissue does not remain confined to localized pathological alterations. Respiratory system damage provokes robust systemic inflammatory reactions throughout the organism, leading to alterations in distant organs, particularly within the gastrointestinal tract. The small intestinal mucosa, owing to its elevated metabolic activity and intensive vascular supply, demonstrates heightened sensitivity to systemic stress and consequently undergoes morphofunctional disruptions under pneumonic conditions. Clinical observations indicate that children with pneumonia frequently manifest gastrointestinal symptoms including digestive dysfunction, diarrhea, abdominal distension, and deterioration of nutritional status. However, the pathophysiological foundations of these alterations and the direct impact of pneumonia on small intestinal structural integrity remain inadequately investigated. Understanding the morphofunctional connections between respiratory and gastrointestinal systems proves essential for developing comprehensive treatment strategies in pediatric practice. The relationship between pulmonary infection and intestinal pathology extends beyond simple clinical correlation. Respiratory insufficiency induces systemic hypoxemia, affecting all highly metabolic tissues. The small intestine, with its rapid cellular turnover and intensive absorptive functions, becomes particularly vulnerable. Additionally, the systemic inflammatory response syndrome activated during pneumonia releases proinflammatory cytokines into circulation, which subsequently interact with intestinal tissues, modifying their morphological architecture and functional capacity.

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Literature review

Tuxtasinova and colleagues have documented alterations in intestinal microbiome composition and dysbiotic state development during pneumonia episodes. Their investigations revealed elevated inflammatory mediator concentrations within the small intestinal mucosa alongside compromised barrier function. Azizova has examined morphological transformations of the gastrointestinal tract during infectious processes in children, identifying reductions in villous height and modifications in crypt depth.

Nurmuxamedova emphasizes intensified intestinal epithelial apoptosis and decelerated regenerative processes during respiratory infections. Sharipova has substantiated microcirculatory bed damage and increased capillary permeability under pneumonic conditions through morphometric methodologies. Mamadalieva has comprehensively assessed the impact of systemic inflammatory syndrome on the gastrointestinal tract in children, observing structural disruptions of enterocyte membranes. While existing literature demonstrates correlations between pneumonia and intestinal pathology, precise morphological alterations within the small intestine and their pathogenetic mechanisms remain incompletely elucidated. Comprehensive analysis of villous architecture, enterocyte ultrastructure, and microcirculatory status warrants particular attention. The gap between clinical observations of gastrointestinal dysfunction and detailed histopathological documentation necessitates systematic investigation employing contemporary morphological techniques.

MAIN BODY

Pneumonia in pediatric populations manifests not merely as localized respiratory inflammation but as a systemic pathological condition encompassing the entire organism. The inflammatory process developing within pulmonary tissue results in augmented circulation of biologically active substances, cytokines, endotoxins, and metabolic products through the bloodstream. These factors directly influence the morphofunctional state of organs possessing elevated metabolic activity, notably the small intestine. The small intestinal mucosa constitutes one of the organism's primary immunological and metabolic barriers, with its structural integrity proving crucial for maintaining overall homeostasis.

Impaired alveolar ventilation during pneumonia progression, combined with decreased arterial oxygen saturation, precipitates systemic hypoxia. The small intestinal mucosa, possessing high regenerative capacity, demonstrates particular sensitivity to oxygen deficiency. Hypoxia consequently attenuates energy metabolism within enterocytes, disrupts mitochondrial function, and intensifies degenerative processes in intracellular structural elements. Microcirculatory alterations manifest through capillary spasm, stasis, and endothelial dysfunction. This condition produces interstitial edema within the intestinal wall, trophic tissue disturbances, and diminished protective characteristics of the mucosa. The concentration of proinflammatory cytokines, including interleukins, tumor necrosis factor, and additional mediators, increases dramatically during pneumonia. These biologically active substances reach the intestinal mucosa through hematogenous routes, provoking local inflammatory processes. Within the small intestine, lymphoid tissue activation occurs alongside cellular infiltration in Peyer patches and alterations in immune cell proportions. Compromised intestinal immune function proceeds with decreased secretory immunoglobulin A synthesis, creating conditions for intensified pathogenic microorganism and toxic

substance effects within the intestinal lumen. Structural reorganization processes occur within the small intestinal mucosa of children afflicted with pneumonia. Villous shortening and alterations in their configuration and density lead to reduced absorptive surface area. Decreased numbers and irregular arrangement of microvilli located on the apical portion of enterocytes cause diminished enzymatic activity. Dystrophic and desquamative processes predominate within the mucosal epithelial layer. In certain instances, loosening of intercellular connections increases intestinal wall permeability, facilitating endotoxin passage into systemic circulation. The morphological alterations of the small intestine directly impact its functional activity. Damage to the enzymatic apparatus of enterocytes results in decreased disaccharidase and peptidase activity. Consequently, food substance degradation and absorption occur inadequately. The functional state of the neuromuscular apparatus within the intestinal wall also undergoes modification during pneumonia. Autonomic nervous system imbalance causes decelerated or irregular peristalsis, intensifying susceptibility to meteorism and malabsorption. These conditions may lead to amplified general intoxication manifestations and prolonged recovery periods in children.

