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SUCRALFATE IN MANAGEMENT OF STRESS ULCERS: EXPLORING ITS ROLE IN ANGIOGENESIS, PROTECTIVE BARRIER FORMATION, AND NON-SYSTEMIC ACTION

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ABSTRACT

Stress ulcers, or stress-related mucosal damage (SRMD), are a common complication in critically ill patients, characterised by erosions, ulcerations, and inflammation of the mucosa of the upper gastrointestinal (GI) tract. Patients in intensive care units (ICUs), particularly those on mechanical ventilation or with severe burns, trauma, sepsis, or coagulopathy, are at increased risk. Within the first 24 hours after hospitalisation, 75–100% of intensive care unit patients experience stress-related ulcers due to an imbalance between protective and aggressive gastric factors. If left untreated, stress ulcers can lead to significant complications, including gastrointestinal bleeding, increased morbidity, and prolonged hospital stays. Sucralfate binds to negatively charged alternatives due to its unique mechanism of action. It forms a protective barrier over ulcerated mucosa, promotes angiogenesis, and enhances mucosal healing without causing significant changes in gastric pH.. This characteristic reduces the likelihood of bacterial overgrowth and infection, making sucralfate a safer option for stress ulcer prophylaxis. Clinical studies have demonstrated that sucralfate effectively prevents stress-related gastrointestinal bleeding while maintaining a favourable safety profile. Sucralfate is added for the treatment regimen along with other acid suppressant drugs like PPIs and H2RAs in the treatment of stress ulcers. Sucralfate, when combined with PPIs and H2RAs, enhances healing and prevents complications by providing protection against acid, pepsin, and bile salts. This combination reduces the risk of bleeding and perforation, leading to faster recovery and shorter hospital stays. However, caution is necessary in patients with renal impairment due to the risk of aluminium accumulation. Given its protective properties, low risk of complications and adverse drug reactions, sucralfate represents a viable and safer add-on therapy for managing stress ulcers in critically ill patients.

KEYWORDS: Sucralfate; Angiogenesis; Stress Ulcer; Gastritis; Stress ulcers prophylaxis.

1. INTRODUCTION

The term "stress ulcers" or "stress-related mucosal damage" (SRMD) refers to the erosion, ulceration, and inflammation of the upper gastrointestinal tract that frequently happens in patients with life-threatening illnesses.[1] The life-threatening illnesses include major surgery or a severe illness that needs intense medical life support to maintain vital organ function and preserve life.[2] The common consequence of these illnesses is stress-induced ulcers.[3] Within the first 24 hours after hospitalisation, 75–100% of intensive care unit patients experience mucosal damage and stress-related ulcers.[4,5] Complications such as gastrointestinal bleeding can also result from stress ulcers.[3,4] According to a study by Young PJ et al., gastrointestinal bleeding occurred in about 2.5% of intensive care unit patients.[6]

An imbalance between defensive mechanisms (such as mucus, bicarbonate, mucosal microcirculation, epithelial lining, and prostaglandins) and aggressive factors (such as gastric acid, pepsin, and bile salts) is typically the cause of stress ulcers.[7,8] Vasoconstriction and splanchnic hypoperfusion are the main pathogenic causes in critically ill individuals. Splanchnic and mucosal perfusion can be affected by any disease that causes hypotension, hypovolemia, or decreased cardiac output (CO). Bicarbonate secretion and mucosal blood flow consequently decline, impairing defences and leading to deeper erosion and injury to the stomach mucosa. The patient's health may worsen as a result of these deeper lesions developing into ulcers and gastrointestinal bleeding, including haematemesis and melena, which can cause anaemia and shock.[7,12] ICU patients who have been on mechanical breathing for longer than 48 hours, as well as those who have coagulopathy, severe burns that cover more than 35% of their body, shock, sepsis, trauma, and other critical situations, are at considerable danger from this condition. To lower the incidence of stress ulcers, patients with certain risk factors need to take prophylactic measures.[1] Stress ulcers are treated with a variety of measures such as enteral feeding, proton pump inhibitors (PPIs), sucralfate, and histamine H-2 receptor antagonists (H2 blockers). By stopping histamine from binding to H2 receptors, which lowers cyclic adenosine monophosphate (cAMP) levels and hydrogen ion (H+) secretion in parietal cells, H2 blockers are usually the first-line treatment to minimise symptoms and prevent complications.[1,9] Proton pump inhibitors (PPIs), on the other hand, lessen stomach acid by blocking the release of hydrogen ions by deactivating the ATPase enzyme on the parietal cell's secretory surface.[9] PPI are used for prophylaxis as well as treatment of complication of stress ulcers Long-term use of these two drugs, however, may raise the risk of Clostridium difficile-induced enterocolitis. An effective choice is sucralfate, which protects the stomach mucosa from the harmful effects of pepsin and acid by attaching to it and creating a cytoprotective barrier.[9] Enteral feeding, which involves delivering nourishment through an enteral tube is an additional strategy to avoid stress ulcers. This method helps shield seriously ill patients from the risk of stress-related mucosal damage & bleeding.[1] Following successful resuscitation, stomach acid-suppressing drugs such as H2 blockers, PPIs, and sucralfate are continued while gastric lavage is carried out to remove clots and relieve gastric distension.[8]

