The comparative effects of oral famotidine and lansoprazole in prophylaxis of aspirin induced peptic ulcer in albino rat

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ABSTRACT

Background: Peptic ulcer is defined as any break in the continuity of gastric or duodenal epithelial layer. There are mainly three factors which are responsible for peptic ulcer disease which are Helicobacter pylori infections, NSAIDs and stress. Famotidine is H₂ receptor blocker and Lansoprazole is proton pump inhibitor which are used prophylactically in aspirin induced peptic ulcer.

Methods: The experimental work was carried out on albino rat. Experiment was carried out with two ulcer protecting agents e.g.-famotidine, lansoprazole and one ulcer producing agent Aspirin. Each ulcer protecting drug was used separately but simultaneously with aspirin to see their ulcer protecting efficacy. There was three groups of animals each consisting of ten albino rats. Ist group was control group which was given aspirin only.2nd group was given aspirin+famotidine, 3rd group was given aspirin+lansoprazole.

Results: The prophylactically ulcer preventing activity was the most with lansoprazole and least with famotidine.

Conclusions: Finally, all the two drugs like famotidine, lansoprazole prevented the ulcerogenic effects of Aspirin effectively, although not completely.

Keywords: NSAIDs, Helicobacter pylori, H₂ Receptor blockers, Proton pump inhibitors

INTRODUCTION

Peptic ulcer is an ulceration of GIT due to continued corrosive action of HCL and pepsin of gastric juice on the gastric mucosa. Aspirin causes gastro-intestinal damage in more than 80% of subjects varying from acute microscopic gastric changes to potentially more serious chronic gastric ulceration or haemorrhages leading to haematemesis or melena. The exact etiology of peptic ulcer is still not properly understand. There are several other factors like hormonal, mechanical, gastric, immunological and food habit also play secondary roles in the causation of ulcers. Stress and strain of modern life also increases the susceptibility of gastric and duodenal mucosa to the corrosive effect of gastric juice. Warren et al have found evidence that Helicobacter pylori is strongly associated with gastritis and peptic ulcer.¹ At present so many drugs are available for reduction of acid secretion like cimetidine, ranitidine, famotidine, omeprazole, lansoprazole pirenzepine etc. Hawkey have studied on cases of bleeding induced by aspirin.² They found in their study that inhibition of acid secretion represents the best strategy to reduce gastric mucosal damage and bleeding.
METHODS

Adult albino rats of either sex of 200 to 300 gm weight was taken. Experiment was carried out with two ulcer protecting agents eg. famotidine and lansoprazole and one ulcer producing agent-Aspirin. Each ulcer protecting drug was used separately but simultaneously with aspirin to see their ulcer protecting efficacy. There was three groups of animals each consisting of ten albino rats. To each group of animals drugs was given as follows; Group 1 was Aspirin only (as control), Group 2 was Aspirin+Famotidine and Group 3 was Aspirin+Lansoprazole.

Aspirin was used in dose of 100mg/kg/body weight of all groups. Famotidine was used in dose of 12mg/kg/body weight of 2nd group animals. Lansoprazole was used in dose of 3 mg/kg/body weight of 3rd groups. All the drugs were used with help of double distilled water. To each group of animals required doses of drugs was given orally in the empty stomach in the morning daily for three days continuously. Before administration of drugs animals were made unconscious with ether to prevent fighting during drug administration. Drugs were administered in the stomach of animals through neonatal Ryle’s tube. Food like grass, bread etc. was allowed after four hours of day administration. On the fourth day all animals of each group were sacrificed. The abdomen was opened in mid-line and stomach with duodenum was removed. The stomach and duodenum was cut through greater curvature and it was washed with physiological saline. Inner surface of stomach and duodenum was examined for ulcers. Stomach and duodenum of some of the animals in each group was photographed.

RESULTS

Table 1 shows Aspirin induced gastric ulcer in albino rats. Doses of Aspirin given was 100mg/kg/body weight orally. Table 2 shows the sizes of ulcer-index which is produced by Aspirin. The size of ulcer-index is measured in mm which is large in measurement in comparison to other table.

Table 1: Aspirin induced gastric ulcer.

| No | Ulcer index in mm |
|----|------------------|
| 1  | 42.0             |
| 2  | 14.0             |
| 3  | 15.0             |
| 4  | 43.0             |
| 5  | 40.0             |
| 6  | 32.0             |
| 7  | 44.0             |
| 8  | 25.0             |
| 9  | 34.0             |
| 10 | 14.0             |

n=10; mean=30.3; S.D=12.81; S.E = +3.99 or -3.99.

Remarks

All the animals developed ulcer intraperitoneal haemorrhages was seen in three of them.

Table 2 shows the ulcers in albino rats which were treated with both Aspirin and Famotidine. Aspirin was given in dose of 100 mg/kg/body weight and Famotidine was given in dose of 12 mg/kg/body weight.

Table 2: Ulcers in albino rats which were treated with both Aspirin and Famotidine.

| Serial number | Ulcer index in mm |
|---------------|------------------|
| 1             | 3.0              |
| 2             | 6.0              |
| 3             | 4.0              |
| 4             | 2.0              |
| 5             | 5.0              |
| 6             | 7.0              |
| 7             | 6.0              |
| 8             | 4.0              |
| 9             | 8.0              |
| 10            | 6.0              |

n=10; mean= 5.1; S.D= 1.87; S.E +0.59 or –0.59; P= more than 0.05-not significant.

