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Potassium channel acid blockers (P-CABs): A Comprehensive review

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Introduction

Acid-related gastrointestinal disorders constitute a substantial global health burden, encompassing a spectrum of conditions such as gastroesophageal reflux disease (GERD), erosive esophagitis, peptic ulcer disease, functional dyspepsia, and *Helicobacter pylori* infection [3]. These disorders are highly prevalent across diverse populations and are associated with chronic symptoms, impaired quality of life, increased healthcare utilization, and significant socioeconomic costs. The pathophysiological hallmark common to these conditions is inappropriate or excessive gastric acid secretion, which disrupts mucosal integrity and perpetuates inflammation within the upper gastrointestinal tract. Consequently, pharmacological suppression of gastric acid secretion has remained the cornerstone of both symptom control and mucosal healing in acid-mediated diseases for several decades [3].

Proton pump inhibitors (PPIs) have dominated the therapeutic landscape of acid suppression since their introduction in the late twentieth century, owing to their ability to irreversibly inhibit the gastric H^+/K^+-ATPase enzyme located on parietal cells. PPIs rapidly replaced histamine-2 receptor antagonists (H2RAs) as first-line therapy due to superior acid suppression, improved healing rates of erosive esophagitis, and enhanced efficacy in *H. pylori* eradication regimens. Despite these advantages, accumulating clinical experience over the past three decades has revealed several pharmacodynamic and pharmacokinetic limitations inherent to PPIs. These include delayed onset of action, dependence on acid-activated prodrug conversion, variability in response related to cytochrome P450 (particularly CYP2C19) polymorphisms, nocturnal acid breakthrough, and incomplete symptom relief in a substantial subset of patients [2].

Clinical dissatisfaction with PPI therapy is especially evident in patients with so-called PPI-refractory GERD, a condition characterized by persistent reflux symptoms despite optimized PPI dosing.

Epidemiological studies suggest that up to 30–40% of patients treated with standard PPI regimens continue to experience troublesome symptoms,
highlighting a clear unmet therapeutic need [3]. In addition, long-term PPI use has been associated with concerns regarding potential adverse effects, including enteric infections, micronutrient malabsorption, renal dysfunction, bone fractures, and possible cardiovascular and neurological risks. Although causality remains debated, these safety concerns have fueled interest in alternative acid-suppressive strategies that may offer
potent efficacy with improved pharmacological predictability and safety profiles [4].

Against this background, potassium-competitive acid blockers (P-CABs) have emerged as a novel class of antisecretory agents designed to overcome several intrinsic limitations of PPIs. Unlike PPIs, which require activation in an acidic environment and bind irreversibly to the proton pump, P-CABs exert their effect by reversibly and competitively inhibiting the potassium-binding site of the H^+/K^+-ATPase. This mechanism allows P-CABs to suppress acid secretion regardless of the pump's activation state, resulting in more rapid, profound, and sustained gastric acid inhibition [14]. Furthermore, P-CABs do not require acid-dependent conversion and demonstrate stable pharmacokinetics, thereby reducing interindividual variability in therapeutic response.

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