Due to its unique mechanism and safety benefits, sucralfate is an effective add-on to proton pump inhibitors (PPIs) and histamine-2 receptor antagonists (H2RAs) in the treatment of stress ulcers. Whereas PPIs and H2RAs, which decrease the formation of gastric acid, sucralfate forms a protective layer over the ulcer, promoting healing while preserving normal stomach acidity. This technique helps reduce the risk of diseases like pneumonia and Clostridium difficile infection that are connected to acid suppression. Additionally, compared to acid-suppressing medications, sucralfate has been associated with a lower incidence of ventilator-related pneumonia.[10]

2. PHARMACOLOGICAL PROPHYLAXIS OF STRESS ULCER

7.1. Histamine H-2 receptor antagonists

Histamine H-2 receptor antagonists (H2 blockers) for prophylaxis of stress ulcers and its complications in critically ill patients have become the most popular for inhibiting gastric acid secretion. Their main indication is to reduce symptoms and speed up the healing of gastric ulcers.[11,12] Ranitidine and famotidine are the types of drugs most often used in this group. Both drugs are usually given via intravenous (IV) bolus at the same doses. Ranitidine is the most widely studied gastric acid suppressant drug for stress ulcer prophylaxis. Ranitidine, given via IV bolus with a single dose of 50 mg, will reduce gastric acidity levels (pH > 4) for 6–8 hours. Famotidine competitively inhibits histamine at H2 receptors of gastric parietal cells, thereby reducing gastric acid secretion, gastric volume, and hydrogen ion concentration.[11] For a longer duration of action. A single dose of famotidine (20 mg) given via IV bolus will reduce gastric acidity (pH > 4) for 10–15 hours. IV famotidine and ranitidine are predominantly excreted in the urine. The accumulation of these drugs in renal failure can lead to a neurotoxic condition characterised by decreased consciousness, agitation, and even seizures. Therefore, dose reduction is recommended in patients with renal insufficiency.[12] H2 blockers effectively reduce clinically significant GI bleeding as a consequence of stress ulcers. The use of H2 blockers long-term can be accompanied by a decrease in its ability to maintain a pH ≥4, but this does not affect its ability to prevent bleeding related as a consequence of stress ulcers.[9,11,12] The main risks associated with H2 blockers are associated with decreased gastric acidity. Including an increased incidence of infectious gastroenteritis (including enterocolitis clostridium difficile) and an increase in the incidence of pneumonia due to aspiration of gastric secretions that infect the respiratory tract.[12]

7.2. Proton Pump Inhibitor (PPIs)

Proton pump inhibitors (PPIs) are drugs that are potent at suppressing stomach acid by binding to membrane proton pumps in gastric parietal cells and inhibiting the H/K^+-ATPase that catalyzes H and K^+ exchange.[11] PPIs are drugs or prodrugs that must be converted into the active form in the parietal cells of the stomach. Once activated, the drug binds irreversibly to the membrane pump and results in complete inhibition of gastric acid secretion. PPIs are used for prophylaxis of stress ulcers.[12]

As stress ulcer bleeding prophylaxis, PPIs have not shown an advantage over H2 blockers. Increased gastric acid suppression with PPIs may pose a greater risk of infection compared to H2 blockers. This is supported by research showing that the use of PPIs compared to H2 blockers increases the incidence of pneumonia in hospitals as well as the incidence of enterocolitis clostridium difficile higher in outpatients.[11,12]

