

# Treatment of peptic ulcer disease with sucralfate: As study of 50 cases

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## Abstract

**Introduction:** Stress related mucosal damage (SRMD), also called stress ulcers, is the cause of upper gastrointestinal bleed in critically ill patients. Head injuries account for majority of stress ulcers. Endoscopically, these appear as multiple erosions or sub mucosal hemorrhages. The risk of bleeding is directly proportional to the severity of stress. 10-20% of patients can have massive bleeding if no prophylactic therapy is given. Pathophysiologic ally, CNS-related stress ulcer has more gastric acidity and higher gastrin levels with normal mucosal barrier as compared to non-CNS related stress ulcer. As bleeding from SRMD is difficult to treat, prophylaxis reduced the relevant clinical consequences. The most important aspect is to treat the underlying cause. Antacids, H<sub>2</sub>-blockers, sucralfate and proton pump inhibitors have been tried, either alone or in combination. **Aims and Objective:** To study the effectiveness of Sucralfate in the treatment of Peptic Ulcers. **Methodology:** This was a case series of 50 cases at tertiary health care in year 2014 were studied. All these 50 known patients of Peptic ulcer disease were diagnosed by endoscopy. All the patients who have given informed written consent were included into study except patients having hypophosphatemia severe renal defect. Graph Pad Prism software was used for statistical analysis **Result:** At the beginning of the treatment the size was 13.1±5.1mm, At 8 week's size was 7.8±4.5mm. 12 weeks the mean size was 3.2±1.2mm. 16 weeks the mean 1.2±.82mm. The reduction in size of the ulcer was significantly lesser than it previous size i.e. (in between 0 wks to 8 wks; P<0.001, t=5.85, df=98: 8wks to 12 wks; P<0.001, t=7.78, df=98: 12 wks to 16 wks; P<0.0001, t=9.73, df=98. Most common side effect of the Sucralfate was Constipation (44%) followed by Headache (20.00%), Nausea Vomiting (12%), Dizziness (12.00%), Indigestion (12%). **Conclusion:** Sucralfate significantly reduces size of the ulcer if it used long duration, so it should be used in the maintenance therapy or prophylactic therapy of the treatment Peptic ulcers.

**Key Words:** Sucralfate, Gastric Ulcer.

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## INTRODUCTION

Stress related mucosal damage (SRMD), also called stress ulcers, is the cause of upper gastrointestinal bleed in critically ill patients. Head injuries account for majority of stress ulcers. Endoscopically, these appear as multiple erosions or sub mucosal hemorrhages. The risk of bleeding is directly proportional to the severity of stress. 10-20% of patients can have massive bleeding if no prophylactic therapy is given. Pathophysiologic ally,

CNS-related stress ulcer has more gastric acidity and higher gastrin levels with normal mucosal barrier as compared to non-CNS related stress ulcer<sup>1</sup>. As bleeding from SRMD is difficult to treat, prophylaxis reduced the relevant clinical consequences<sup>2,3</sup>. The most important aspect is to treat the underlying cause<sup>4</sup>. Antacids, H<sub>2</sub>-blockers, sucralfate and proton pump inhibitors have been tried, either alone or in combination<sup>5, 6</sup>. It is a complex. Salt of sucrose sulphate and aluminium hydroxide, which is poorly soluble in water and minimally indilute acids and alkalies. When dissolved in stomach contents, after releasing aluminum salt it becomes strongly negative and combines with mucin to form a viscous suspension that binds with normal as well as defective mucosa. It binds pepsin but lacks anti-ulcer efficacy<sup>7,8</sup>. A combination of different actions enables sucralfate to prevent mucosal injury, these are its antiseptic effect, acting as a physical barrier, increasing the production, viscosity, hypophosphatemia and aluminum, carbohydrate content of mucosa making it more acid resistant. It also promotes

prostaglandin mediated and independent bicarbonate output from gastric and duodenal mucosa Its effect on tissue growth regeneration and repair is also contributory<sup>7-10</sup>. Sucralfate has been reported as a safe drug during last 10 years of its use<sup>11,12</sup>. No systemic toxicity, teratogenicity or tumor producing effect has been reported, except hypophosphatemia and aluminum intoxication in patients with renal defect<sup>13, 14</sup>.

## METHODOLOGY

This was case series of 50 cases of tertiary health care in year 2014 were studied. All these 50 known patients of peptic ulcer disease were diagnosed by endoscopy All the assessment of the Gastric ulcer Done by the digital Fiber Optic Endoscopy and response to the treatment in the form of the size of the ulcer were noted. Graph Pad Prism software was used for statistical analysis:

## RESULT

**Table 1:** Distribution of the patients as per Average Size of Ulcer with Time since treatment

Time since treatment (in Weeks)	Average Size of Ulcer (n=50) (Mean±SD)	
At the Starting(0 weeks)	13.1±5.1mm	P<0.001, t=5.85,df=98.
8 weeks	7.8±4.5mm	P<0.001,t=7.78,df=98.
12 weeks	3.2±1.2mm	P<0.0001,t=9.73,df=98.
16 weeks	1.2±.82mm	P<0.0001,t=5.71,df=98.

