Market Assessment of Anti Ulcerent Drug in Bareilly, Budaun, Moradabad and Rampur in Uttar Pradesh

By

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TO WHOMSOEVER MAY CONCERN

This is to certify that **Arun Rathore** student of MBA Pharmaceutical Management from The IIHMR University, Jaipur has undergone training at **IIHMR University** 7th February , 2016 to 6th May, 2016

The Candidate has successfully carried out the study designated to him during internship training and his approach to the study has been sincere, scientific and analytical. The Internship is in fulfillment of the course requirements

I wish him all success in all his future endeavors.

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Dean, Academics and Student Affair

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CERTIFICATE BY SCHOLAR

This is to certify that the dissertation titled "Market assessment of anti ulcerent drugs in Bareilly, Budaun, Moradabad and Rampur in Uttar Pradesh and submitted by **Arun Rathore** Enrollment No. **IIHMR-U/MBA-PM/2014-2016/6-209** under the supervision of **Dr. Nirmal kumar gubani** for award of MBA in Pharmaceutical management of the University carried out during the period from **2014 to 2016** embodies my original work and has not formed the basis for the award of any degree, diploma associate ship, fellowship, titles in this or any other Institute or other similar institution of higher learning.

Arun Rathore

Signature

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Last but not the least I thank "GOD" the Almightily, Parents, for their blessing and courage to ladder the success.

Thankful I ever remain……

ARUN RATHORE

CONTENTS

Introduction –

For more than a century, peptic ulcer disease has been a major cause of morbidity and mortality. In the early part of the 20th century, when stress and diet were judged to be important pathogenetic factors for peptic ulceration, patients with peptic ulcers were treated with hospitalization, bed rest, and the prescription of "bland" diets. By the 1950s, investigators and clinicians had focused their attention primarily on the pathogenetic role of gastric acid, and antacid therapy had become the treatment of choice for peptic ulcer disease. Antacids given at very high doses healed about 80% of duodenal ulcers after 4 weeks of therapy in comparison with placebo.[1]

The histamine H_2 receptor antagonist cimetidine became available for clinical use in 1977. H_2 receptor antagonists produced good ulcer healing rates, ranging from 80% to 95%, after 6 to 8 weeks of therapy. Acid suppression with antisecretory therapy rapidly emerged as the treatment of choice for patients with peptic ulcer disease. With the advent of proton pump inhibitors (PPIs) in the 1980s, even more potent acid suppression and higher rates of ulcer healing could be achieved. Although most acute peptic ulcerations healed with acid suppression therapy, the majority of patients experienced recurrences within 1 year of discontinuing treatment with antacids or antisecretory agents alone.[2]

For most of the 20th century, therefore, peptic ulcer disease was considered a chronic, incurable disorder characterized by frequent exacerbations and remissions. The discovery of the link between Helicobacter pylori and peptic ulcer by Marshall and Warren[3] in the mid-1980s led to another revolution in ulcer therapy. Now there is overwhelming evidence to support H. pylori infection as the most important cause of duodenal and gastric ulcers worldwide. Curing the infection not only heals peptic ulcer but also prevents ulcer relapse. [4] [5]

Although hospitalizations for uncomplicated peptic ulcers in western countries had begun to decline by the 1950s, there was an increase in admissions for ulcer hemorrhage and perforation among the elderly. [6] [7] [8] This increase has been attributed to the greater use of non-steroidal anti-inflammatory drugs (NSAIDs) and low-dose aspirin. The widespread use of NSAIDs has led to an epidemic of ulcer complications and deaths.[9] In the United States, use of prescription NSAIDs accounts for about 25% of all reported adverse drug reactions. Cotherapy with antiulcer drugs and the replacement of NSAIDs with cyclooxygenase-2 (COX-2) inhibitors have become acceptable treatments for patients who are at risk for peptic ulcer disease.

With the declining prevalence of H. pylori infection, the proportion of patients with idiopathic ulcers is growing, at least in the United States, where the reported proportion is between 20% and 30%. [10][11]

In Asia, the proportion of idiopathic ulcers is much lower, at 1% to 4%. [12] [13] It has been argued that as the incidence of H. pylori ulcers falls, a greater proportion of idiopathic ulcers will be seen.[14]

In a 5-year cohort of 435 patients with duodenal ulcer described in a study published in 1993, only 6 patients were identified to have H. pylori–negative idiopathic duodenal ulcers.[15] Their serumgastrin responses and peak gastric acid output values were significantly higher than those of H.pylori–negative controls without ulcers. Thus, long-term prophylaxis with antisecretory drugs isadvisable (see later), although this recommendation is not evidence-based.