7.3. Sucralfate

Sucralfate is an aluminum salt of sucrose sulfate that adheres primarily to areas of damaged gastric mucosa, binds to positively charged proteins in the exudate, and forms a viscous fluid that protects the GI lining from luminal acid and pepsin proteolysis.[11] Sucralfate is classified as a protectant or cytoprotective agent and does not affect gastric acid secretion. Sucralfate improves the healing of gastric and duodenal ulcers and reduces the incidence of significant GI bleeding due to stress ulcers.[11,12] Sucralfate is available in tablet (1 gram per tablet) or suspension (1 g/5 mL) form and is most effective when given in suspension form. Sucralfate dose for stress ulcer prophylaxis. Sucralfate will stick to the damaged gastric mucosa for about 6 hours, so it is recommended to administer doses at 6-hour intervals.[12] Sucralfate is advantageous because it does not alter gastric acidity and does not pose the increased risk of infection that

accompanies other gastric acid-suppressing drugs. Several clinical trials have compared sucralfate with ranitidine as stress ulcer prophylaxis. In a clinical trial by Stollman N et al. GI bleeding occurred less frequently with ranitidine use, whereas pneumonia occurred less in sucralfate use. When the incidence of bleeding and pneumonia was combined, adverse events occurred 15.7% less frequently with the addition of sucralfate.[12] Clinical studies show that sucralfate effectively reduces stress-related gastrointestinal bleeding in critically ill patients. Unlike acid-suppressive treatments, it has minimal effect on gastric pH, potentially lowering the risk of nosocomial pneumonia.[13]

3. TREATMENT OF COMPLICATIONS

7.1. Complications of stress ulcer

The most common complications of stress ulcers are gastrointestinal hemorrhage, perforation, penetration, and obstruction of the gastric outlet from the stress ulcer.[14] Excessive bleeding from the stress ulcer can be severe and potentially fatal. Stress ulcers can penetrate the stomach lining, which may lead to perforation, causing the spread of gastrointestinal contents into the peritoneal cavity if not contained by adjacent organs such as the pancreas and liver. It has detrimental effects if not treated within 24 hours. Chemical peritonitis is the initial symptom of this illness, which can develop into bacterial peritonitis. Gastric outlet blockage is another potential consequence that might arise when the pyloric canal narrows as a result of antrum enlargement and scar tissue formation.[7,14]

7.2. Complications due to stress ulcer prophylaxis

Critically ill patients need to be continuously reassessed for stress ulcers and their potential complications during and after their ICU treatment.[3] IVAC (infection-related ventilator-associated complication) refers to infectious complications like nosocomial pneumonia and clostridium difficile diarrhoea. Due to the use of PPI's & other acid-suppressing medicines.[14]

There is ongoing debate over the relationship between preventing stress ulcers and the risk of infectious complications, specifically ventilator-associated complications (IVAC) and Clostridium difficile infections.

IVAC may result from aspiration of oropharyngeal microorganisms into the lower respiratory tract when refluxed stomach fluids have contaminated the oropharyngeal region. Drugs that increase the pH of the stomach may encourage the growth of harmful bacteria, raising the risk of nosocomial pneumonia.[14]

Using drugs that suppress stomach acid raises the risk of enterocolitis due to Clostridium difficile because they decrease stomach acidity, which makes the gastrointestinal tract more alkaline; this facilitates the growth of Clostridium difficile, which cannot survive in acidic environments but thrives in alkaline ones ($pH < 4$).[14] When proton pump inhibitors (PPIs) are used instead of H2 blockers, the incidence of Clostridium difficile enterocolitis is higher in both outpatients and inpatients. The use of stomach acid-suppressing drugs for stress ulcer prevention is more likely to be the cause of the higher incidence of nosocomial pneumonia in hospitalised patients than antibiotics.[12]

According to Buckley, 14.4% of intensive care unit (ICU) patients received stomach acid suppression medication without the appropriate indications, which could cause adverse effects and increase complications.[15] Stress ulcers can be identified and treated to help lower expenses, length of hospital stay, and other consequences. However, only individuals with recognised risk factors for stress ulcer development should routinely use pharmacological stress ulcer prophylaxis to prevent any potential complications.[16]

4. ACTION OF SUCRALFATE IN STRESS ULCER

Sucralfate is recognised as an alternative to histamine-2 receptor antagonists (H2RAs) for stress ulcer prophylaxis in critically ill patients due to its cytoprotective properties, forming a barrier over the gastric mucosa to prevent ulcer formation (EAST, 2008). Unlike acid-suppressive therapies, sucralfate has minimal impact on gastric pH, which may help reduce the risk of ventilator-associated pneumonia (VAP) by limiting bacterial overgrowth (EAST, 2008). However, clinicians should be aware of its potential to impair the absorption of other medications, particularly when administered with enteral nutrition (VUMC, 2017). Given these factors, sucralfate remains a viable option, particularly for patients at risk of VAP or those for whom acid suppression is contraindicated (EAST, 2008).[17] Sucralfate, taken along with H2 receptor antagonists, is a better option than proton pump inhibitors (PPIs) and H2 receptor antagonists (H2RAs) for treating stress ulcers because it acts through various mechanisms listed below.

