IJNRD.ORG ISSN : 2456-4184
INTERNATIONAL JOURNAL OF NOVEL RESEARCH AND DEVELOPMENT (IJNRD) | IJNRD.ORG
An International Open Access, Peer-reviewed, Refereed Journal

A REVIEW ARTICLE OF SUCRALFATE SUPRESSES HELICOBACTER PYLORI INFECTED AND REDUCES GASTRIC ACID SECRETION BY 50% IN PATIENTS WITH DUODENAL ULCER

[1]NABANITA BIJOY DAS, [1]M-PHARMA(PHARMACEUTICS)

[1]Department of pharmaceutics,
[1]Hillside college of pharmacy, Bangalore-560062, Karnataka, India

ABSTRACT

Background and Objectives:

Sucralfate[1] is a drug used to treat various medical conditions, including duodenal ulcers[2]. Sucralfate creates a protective barrier in the stomach, boosts bicarbonate production, and exhibits properties that help in tissue repair and reduce the impact of pepsin. This article explores the wide-ranging uses of sucralfate, including its effectiveness, how it works, ways it's administered, and how to monitor its use. The main goal of the study was to assess how sucralfate affects *Helicobacter pylori* (H. pylori) and the excessive gastric acid secretion it triggers in patients with duodenal ulcers.3

Sucralfate, a basic aluminum salt derived from sucrose octasulfate, was designed to neutralize both stomach acid and pepsin. Its chemical makeup differs from other sulfated compounds because it's a base and originates from the disaccharide sucrose. Early research showed that sulfated disaccharides lacked the blood-thinning effects of sulfated polysaccharides and that ulcer protection was tied to their sulfation levels. Sucralfate has been more effective in protecting against ulcers in animal studies than a combination of sucrose octasulfate and aluminum hydroxide.

One of sucralfate's unique features is its transformation into a thick, viscous gel when it comes into contact with gastric acid, allowing it to buffer the acid. This protective function continues in the duodenum. It forms stable bonds with proteins, blocking their breakdown by inhibiting the

interaction between pepsin and the proteins. Additionally, sucralfate directly absorbs pepsin and bile salts, providing a robust defense against the harmful effects of acid, pepsin, and bile.
Research on *Helicobacter pylori* has confirmed the bacterium's significant role in causing and recurring peptic ulcers, prompting many treatment strategies aimed at eradicating it.A recent trend in curative therapy has been so-called triple therapy, using a proton pump inhibitor and two different antimicrobials. Sucralfate, which is a widely used cytoprotective agent for the gastric mucosa, is reported to inhibit several of the activities of H. pylori and to enhance the anti-H.pylori activity of antimicrobial agents. Therefore, several studies of sucralfate-based eradication therapy have been reported recently. However, the efficacy and safety of sucralfate-based therapy are still controversial. The present study was designed to evaluate the efficacy and safety of sucralfate in combination with amoxicillin and clarithromycin as eradication therapy for H. pylori, in comparison with lansoprazole-based triple therapy.

Methodology:
Basal and gastrin- releasing peptide (GRP)[4] stimulated gastrin release and acid secretion. H. Pylori density gastric unease activity and severity of gastritis were studied in patients with duodenal ulcer who were positive for H. Pylori before, during and after 4 weeks treatment with sucralfate (2g twice daily).

Conclusion:
These findings indicate that sucralfate markedly suppresses H.pylori infection and accompanying hypersecretion[5] of acid and the infection induced in patients with duodenal ulcer .These effect are likely to be important mechanism by which the drug promotes duodenal ulcer healing.

Key words: *SUCRALFATE;* Duodenal Ulcer; H. Pylori; H2 receptar blocker[4]; Histopathology.

INTRODUCTION:

Sucralfate is an effective therapeutic agent in the treatment of duodenal ulceration achieving healing rates comparable with those of H2 receptor blocker [6]. However the relapse rate after duodenal ulcer (DU) healing with sucralfate is lower than that after healing with H2 receptor blocker. Sucralfate therapy increases secretion of gastro duodenal bicarbonate and gastric mucus and alters the physico- chemical properties of mucus leading to enhance protective effect. Sucralfate also enhances gastric in a mucosal blood flow and vascular integrity[7]. Finally an important protective mechanism lies in ability to increase proliferative activity by enhancing the effect of epidermal growth factor and mutagen on the gastric mucosa

In the view of above effect sucralfate traditionally has been classified within the site protective or cyto protective group of ulcer healing drugs which have been assumed to heal peptic ulcer by mechanism independent of acid suppression. However some recent studies have suggested that sucralfate may inhibit acid secretion.