Peptic ulcer disease represents a serious medical problem. According to latest WHO data published in may 2014 peptic ulcer disease deaths in India reached 85487 or 0.96 % of total deaths. the age adjusted death rate is 9.12 per 100,000 of population ranks India 26 in the world Interestingly, those at the highest risk of contracting peptic ulcer disease are those generations born around the middle of the 20th century Ulcers can develop in the esophagus, stomach or duodenum, at the margin of a gastroenterostomy, in the jejunum, in Zollinger Ellison syndrome, and in association with a Meckel's diverticulum containing ectopic gastric mucosa. Peptic ulcer disease is one of several disorders of the upper gastrointestinal tract that is caused, at least partially, by gastric acid. Patients with peptic ulcer disease may present with a range of symptoms, from mild abdominal discomfort to catastrophic perforation and bleeding.

Gastric and duodenal ulcers are breaks in the gastric and duodenal mucosa. Both gastric and duodenal ulcers relate to the corrosive action of pepsin and hydrochloric acid on the mucosa of the upper gastrointestinal tract. Ulcers generally range between 3 mm and several centimeters in diameter.

ANTISECRETORY AND ACID-NEUTRALIZING AGENTS-

Before the discovery of H. pylori as a causal factor in peptic ulcer disease, antiulcer drugs were the mainstays of treatment. Antiulcer therapy is not routinely required for patients with uncomplicated H. pylori ulcers in whom the bacterium is successfully eradicated, but antiulcer drugs do play an important role in promoting healing of large ulcers, preventing early recurrent bleeding after endoscopic therapy for bleeding ulcers, reducing the risk of ulcer relapse associated with NSAIDs, and treating idiopathic ulcers. Specific therapies for peptic ulcer are discussed in the following sections.

ANTACIDS-

Mechanisms of Action

Peterson and coworkers[1] showed in 1977 that a liquid antacid preparation of magnesium–aluminum hydroxide, administered in a dosage of 30 mL 1 and 3 hours after meals and at bedtime (approximately 1000 mmol neutralizing capacity per day) was more effective than placebo for hastening the healing of duodenal ulcer. Although it was thought at the time that antacids promote ulcer healing by neutralizing gastric acid, later studies showed that far smaller doses of antacids (as low as 120 mmol per day) had virtually identical efficacy for healing peptic ulcerations.[16]

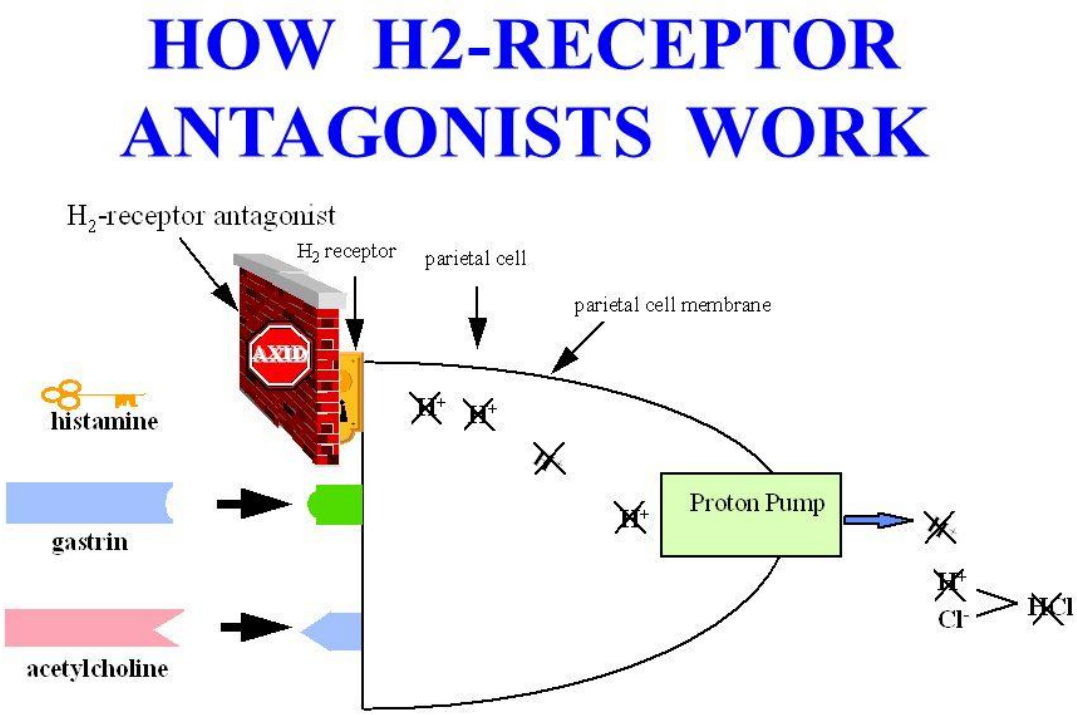
The precise mechanisms by which antacids hasten the healing of peptic ulcerations are not clear, but a variety of cytoprotective effects have been proposed for these agents, especially those that contain aluminum.[17]