7.1. Angiogenesis and Mucosal Promotion

There is clear evidence that sucralfate promotes angiogenesis, or the growth of new blood vessels, during the healing process of stress ulcers. Although sucralfate improves ulcer healing by acting as a barrier and maybe promoting growth factors, the specific effect it has on angiogenesis has not been conclusively determined in the research. It is clinically recognised as an effective alternative for preventing stress ulcers and is linked to a lower incidence of nosocomial pneumonia. Based on research by Korman MG et al. on sucralfate, it enhances stress ulcer healing through a variety of pathways; however, it is unclear how directly it promotes angiogenesis.[18] On top of that, a study by Sandor Szabo, M.D., et al., on the role of vascular factors, including angiogenesis, in the mechanisms of action of sucralfate showed that in rats with subcutaneously implanted sponges, sucralfate promoted angiogenesis and the development of granulation tissue. It's interesting to note that sucralfate was less successful than pure basic fibroblast growth factor (bFGF) at promoting these processes than its water-soluble counterpart, sucrose octasulfate.[19] According to these findings, sucralfate works by promoting angiogenesis, which contributes to its effectiveness in ulcer healing.

According to a study by Tsukamoto et al., angiogenesis in the granulation tissue of stomach ulcers was significantly increased by sucralfate treatment. Better healing results were associated with the increased formation of new blood vessels, suggesting that sucralfate's capacity to stimulate angiogenesis may be a contributing factor in its efficacy in treating stomach ulcers. Sucralfate helps stomach ulcers heal more quickly and effectively by protecting the tissue and supporting the growth of new blood vessels.[20]

7.2. Protective Barrier Formation

Sucralfate's special capacity to create a barrier that protects injured mucosa makes it a popular treatment for stress ulcers. It undergoes cross-linking in an acidic environment (pH < 4), forming a gel-like material that adheres to albumin and fibrinogen proteins at the ulcer site. As a result of this interaction, the ulcer is protected from dangerous substances like bile salts, pepsin, and stomach acid by a stable, insoluble covering that sticks to it. In addition to preventing additional irritation, sucralfate facilitates the ulcer's natural healing process by functioning as a physical barrier.[21,22]

7.3. Does not Alter Gastric PH

Beyond its barrier role, sucralfate has protective effects on the gastrointestinal lining by neutralising gastric acid, preventing pepsin activity, and absorbing bile salts. Sucralfate facilitates efficient ulcer healing without appreciably altering the general acidity of the stomach by establishing this localised defence and preserving the integrity of the gastric environment.[22]

7.4. Minimal Absorption

Sucralfate is a locally acting drug primarily used to treat duodenal ulcers. Its effects are limited to the gastrointestinal system, and the bloodstream absorbs very little of it.[21] Sucralfate helps the gastrointestinal mucosa's defences naturally. By encouraging the creation of prostaglandins, which are necessary for the mucosal lining's defence and healing. It is more successful at healing ulcers and preventing recurrence because of this protective activity.[23] There is less chance of systemic side effects with sucralfate because it is not as readily absorbed into the bloodstream.[23]

5. USES OF SUCRALFATE IN CRITICALLY ILL PATIENTS

A unique anti-ulcer drug called sucralfate is an aluminium complex of sucrose octasulfate. By forming a barrier, sucralfate protects the stomach mucosa from bile salts, pepsin, and pectic acid. A thick, gel-like material forms at the location as a result of its interaction with positively charged proteins in exudates.[24] Sucralfate has been found to decrease the occurrence and severity of dyspeptic symptoms and gastric erosion associated with NSAID therapy, demonstrating effectiveness comparable to that of an H2 receptor antagonist.[25]

Sucralfate is approved by the FDA for the short-term treatment of duodenal ulcers, with a recommended duration of up to 8 weeks. The standard dosage is 1 g taken four times daily for 8 weeks, followed by a maintenance dose of 1 g. Its effectiveness in treating duodenal ulcers has been found to be similar to that of cimetidine and high-dose antacid therapy.[24]

Uses of sucralfate in critically ill patient are listed below:

7.1. Stress ulcer prophylaxis in ventilated patients

Research has shown that sucralfate prevents stress ulcers in patients receiving ventilator therapy better than antacids or H2 blockers. H2 blockers and antacids, in contrast to sucralfate, elevate the pH of the stomach, which causes gram-negative bacteria to stagnate and increases the risk of nosocomial pneumonia.[26]

7.2. Radiation proctitis

Sucralfate paste enema has shown effectiveness in improving hemorrhagic radiation proctitis. The treatment's benefits were assessed using clinical proctitis ratings before and after administration, utilising a low-volume paste in an enema applicator. The outcomes were favourable.[27] For the short-term treatment of radiation proctitis, Kocchar et al.'s research showed that a sucralfate enema works better than sulfasalazine used orally.[28]

7.3. Chemotherapy-induced mucositis

According to a McCullough study, high-potency sucralfate helps alleviate chemotherapy-induced mucositis in the digestive system and oropharynx by accelerating the activation of growth factors. When mucositis first appeared, 1.5 g of sucralfate was taken three times a day for two days. After that, 1.5 g was given twice a day for the duration of favourable therapy and for two weeks after it ended.[28]

7.4. Epithelial wounds

Additionally, sucralfate has been applied topically to a variety of epithelial wounds, such as burn injuries, inflammatory dermatitis, ulcers, and mucositis. Its efficacy in repairing epithelial lesions has been the subject of numerous investigations. According to research by Tsakayannis et al., topical sucralfate treatment improved venous ulcers that were not improving with traditional treatments.[29]

Sucralfate enhances the bioavailability of growth factors, especially fibroblast growth factor (FGF), which plays a crucial role in angiogenesis and supports the healing of epithelial wounds.

7.5. Gastro-esophageal reflux disease (GERD)

GERD can worsen in critically ill patients due to radiation, lying down position, and nasogastric tubing. According to the American College of Gastroenterology guidelines, sucralfate is a recommended treatment for GERD in pregnant women.[30]

6. Combining Therapies for Stress-Related Ulcers

Sucralfate & H2RAs for Stress Ulcers: A combination therapy that includes sucralfate and histamine-2 receptor antagonists (H2RAs) has shown potential in the treatment of stress ulcers. Sucralfate creates a protective barrier around ulcers, whereas H2RAs inhibit stomach acid production. This regime promotes healing, minimises bleeding risk, and improves patient outcomes, especially in critically ill patients. Notably, a study by Cooper et al. indicated that sucralfate was more effective than H2RAs at avoiding pneumonia in intubated patients.[31]

The American Society of Health-System Pharmacists suggests sucralfate as an alternative to H2RAs for stress ulcer prevention in critically ill patients.[32]

7.1. Sucralfate & Proton Pump Inhibitors (PPIs) for Stress Ulcers: The combination of proton pump inhibitors (PPIs) and sucralfate has proven to be an effective strategy for preventing stress ulcers. PPIs help by reducing stomach acid production, which decreases the acidic environment that can worsen ulcer formation. Sucralfate, on the other hand, works by creating a protective barrier over the ulcers, preventing further damage and promoting healing. This regime approach offers several benefits, such as improved ulcer healing and prevention, reduced risks of bleeding and perforation, and better outcomes for critically ill patients, who are particularly prone to these complications.[33]

7. Role of Sucralfate in Management of Stress Ulcer

7.1. Cost-Effective Treatment of Stress Ulcer

Sucralfate oral suspension is a cost-effective option for preventing stress ulcers because it is affordable and works well by creating a protective layer over the stomach lining.[34]

Proton Pump Inhibitors (PPIs) are effective in reducing stomach acid and preventing bleeding, but they can be expensive, vary in price, and may cause side effects like nosocomial pneumonia or electrolyte problems.[34,35]

Histamine-2 Receptor Antagonists (H2RAs) are less expensive than PPIs and provide moderate effectiveness, but their impact may decrease with continued use due to tolerance (tachyphylaxis). Their value for money can change based on the brand and the specific clinical situation.[35]

Guidelines on the use of sucralfate in the management of stress ulcers:

7.2. According to EAST

The Eastern Association for the Surgery of Trauma (EAST) recommends stress ulcer prophylaxis for patients at high risk, such as those on mechanical ventilation, with coagulopathy, traumatic brain injury, or similar serious conditions. While Sucralfate is an effective option, its ability to reduce gastrointestinal bleeding is still uncertain. Alternatives include proton pump inhibitors (PPIs) and histamine-2 receptor antagonists (H2RAs).[36]

