



Review

Antibiotic Utilization in Acute Pancreatitis: A Narrative Review

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Abstract: Acute pancreatitis is a complex inflammatory disease with significant morbidity and mortality. Despite advances in its management, the role of antibiotics in the prophylaxis and treatment of acute pancreatitis remains controversial. The aim of this comprehensive review is to analyze current evidence on the use of antibiotics in acute pancreatitis, focusing on prophylactic and therapeutic strategies. Prophylactic use aims to prevent local and systemic infections. However, recent studies have questioned the routine use of antibiotics for prophylaxis and highlighted the potential risks of antibiotic resistance and adverse effects. In selected high-risk cases, such as infected necrotizing pancreatitis, prophylactic antibiotic therapy may still be beneficial. As for therapeutic use, antibiotics are usually used to treat infected pancreatic necrosis and extrapancreatic infections. When selecting an antibiotic, the microbiologic profile and local resistance patterns should be considered. Combination therapy with broad-spectrum antibiotics is often recommended to cover both Gram-positive and Gram-negative pathogens. Recent research has highlighted the importance of individualized approaches to antibiotic use in acute pancreatitis and underscored the need for a tailored approach based on patient-specific factors. This review also highlights the potential role of new antimicrobial agents and alternative strategies, such as probiotics, in the management of acute pancreatitis.

Keywords: antibiotics; prophylaxis; acute pancreatitis; probiotics



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1. Introduction

Acute pancreatitis (AP) is a serious inflammatory process that can range from a mild, self-limiting condition to a life-threatening disease with significant morbidity and mortality. The exact mechanisms that trigger the onset of AP are not yet fully understood but are thought to involve a complex interaction of genetic, environmental, and lifestyle factors. The initial event in the development of AP is the inappropriate activation of pancreatic enzymes within the acinar cells; under pathological conditions, such as bile reflux, alcohol abuse, or gallstones, the activation of pancreatic enzymes occurs prematurely within the pancreas itself. Once activated, they can directly damage acinar cells, leading to their necrosis. The release of pro-inflammatory cytokines, such as interleukin-1 β (IL-1 β) and tumor necrosis factor-alpha (TNF- α), further amplifies the inflammatory response. These cytokines recruit and activate immune cells, including neutrophils and macrophages, which infiltrate the pancreas and release additional inflammatory mediators, exacerbating tissue damage and promoting the production of reactive oxygen species (ROS), resulting in oxidative stress [1]. Calcium signaling also plays a critical role: intracellular calcium overload occurs due to both the release from intracellular stores and the influx from the

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