HISTAMINE H2 RECEPTOR ANTAGONISTS-

Mechanisms of Action

Currently, four H_2 receptor antagonists are available—cimetidine (Tagamet), ranitidine (Zantac), famotidine (Pepcid), and nizatidine (Axid). All four agents are available without prescription (over the counter) in the United States.

They share an aromatic ring system and a flexible side chain. These compounds are competitive inhibitors of histamine-stimulated acid secretion, although famotidine appears to have some component of noncompetitive inhibition as well.[19] In addition to blocking histamine-stimulated gastric acid secretion, all four agents suppress basal acid output as well as acid output stimulated by meals (see Chapter 47).



Pharmacokinetics

The H_2 receptor antagonists are well absorbed after oral dosing, and absorption is not affected by food. Peak blood levels are achieved within 1 to 3 hours after an oral dose. These drugs are well distributed throughout the body, and all cross the blood-brain barrier and the placenta. [20] [21]

After oral administration, cimetidine, ranitidine, and famotidine undergo first-pass hepatic metabolism, which reduces their bioavailability by 35% to 60%. In contrast, nizatidine does not undergo first-pass metabolism, and its bioavailability approaches 100% with oral dosing. When administered in the evening, the drugs are especially effective in suppressing basal acid output at night.[22] .

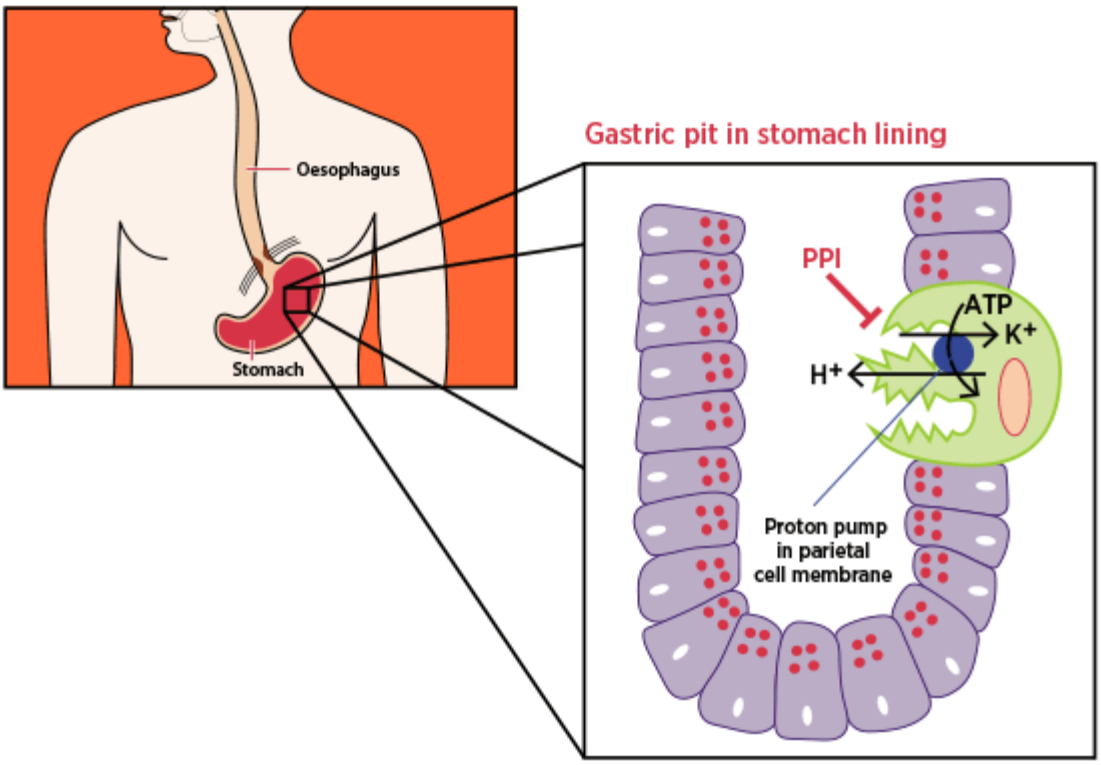
This effect appears to be particularly important, because the healing rate for peptic ulcers with antisecretory therapy correlates strongly with the level of reduction in nocturnal gastric acidity.[23]