Results

Morphological examination of small intestinal mucosa in children with pneumonia identified several characteristic alterations. Relative reduction in mucosal thickness, decreased villous height, and villous deformation were recorded in nearly all cases. Enterocyte desquamation at villous apices was observed, leading to compromised superficial epithelial integrity. At the microscopic level, dystrophic changes appear within enterocyte cytoplasm. Organellar swelling, disruption of mitochondrial cristae, and endoplasmic reticulum dilation were noted. Chromatin condensation within nuclei and apoptotic change markers in certain cells were recorded.

Structural disruptions emerge in brush border microvilli, causing reduced resorptive surface and impaired nutrient absorption. Inflammatory infiltration was identified within the lamina propria layer. Increased numbers of lymphocytes, plasma cells, and macrophages were observed. Capillaries and venules appeared dilated, with erythrocyte extravasation and perivascular edema signs present. Stasis phenomena, endothelial cell swelling, and microthrombus formation in certain areas were recorded within the microcirculatory bed. Alterations in goblet cell numbers and compromised secretory activity were determined. Decreased mucus production weakens the protective barrier, facilitating microorganism penetration into the mucosa. Reduced regenerative activity was observed in the crypt zone, leading to decelerated mucosal restoration processes. Within the submucosa layer, vascular congestion, edema, and increased fibroblast activity were identified. Fibrotic change signs appeared in some cases. Dystrophic alterations and inflammatory element infiltration were observed among muscularis mucosae cells.

Discussion

The obtained results demonstrate significant pneumonic impact on small intestinal morphofunctional status. Villous atrophy and enterocyte damage are explained through several mechanisms. Primarily, respiratory insufficiency and hypoxemia generate tissue oxygen deficiency conditions. Small intestinal epithelium, possessing elevated metabolic activity, demonstrates extreme hypoxia

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sensitivity. Oxygen deficiency disrupts cellular energy metabolism, decreases ATP synthesis, and damages cellular structures.

Secondarily, the systemic inflammatory reaction developing during pneumonia plays a crucial role. Proinflammatory cytokines, including interleukin-1, interleukin-6, and tumor necrosis factor, disseminate through blood and affect intestinal tissues. These cytokines disrupt endothelial function, increase capillary permeability, and enhance inflammatory cell migration into tissues. Microcirculatory disturbances deteriorate trophic supply to small intestinal mucosa. Capillary stasis, microthrombosis, and endothelial damage lead to tissue ischemic conditions, subsequently causing enterocyte dystrophy and necrosis. Epithelial barrier damage leads to increased intestinal permeability. Tight junction structure between enterocytes becomes disrupted, creating conditions for bacterial and toxin translocation. Bacterial translocation intensifies systemic endotoxicosis and elevates sepsis development risk. Decreased goblet cell function leads to mucous layer thinning, facilitating microorganism adhesion to epithelial surfaces. Decelerated regeneration processes indicate diminished mucosal restoration capacity. Reduced proliferative activity of stem cells within the crypt zone decelerates enterocyte renewal, leading to prolonged mucosal structural disruption and decreased resorptive function. Clinically, these morphological alterations manifest through impaired nutrient absorption, diarrhea, and nutritional status deterioration. Children face increased risk of developing nutritional deficiency, further weakening immune system function and worsening disease prognosis. Therefore, pneumonia treatment requires not only antibacterial therapy but also gastrointestinal tract supportive measures. Comparing with other investigators' results, our data confirm general tendencies while providing detailed morphological change descriptions. The degree of villous atrophy and enterocyte damage character are determined to depend on disease severity and duration, proving significant for prognosis assessment and treatment tactic selection.

Pneumonia in children leads to significant disruption of small intestinal morphofunctional status. Villous atrophy, enterocyte dystrophy, microcirculatory disturbances, and inflammatory infiltration demonstrate the systemic impact of respiratory infection. These alterations connect with hypoxia, endotoxicosis, and proinflammatory cytokine effects. Comprehensive approaches supporting gastrointestinal tract function during pneumonia treatment in pediatric practice prove necessary, contributing to improved disease prognosis and complication prevention. Recognition of these morphofunctional connections enables clinicians to implement more effective therapeutic strategies addressing both primary respiratory pathology and secondary gastrointestinal complications.

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