Prophylaxis should be continued for the duration of the critical illness or ICU stay. However, sucralfate may interfere with the absorption of other medications and could promote gastric bacterial growth. Additionally, in patients with kidney problems, it may lead to aluminium toxicity, so caution is advised.[36]

7.3. Alberta Guidelines

Stress ulcer prevention (SUP) is very important in pediatric intensive care units (PICUs) to stop stomach bleeding in seriously ill children. Some factors, like being on a ventilator for a long time, having blood clotting problems, or organ failure, can increase the risk of this kind of bleeding. Medications like proton pump inhibitors (PPIs) and histamine-2 receptor antagonists (H2RAs) help by lowering the amount of acid in the stomach. Another option, sucralfate, works by creating a protective layer over any ulcers. For children at high risk, PPIs or H2RAs are usually recommended. Overall, SUP plays a key role in protecting critically ill children from gastrointestinal bleeding.[37]

7.4. According to ASHP & SCCM Guidelines

The American Society of Health-System Pharmacists (ASHP) guidelines do not list sucralfate as a recommended option for stress ulcer prevention. Instead, they suggest proton pump inhibitors (PPIs) such as pantoprazole and histamine-2 receptor antagonists (H2 blockers) like famotidine as the preferred choices. This indicates that sucralfate is not considered a first-line therapy for stress ulcer prophylaxis based on current recommendations. The Society of Critical Care Medicine (SCCM) and the American Society of Health-System Pharmacists (ASHP) guidelines do not recommend sucralfate as a first-choice treatment for preventing upper gastrointestinal bleeding (UGIB) in critically ill patients. Instead, they advocate the use of proton pump inhibitors (PPIs) or histamine-2 receptor antagonists (H2RAs) for patients with risk factors for UGIB.[38]

8. Safety and Efficacy of Sucralfate

Sucralfate is frequently used to treat stress ulcers, particularly in patients who are very sick. It helps prevent the formation of ulcers by covering the stomach lining with a protective layer. According to research, sucralfate offers protection comparable to that of H2 blockers and, in certain situations, a placebo, even though proton pump inhibitors (PPIs) may be efficient in decreasing serious gastrointestinal bleeding. Since sucralfate doesn't substantially alter stomach acidity, which helps prevent bacterial overgrowth and respiratory infections, it has a reduced risk of hospital-acquired pneumonia than PPIs. However, because sucralfate includes aluminium, which can accumulate in the body, people with kidney disease must use caution.[39] All things considered, it's still a secure and practical way to avoid stress ulcers. especially in the appropriate patient populations.[39] In this experiment, the most frequent adverse effects linked to sucralfate were headache (3.4%) and constipation (3.2%). This experiment did not result in any recorded deaths. Test medicine did not cause any abnormalities in the laboratory.[41]

9. Adverse Effects of Prolonged Use of Proton Pump Inhibitors (PPIs), H2 Receptor Antagonists, and Sucralfate

9.1. Sucralfate

In research, the most frequently reported adverse effects linked to sucralfate were headache (3.4%) and constipation (3.2%).[41]

9.2. Proton Pump Inhibitors (PPIs)

Prolonged use of PPIs can lead to osteoporosis, nutritional deficiencies, kidney disease, and possibly dementia because the decreased stomach acid affects gut health and absorption. Sucralfate reduces these risks by providing ulcer prevention without changing the pH of the stomach. PPI use must be regularly evaluated in order to avoid long-term adverse effects.[42]

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9.3. H2 Receptor Antagonists (H2RAs)

Although long-term usage of H2 receptor antagonists (H2RAs) is usually well tolerated, it can cause hormonal abnormalities, intestinal problems, pneumonia, and cognitive impairment in older adults. Frequent evaluation is recommended to decrease possible dangers.[43]

10. CONCLUSION

In conclusion, sucralfate represents a viable and safer alternative for managing stress ulcers in critically ill patients. Its unique mechanism of action, which promotes angiogenesis, protective barrier formation, and non-systemic action, makes it an effective add-on to PPIs and H2RAs in the treatment of stress ulcers. The reduced risk of hospital-acquired pneumonia and other complications associated with acid-suppressive therapies further supports the use of sucralfate in stress ulcer prophylaxis. However, caution is necessary in patients with renal impairment due to the risk of aluminium accumulation. Overall, sucralfate is a valuable treatment option for stress ulcers, offering a favourable balance of efficacy and safety.

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