All four H_2 receptor antagonists are eliminated by a combination of renal excretion and hepatic metabolism. The renal excretion is accomplished both by glomerular filtration and by tubular secretion of the agents. Sixty percent to 80% of orally administered cimetidine, ranitidine, or famotidine is cleared by the liver, whereas the elimination of oral nizatidine is accomplished primarily through renal excretion. After intravenous administration, in contrast, all four agents are eliminated principally through renal excretion. Plasma concentrations of H_2 receptor antagonists are affected by renal insufficiency. It is recommended that the doses be cut in half for patients whose creatinine clearance is 15 to 30 mL/min for cimetidine and famotidine or less than 50 mL/min for nizatidine and ranitidine.[24] Dialysis does not remove substantial amounts of the H_2 receptor antagonists, so dose adjustments for dialysis are not necessary. Liver failure has been found to prolong the half-life of cimetidine, but dose reductions are generally not needed for patients with hepatic failure unless it is accompanied by renal insufficiency.[19]

PROTON PUMP INHIBITORS

Mechanism and Site of Action

The PPIs are a class of drugs that decrease gastric acid secretion through inhibition of H+,K+-ATPase, the proton pump of the parietal cell (see Chapter 47). Currently, five PPIs are used widely as antisecretory agents—omeprazole (Prilosec), esomeprazole (Nexium; the S optical isomer of omeprazole), lansoprazole (Prevacid), pantoprazole (Protonix), and rabeprazole (Aciphex). These compounds, all substituted benzimidazoles, are weak bases, with a pKa of approximately 4.0 for omeprazole, lansoprazole, and pantoprazole, and approximately 5.0 for rabeprazole. These agents are prodrugs that must be activated by acid to effect inhibition of H+,K+-ATPase. However, the prodrugs are acid-labile compounds that must be protected from degradation by stomach acid during oral administration.[35]



Pharmacokinetics

The PPIs are well absorbed after oral dosing, and the simultaneous administration of antacids does not appear to affect their bioavailability. Food may delay the absorption of lansoprazole, pantoprazole, and rabeprazole,[35] but this delay does not alter the area under the plasma concentration–time curve, which is a key factor in achieving clinical efficacy for these agents.

Absorption of the enteric-coated agents may be erratic, and peak serum concentrations are not achieved until 2 to 5 hours after oral administration. Although the plasma half-life of the PPIs is short (<2 hours), the duration of acid inhibition is long (>24 hours) as a result of covalent binding to the H+,K+-ATPase.

Newer PPIs inhibit H+,K+-ATPase more rapidly than omeprazole, and emerging clinical data support potential clinical benefits resulting from this pharmacologic property. All PPIs undergo significant hepatic metabolism. Because there is no direct toxicity from PPIs, dose adjustments are not required even in patients with significant renal or hepatic impairment.[36] However, there are significant genetic polymorphisms for one of the CYP isoenzymes involved in PPI metabolism, CYP2C19. Approximately 3% of white persons and 15% of Asians are deficient in CYP2C19. This polymorphism has been shown to substantially raise plasma levels of omeprazole, lansoprazole, and pantoprazole but not those of rabeprazole. [37] [38]

As a result of their requirement for concentration and activation in acidic compartments, the PPIs bind predominantly to those proton pumps that are actively secreting acid. Thus, the efficacy of the PPIs for inhibiting acid secretion is limited if they are administered during the fasting state, when only approximately 5% of the stomach's proton pumps are

active. With meal stimulation, in contrast, 60% to 70% of the proton pumps actively secrete acid. Thus, the PPIs are most effective if they are
administered immediately before meals. For once-daily dosing, it is recommended that the PPIs be taken immediately before breakfast.[39] Eradication of H. pylori infection has been found to render PPIs somewhat less effective in elevating the gastric pH in patients with duodenal ulcer.[40] The mechanism by which H. pylori infection augments the pH-elevating effect of the PPIs is not clear. Conceivably, this phenomenon might be a consequence either of alkaline ammonia produced from urea by the organism or, more likely, of the greater gastric bicarbonate secretion and lesser gastric acid secretion associated with ongoing infection.[41]