A study has evaluated nocturnal acid output which decreased after ulcer healing with sucralfate[8]. However, more recent work by "slomiany et al" has suggested that sucralfate is also able to increase the expression of epidermal growth factor and platelet derived growth factor receptor in the rat gastric mucosa.[9]

PATIENTS AND METHODS:

9 patients (7 man)with H.pylori positive disease were studied. Their age range from 19 to 56 years, each had ulcer confirm by endoscopic examination and everyone had receive H2 receptor therapy.

All 9 patient where studied at entry and on the final day of 4 week treatment with sucralfate. 6 patients where reassessed on 3rd occasion. On each of the study morning, patient swallowed 50 ml of water at 6:00 a.m. On the second day of study the patient swallowed 2 tablet of sucralfate with 20 ml of water.

ENDOSCPIC EXAMINATION:

All patients were induced with IV midazolam[10-12] under mild sedation and underwent endoscopy. To confirm the patient H. Pylori status initially an antral biopsy specimen was taken for urease slide test and endoscopic biopsy specimen for histopathology analysis was obtained from the gastric antrum in the next test.

- **ANALYSIS OF PLASMA GASTRIN CONCENTRATION:**

The sample of blood for plasma gastrin analysis was collected into heparin tube at -20 degree centigrade. Plasma gastrin level were estimated by radio immuno essay using antibody R-98 which has a lower limit of detection of 5- 10 mg\L.[13]

Histopathology: Antral biopsy specimen was assessed t for the presence, type and intensity of inflammatory infiltrate and for the density of H.pylori colonization.[14-15]

Statistical analysis: It was perform using Wilcoxon test[16], AP value of less than 0.05 was considered significant.

Results: None of the patient had active DU at any of the endoscopic examination.

- **BASAL GASTRIN CONCENTRATION** :

Basal gastrin concentration at entry was similar to concentration after 4 week of treatment with sucralfate.

Basal Acid Output: The median basal acid output decreased from 5.2 mmol/h at entry to 2.5 mmol/h after 4 week of treatment with sucralfate. For the 6 patients studied on three occasions, median basal acid output decreased from 4.6 mmol/h at entry to 1.9mmol/h.

Results: The basal acid output 3 weeks after the withdrawal of treatment did not differ significantly from that at entry.

- **ACID RESPONSE TO GRP:**

The Median GRP stimulated gastric acid output decreased from 29.2 mmol/h to 16.4 with treatment. For the 6 patient studied on three occasion, the median GRP stimulated acid output decreased from 31.4 mmol/h at entry to 14.3 mmol/h.[17, 18, 19]

Histology: The antral gastritis score[20] at entry (5-8) was similar to the after treatment (4-7) and after withdrawal of the treatment

RESULT:

The GRP stimulated acid output 3 weeks after cessation of treatment was still slightly lower than that at entry into the study (P =0.013

DISCUSSION

From the above studies we can conclude that sucralfate decreases both basal and GRP stimuli secretion[21]. Also we notice that there was significant increase in acid secretion after discontinuing the sucralfate therapy which confirms from that initial acid suppression was effect of the drug.
Also in previous studies it was not clear that whether the change in acid secretion due to sucralfate cause the effect of ulcer healing. But in the present study we included only patient with healed ulcer, therefore the change in acidic secretion cannot be attributed to change in acidic status.[22, 23]
In addition to suppression of acid secretion, it also showed suppression in the density of H.pylori colonization. The decrease in colonization density in our studies was supported by suppression of urease activity. The mechanism by which sucralfate give such activity is unclear however it is confirmed that it has antibacterial effect on E.coli.[24]
Decrease in acid secretion in patient with ulcer is secondary to drug suppression and bacterium itself is responsible for increased acid secretion by H.pylori infection. This shows that the efficacy of sucralfate is suppress of infection and decrease in acid secretion.[25]
In conclusion this studies indicate that sucralfate significantly decreases H.pylori infection which account for decrease acidic secretion and also in patients with DU, sucralfate significantly decreases both basal and GRP stimulated acid.

Conclusion:

These findings suggest that sucralfate significantly reduces H. pylori infection and the associated acid hypersecretion in patients with duodenal ulcers. This effect is likely a key mechanism through which the drug aids in the healing of duodenal ulcers.

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