From **Table 1:** It is clear that at the beginning of the treatment the size was 13.1±5.1mm, At8 week's size was7.8±4.5mm.12 weeks the mean size was 3.2±1.2mm. 16 weeks the mean 1.2±.82mm. The reduction in size of the significantly lesser than it previous size i.e. (in between 0 wks to 8 wks;P<0.001, t=5.85,df=98: 8wks to 12 wks;P<0.001,t=7.78,df=98: 12 wksto 16 wks; P<0.0001,t=9.73,df=98.

**Table 2:** Distribution of the patients with respectto the side effects reported by them

Side effects	Group A (n=50)
Constipation	22(44%)
Nausea Vomiting	6 (12%)
Headache	10(20.00%)
Dizziness	6 (12.00%)
Indigestion	6 (12%)
Total	50(100%)

From **Table 2:** It is clear that Most common side effect of the Sucralfate was Constipation (44%) followed by Headache(20.00%), Nausea Vomiting(12%), Dizziness(12.00%), Indigestion(12%).

## DISCUSSION

A combination of different actions enables Sucralfate to prevent mucosal injury; these are its antiseptic effect, acting as a physical barrier, increasing the production, viscosity, hypoacidity and aluminum, carbohydrate content of mucosa making it more acid resistant. It also promotes prostaglandin mediated and independent bicarbonate output from gastric and duodenal mucosa its effect on tissue growth regeneration and repair is also contributory<sup>7-10</sup>. In our study It is clear that at the beginning of the treatment in 13.1±5.1mm, At 8 week's size was7.8±4.5mm.12 weeks the mean size was 3.2±1.2mm. 16 weeks the mean 1.2±.82mm. The reduction in size of the significantly lesser than it previous size i.e. (in between 0 wks to 8 wks;P<0.001, t=5.85,df=98: 8wks to 12 wks;P<0.001,t=7.78,df=98: 12 wks to 16 wks; P<0.0001,t=9.73,df=98. The size of the ulcer significantly decreased over weeks after the treatment so long term treatment with sucralfate significantly decreases the ulcer size and helps in healing the ulcer this can be explained that; because of its antibacterial activity as peptic ulcer are recurrent mainly due to the infection of H.Pylori infection, so it is more effective in treatment of the peptic ulcer and as it works after long duration of treatment so it should be used in the maintenance therapy or prophylactic therapy of the treatment Peptic ulcers.

## CONCLUSION

Sucralfate significantly reduces size of the ulcer if it used long duration, so it should be used in the maintenance therapy or prophylactic therapy of the treatment Peptic ulcers.

## REFERENCES

1. Bowen CJ, Fleming WH, Thompson JC. Increased gastrin release following penetrating central nervous system injury.Surgery 1974; 75:720-4.
2. Zuckerman GR, Shuman R. Therapeutic goals and treatment options for prevention of stress ulcer syndrome. Am J Med 1987; 83:29-35.
3. Mahapatra AK, Tandon PN, Bhatia R, et al. Head injury patients who talked and died (Analysis of 35 patients). Indian J Surg 1993; 55:361-6.
4. Cheung LY. Pathogenesis, prophylaxis and treatment of stress gastritis. Am J Surg 1988; 156:437-40.
5. Tryba M. Prophylaxis of stress ulcer bleeding. J ClinGastroenterol 1991; 13:S44.
6. Coker A, Yüzer Y, Yetgin S, et al. Stress ulcer prophylaxis: a comparison of four agents. Brit J Surg 1995; 82:44-5.
7. Nagaahima. R. and Sam Loff. I.M. Aggressive factors. II. Pepsin, in peptic ulcer disease. Edited by F.P. Brooks. S. Cohen. R.D. Soloway. Edinburgh. churchillLivingstonc. 1985; pp. 181-214.

8. Nagashima, It and Yoshida, N. Sucralfate, a basic aluminium salt of sucrose sulfate. I. BehavioursingastroduodenalpH.Aetneimittelforachung. 1979; 29:1668-76.
9. Szerbo, S. and Hollander, D. Pathways of gastrointestinal protection and repair: mechanism of action of sucralfate. Am.J.Med., 1989; 80 (suppl. 6A): 23-31.
10. Iouander, D. and Tarawski, A. The protective and therapeutic mechanism of sucralfate. i. Gastroenterol., 1990; 173 (suppl): 1-S.
11. Ishimori, A. Safety experience with sucralfate in Japan. i. cun. Gastroenterol., 1981; 3 (suppl.2) 169-73.
12. Fisher, ItS. Sucralfate: a review of drug tolerance and safety. J. Clin. Gastroenterol. 1981; (suppl. 2):181-4.
13. Robertson, J.A., Salusky, I.B., Goodman, W.G., Norris, N.E. and Coburn, J.W. Sucralfate. intestinal aluminium absorption and aluminium toxicity in a patient on dialysis. Ann. Intern. Med., 1989; 111:179-81.
14. Leung, ACT. Henderson, S. Macher, R., Dobbie, I. Halls, D. and Fell, G. Once daily aluminium containing phosphate binding agent in dialysis patients. J.Int. Biomed. Info. Data, 1984; S: 17-22

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