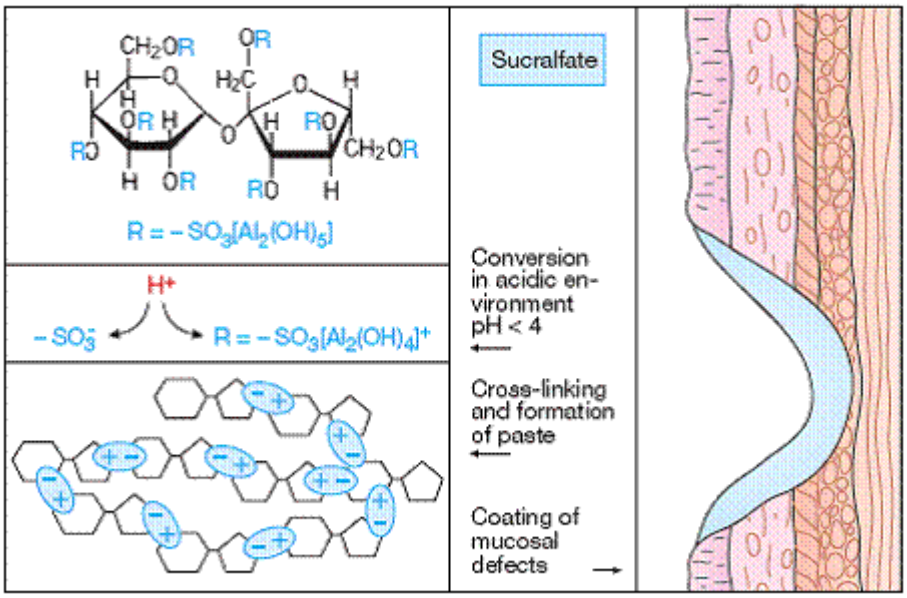
MUCOSA-PROTECTIVE AGENTS

Sucralfate

Mechanisms of Action

Sucralfate (Carafate) is a complex metal salt of sulfated sucrose. Although the sucralfate molecule contains aluminum hydroxide, the agent has little acid-neutralizing capacity. When exposed to gastric acid, the aluminum hydroxide dissociates, leaving sulfate anions that can bind electrostatically to positively charged proteins in damaged tissue. In this fashion, sucralfate adheres to ulcer craters, where it appears to form a protective barrier that may prevent further acid-peptic attack. Other proposed beneficial effects of sucralfate are enhancement of mucosal prostaglandin levels, stimulation of mucus and bicarbonate secretion, binding of bile salts, binding of epidermal growth factors, and promotion of angiogenesis.-

Sucralfate has demonstrated efficacy (similar to that of the H_2 receptor antagonists) in healing duodenal ulcer when given in a dose of 1 g four times daily.The drug has demonstrated efficacy in the treatment of gastric ulcer as well, but sucralfate has not been approved by the FDA for this indication. Compared with the H_2 receptor antagonists and the PPIs, however, there is much less published experience with sucralfate.[42]



A. Chemical structure and protective effect of sucralfate

Pharmacokinetics

Less than 5% of the sucralfate administered is absorbed owing to its poor solubility. The drug is excreted in feces. The high aluminum content causes a small but significant rise in serum and urine aluminum levels within 2 days. In patients with normal renal function, the minor amounts of aluminum absorption with short-term therapy are of no clinical significance.

