

## Comparative Effectiveness of Proton Pump Inhibitors vs. H2 Blockers in Stress Ulcer Prophylaxis for Cirrhotic Patients

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### DESCRIPTION

Stress-Related Mucosal Disease (SRMD) and upper Gastrointestinal (GI) bleeding are recognized complications in critically ill patients, especially those with cirrhosis due to their increased portal pressure, coagulopathy and impaired mucosal defenses [1,2]. The prophylactic use of acid-suppressive therapies, primarily Proton Pump Inhibitors (PPIs) and Histamine-2 receptor blockers (H2 blockers), has become routine in high-risk populations to reduce the incidence of stress ulcers and consequent bleeding [3]. However, in cirrhotic patients, the choice between PPIs and H2 blockers for stress ulcer prophylaxis remains controversial due to varying efficacy, potential adverse effects and influence on infectious complications such as Spontaneous Bacterial Peritonitis (SBP) and Clostridioides Difficile Infection (CDI) [4-6].

This communication aims to summarize recent comparative data on the effectiveness and safety of PPIs versus H2 blockers in stress ulcer prophylaxis among cirrhotic patients, offering insight into current best practices and areas needing further study. Recent observational studies and meta-analyses have highlighted that while both PPIs and H2 blockers reduce gastric acidity and theoretically prevent mucosal injury, PPIs offer superior acid suppression due to their mechanism of inhibiting the H<sup>+</sup>/K<sup>+</sup> ATPase proton pump in parietal cells. This heightened suppression may confer a greater protective effect against stress-related ulcers. However, concerns have arisen regarding PPI-associated risks, particularly infections, given the altered gut microbiome and immune dysfunction in cirrhosis[7].

A large retrospective cohort study by Wiener I, et al.[8] comparing outcomes in cirrhotic patients receiving PPIs versus H2 blockers found no significant difference in the incidence of upper GI bleeding during hospitalization. However, the PPI group demonstrated a statistically significant higher incidence of SBP and CDI, consistent with prior findings. These results suggest that while PPIs might be slightly more effective in acid suppression, their use may predispose patients to infectious complications, which carry substantial morbidity and mortality

in cirrhosis. Randomized Controlled Trials (RCTs) in this domain remain sparse. A small RCT by scientists showed that stress ulcer prophylaxis with ranitidine (an H2 blocker) was associated with fewer infectious complications compared to PPIs, though the trial was underpowered to detect differences in bleeding rates. A systematic review and meta-analysis by Sherid M, et al.[9] encompassing both observational and RCT data echoed these findings, concluding that H2 blockers might offer a safer profile with comparable effectiveness for bleeding prevention.

Pathophysiological insights explain these clinical observations. PPIs induce more profound and sustained gastric hypochlorhydria than H2 blockers, facilitating bacterial overgrowth and translocation a mechanism pivotal in SBP pathogenesis. Cirrhotic patients, already at risk due to impaired intestinal barrier and immune dysfunction, may thus be particularly vulnerable to PPI-related infections. On the other hand, inadequate acid suppression from H2 blockers might risk breakthrough bleeding, especially in advanced cirrhosis with portal hypertensive gastropathy. The clinical dilemma is further complicated by the frequent empirical and prolonged use of acid suppressants beyond ICU stays or the active bleeding period. Several retrospective analyses warn against indiscriminate PPI use in cirrhosis, advocating strict adherence to guideline-based indications and early de-escalation [10].

Current consensus guidelines, including those from the American Association for the Study of Liver Diseases (AASLD), recommend acid suppressive therapy only for patients with clear indications, such as active bleeding or mechanical ventilation longer than 48 hours, but do not explicitly favor PPIs over H2 blockers. Individualized risk assessment is paramount, balancing bleeding risk against infection susceptibility. Given the limitations of available evidence, prospective large-scale RCTs are urgently needed to definitively compare the efficacy and safety of PPIs versus H2 blockers in this population. Additionally, studies evaluating optimal dosing, duration and timing of prophylaxis could inform more nuanced protocols.

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## CONCLUSION

In cirrhotic patients requiring stress ulcer prophylaxis, both PPIs and H2 blockers effectively reduce gastric acid secretion and risk of bleeding. However, PPIs may carry a higher risk of infectious complications such as SBP and CDI, potentially due to more profound acid suppression and consequent bacterial overgrowth. H2 blockers, while possibly less potent in acid suppression, appear to offer a safer profile in terms of infection risk, with comparable bleeding prevention based on limited data. Clinicians in high-income countries should tailor acid suppression therapy in cirrhosis carefully, adhering to evidence-based indications and limiting unnecessary prolonged use. Until robust RCT data become available, a cautious approach favouring H2 blockers or short-course PPI therapy in selected patients may optimize outcomes. Enhanced stewardship of acid-suppressive medications and multidisciplinary management remain essential to balance bleeding prophylaxis against infection risks in this vulnerable population.

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