Table 3 shows the measurement of ulcer-index in mm which is less as comparison to Table 1 because in Table 2 the measurement of ulcer-index is less due to effect of Famotidine which is H2 receptor blocker.

Table 3: Ulcers in albino rats which were treated with both Aspirin and Lansoprazole.

| Serial number | Ulcer index in mm |
|---------------|------------------|
| 1             | 4.0              |
| 2             | 4.0              |
| 3             | 3.0              |
| 4             | 5.0              |
| 5             | 4.0              |
| 6             | 2.0              |
| 7             | 2.0              |
| 8             | 4.0              |
| 9             | 4.0              |
| 10            | 3.0              |

n=10; mean=3.5; S.D=1.05; S.E = +0.33 or -0.33; P=more than 0.05-not significant.

Remarks

All animals developed ulcers.

Table 3 shows the size of ulcer-index which is much less as compared to other tables due to effect of Lansoprazole which is proton pump inhibitor. The above Table showing the ulcer index and standard error. The ulcer – index and standard error is much less as compared to others when Lansoprazole is used with Aspirin.
In some cases ulcers were multiple and so each area of damaged mucosa was measured (in mm2) and summed up and regarded as ulcer index. Aspirin inhibits prostaglandins synthesis by an action on enzyme prostaglandin synthetase. Prostaglandin exert a cytoprotective effect on gastric mucosa.3 Prostaglandins depress gastric secretion and produce gastric mucosal vasodilatation. Omeprazole is the first antisecretory drug to produce a sustained reduction of gastric acidity for 24 hours after a single dose.4 Genetic studies suggest that there is a strong familial tendency to duodenal ulceration.5 Cigarette smoking also plays an important role in causation of peptic ulceration.6 Amstrong C.P has reported that the use of non-steroidal anti-inflammatory drugs is frequently associated with damage to the upper gastrointestinal mucosa, perforations and bleeding especially in the elderly.7 Wastell used cimetidine clinically and found that it reduces meal stimulated acid secretion to about 70%.8 Rogenes found in their study that Ranitidine effectively protects the damage caused by ingestion of Aspirin.9 Lindberg studied that inhibitory action of omeprazole on acid secretion and found that it markedly inhibits both basal and stimulated gastric acid secretion.10 Lansoprazole is used in the treatment of gastric and duodenal ulcer along with NSAIDs induced ulcer. Lansoprazole is also used in Zollinger-Ellison syndrome.11 Famotidine is also used in prevention of NSAID-induced peptic ulcers.12

CONCLUSION

All the two drugs famotidine, lansoprazole prevent the ulcerogenic effect of Aspirin effectively, although not completely. This prophylactic ulcer preventing activity was the most with lansoprazole and the least with famotidine.

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Table 4: Effects of drugs on gastric mucosa in albino rats.

| Drugs used        | n= animals used | Dose= mg/kg | Ulcer index and standard error |
|-------------------|-----------------|-------------|--------------------------------|
| Aspirin           | 10              | 100         | 30.3 , + 3.99 or -3.99          |
| Aspirin+Famotidine| 10              | 100+12      | 5.1 , +0.59 or – 0.59           |
| Aspirin+Lansoprazole| 10             | 100+3       | 3.5, +0.33 or – 0.33            |

REFERENCES

1. Waren JR, Marshall BJ. Unidentified curved bacillus gastric epithelium in active chronic gastritis. Lancet. 1983;1:1273-5.
2. Hawkey CJ, Prichard PJ; Somerville KW; Strategies for preventing aspirin induced gastric bleeding. Scand J Gastroenterol. 1986;21:170-3.
3. Guth PH, Paulsen E. Topical aspirin plus HCl gastric lesions in the rat. Cytoprotective effect of prostaglandin, cimetidine, and probanthine. Gastroenterol. 1979;76:88.
4. Prichard PJ, Mitchellam GM, Walt RP. Human gastric mucosal bleeding induced by low dose aspirin but not warfarin. BMJ. 1985;298:493-6.
5. Rotter JI. 1979 Duodenal ulcer disease associated with elevated serum pepsinogen. Engl J Med. 1986;300:63.
6. Friedman GD. Cigarette, alcohol, coffee and peptic ulcer. N Engl J Med. 1974;290:469.
7. Armstrong CP, Blower AL. Non-steroidal anti-inflammatory drugs and life threatening complications of peptic ulceration. Gut. 1987;28:527-32.
8. Wastell C, Lance P. Cimetidine The Westminster Hospital Symposium. Edinburg, Churchill Livingstone. 1987.
9. Rogenes PR, Berkowitz JM, Sharp JT, Warner CW. Ranitidine protects against gastroduodenal mucosal damage associated with chronic aspirin therapy. Arch Intern Med. 1987;147:2137-9.
10. Lindberg P. A proton-pump inhibitor expedition: the case histories of omeprazole and esomeprazole. Trends in Pharmacol Sci. 1987;8:399-402.
11. Hirschowitz BI, Mohnen J, Shaw S. Long-term treatment with lansoprazole for patients with Zollinger-Ellison syndrome. Aliment Pharmacol Ther. 1996;10(4):507-22.
12. Corte R, Caselli M, Castellino G, Bajocchi G, Trotta F. Prophylaxis and treatment of NSAID-induced gastroduodenal disorders. Drug Safety. 1999;20(6):527-43.

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