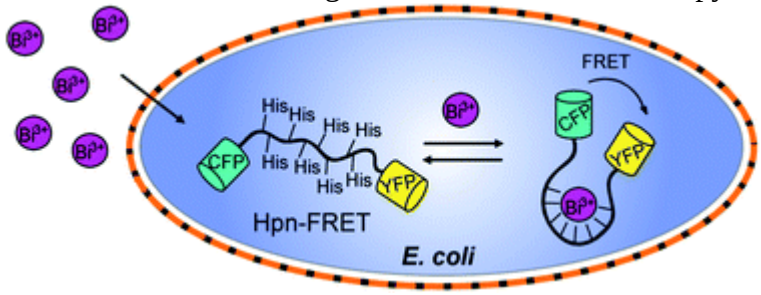
Toxicity and Drug Interactions

Because of the lack of systemic absorption, sucralfate appears to have no systemic toxicity. Gastric bezoar formation has been reported uncommonly. Concern has been raised about the potential for aluminum neurotoxicity in patients with chronic renal failure. However, the impact on the disposition of aluminum in the body has not been adequately studied in renal failure. Sucralfate is best avoided in this population. The drug can bind to a number of medications, including phenytoin and warfarin, reducing their absorption. Important drug interactions appear to be rare, however, and can be avoided entirely if sucralfate is administered separately from other medications.[43]

Bismuth

Mechanisms of Action

Bismuth preparations have been used widely to treat diarrhea, abdominal pain, and dyspepsia for hundreds of years. Two colloidal preparations of bismuth have been most commonly used, colloidal bismuth subcitrate and bismuth subsalicylate (e.g., Pepto-Bismol). These agents have some demonstrated efficacy in healing peptic ulcers, but the mechanisms underlying this therapeutic effect are not clear.[55] The bismuth forms complexes with mucus that appear to coat ulcer craters, perhaps affording protection from acid-peptic attack. Effects on increasing mucosal prostaglandin synthesis and bicarbonate secretion also have been proposed, and bismuth has documented antimicrobial actions against H. pylori. Bismuth has been approved by the FDA for use in combination with other agents for the treatment of H. pylori infection .



Pharmacokinetics

Bismuth is largely unabsorbed and is excreted in the feces. Colonic bacteria convert bismuth subcitrate and bismuth subsalicylate to bismuth sulfide, which turns the stools black. Trace amounts of bismuth are absorbed in the upper GI tract. Absorbed bismuth is slowly excreted in the urine for 3 months or longer.[44]

Toxicity

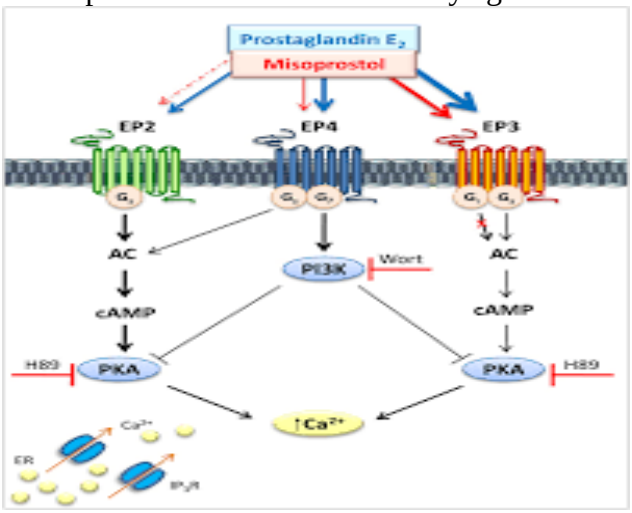
Short-term, standard-dose therapy with bismuth appears to carry little risk of toxicity. However, there is the potential for bismuth neurotoxicity if the agent is given for extended periods in high dosage, especially in patients with renal failure.

Prostaglandin E Analogs

Mechanisms of Action

Endogenous prostaglandins, including prostaglandin E_2 (PGE2), regulate mucosal blood flow, epithelial cell proliferation, epithelial restitution, mucosal immunocyte function, mucus and bicarbonate secretion, and basal acid secretion.[57] There is substantial evidence that the ulcerogenic effect of an NSAID correlates well with its ability to suppress prostaglandin synthesis. Misoprostol, a prostaglandin E1 analog, is the only prostaglandin analog approved by the FDA for the prevention of NSAID-induced ulcer disease.

The drug not only enhances mucosal defense mechanisms but also inhibits gastric acid secretion. After binding to the prostaglandin receptor on the parietal cell, misoprostol inhibits gastric acid secretion in a dose-dependent manner that is mediated through inhibition of histamine-stimulated cyclic adenosine monophosphate (cAMP) production.[60] It has been shown that misoprostol significantly reduces nocturnal, basal, and mealstimulated acid secretion at a standard therapeutic dose, although the effect is not as potent as that of antisecretory agents.



Pharmacokinetics

Misoprostol is well absorbed after oral administration. The plasma concentration peaks at about 30 minutes, with a serum half-life of approximately 1.5 hours. The drug has no effect on hepatic cytochrome P-450. Misoprostol metabolites are excreted in the urine, but dose reduction is unnecessary in patients with chronic renal failure.

Toxicity

Dose-related diarrhea is the most common side effect, occurring in up to 30% of patients and limiting the usefulness of misoprostol. Diarrhea is related to prostaglandin-induced increases in intestinal water and electrolyte secretion or acceleration of intestinal transit time. Administration of misoprostol with food may reduce diarrhea. Prostaglandins stimulate uterine smooth muscle. Uterine bleeding has been reported with prostaglandin analogs during the first trimester of pregnancy. Misoprostol is therefore contraindicated in women who may be pregnant.

Need for study –

Ulcer disease has become a disease predominantly affecting the older population, with the peak incidence occurring between 55 and 65 years of age. In men, duodenal ulcers were more common than gastric ulcers; in women, the converse was found to be true. Thirty-five percent of patients diagnosed with gastric ulcers will suffer serious complications. Although mortality rates from peptic ulcer disease are low, the high prevalence and the resulting pain, suffering, and expense are very costly. Many pharmaceutical companies have launched many drugs for ulcer. There are number of drugs are available in market with different mode of action like PPI and H_2 blockers, etc which help in curing this disease.

The purpose of this research is to analyze the current and future anti ulcer drugs condition to help marketing manager making strategies according feedback from participants.

Research objective

Ulcer disease has become a disease predominantly affecting the older population, with the peak incidence occurring between 55 and 65 years of age. In men, duodenal ulcers were more common than gastric ulcers; in women, the converse was found to be true. Thirty-five percent of patients diagnosed with gastric ulcers will suffer serious complications. Although mortality rates from peptic ulcer disease are low, the high prevalence and the resulting pain, suffering, and expense are very costly. Many pharmaceutical companies have launched many drugs for ulcer. There are numbers of drugs are available in market with different mode of action like PPI and H2 blockers, etc which help us to cure this disease. So with the help of this study we can find out the market potential of these anti ulcerent drugs in Bareilly, Moradabad Rampur and Budaun in Uttar Pradesh.

The purpose suggests the following research questions:

1- To find out the most selling anti ulcerent brands in selected cities of Uttar Pradesh.
2- To find out the most selling anti ulcerent dosage form in selected cities of Uttar Pradesh.
3- To find out the most selling anti ulcerent dosage strength in selected cities of Uttar Pradesh.
4- To find out the most selling combination of anti ulcerent drugs in selected cities of Uttar Pradesh.
5- To find out the most selling OTC drugs in selected cities of Uttar Pradesh.

Research methodology

Participants & apparatus
The survey was taken in selected cities in Uttar Pradesh by sending questionnaire as many as possible. Only those chemists who have completed the questionnaire will be accounted as valid survey. There are 200 chemists who have completed the questionnaire. Microsoft Office Excel to do statistic and data analysis

Research methods

The research method of this study is a survey research, which collect information from participant through a questionnaire. The purpose of this research is to analyze the current and future anti ulcer drugs market to help marketing manager making strategies according feedback from chemists. After questionnaires sent out, and later collected data from feedbacks, the quantitative research method will be used in this research.

Method selection

This section is dedicated to selection of research method in this paper. The research methods can be classified in many ways, but the most common difference is between qualitative and quantitative approaches. Qualitative research method can be broadly defined as "any kind of research that produces findings not arrived at by means of statistical procedures or other means of quantification" (Strauss and Corbin, 1990). It also can be simply says that this research method is non quantitative. In contrast, quantitative research method deal with measurable characteristics by using structured questions (e.g. questionnaire) and a formalized procedure of data collection.

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Questionnaire design

The design of questionnaire is very rely on what data need to be collected and how to analyze the data, and the questions asked in the questionnaire are need to be defined prior to data collection. According to the aims and objectives of this study, the questionnaire was prepared to collect data about the market assessment of anti ulcerent from this survey, following information will be gathered, best anti ulcerent brand best dosage from best selling price and all question. These questionnaires are completed by the respondents themselves, and the answering process is totally without any of intervention from the researcher. After the survey finished and all the data gathered, some of the questionnaires may not acceptable for research due to the lack of answers for the important questions. In the most cases it can enable researchers to reduce the amount of

data needed to collect in order to keep the result reliable. The complete questionnaire is given at the end as per annexure 1.
A Sample Design is a definite plan for obtaining a sample from a given population. It refers to the technique of the procedure adopted in selecting items for the sampling design is as below:

Sample size:

The substantial portions of the target customer that are sampled to achieve reliable result are 200.

it was determined by the formula

$$ss = \frac{Z^2 * (p) * (1-p)}{c^2}$$

Where:

Z = Z value (e.g. 1.96 for 95% confidence level)
p = percentage picking a choice, expressed as decimal
c = confidence interval, expressed as decimal

Z value was taken = 95 %

P value was taken= 450

C value was taken= 5

Universe:

Chemist of braj region of u.p- bareilly , budaun, Rampur ,Moradabad

Sampling method:

- Non probability convenience sampling.

Collection of data

Data collection

The study was conducted by the means of personal interview with respondents and the information given by them were directly recorded on questionnaire.
For the purpose of analyzing the data it is necessary to collect the vital information.

- Primary Data- Questionnaire method

Limitations of study

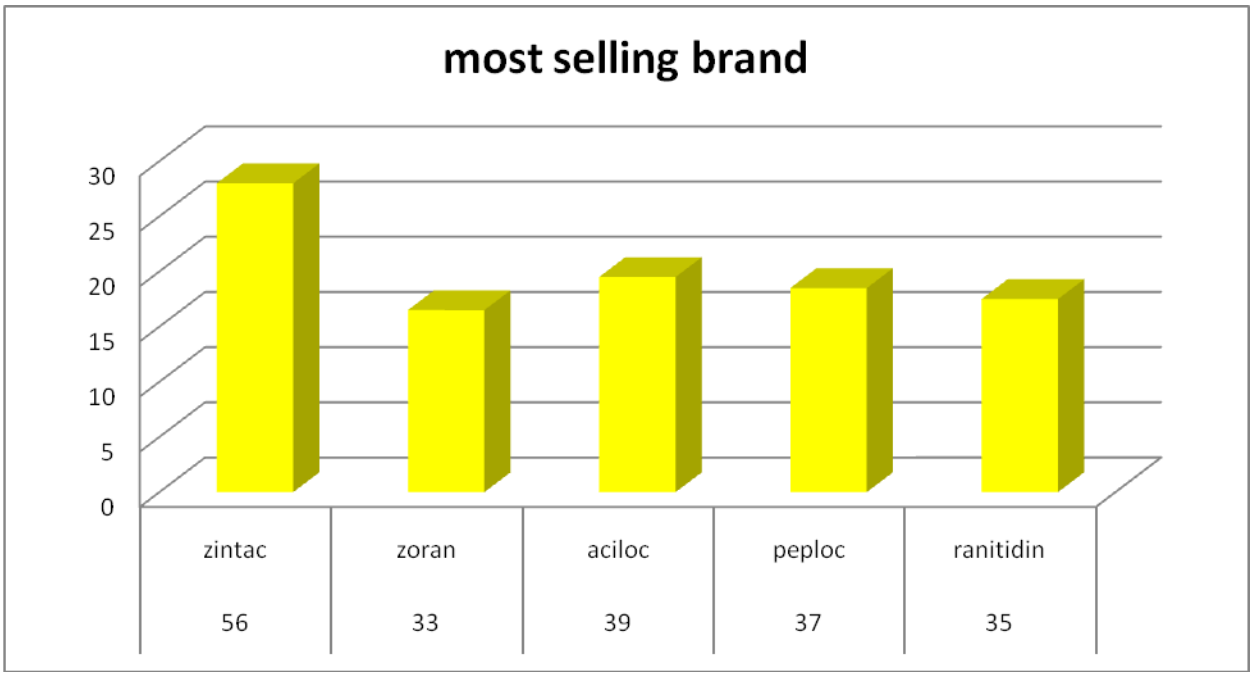
- People were not ready to fill in the questionnaire.
- Many of the surveyed people did not reply all the questions.
- The time period given for study was very limited.

DATA ANALYSIS AND INTERPRETATION

1- The most selling brand of anti ulcerent in selected cities in Uttar Pradesh.

No of chemist	Brand	Total %
56	Zintac (*Ranitidine*)	28
33	Zoran(Ranitidine)	16.5
39	Aciloc (Ranitidine)	19.5
37	Peploc (Ranitidine)	18.5
35	Ranitidine (Ranitidine)	17.5

No. of chemist	Price for ten tablets	Total %

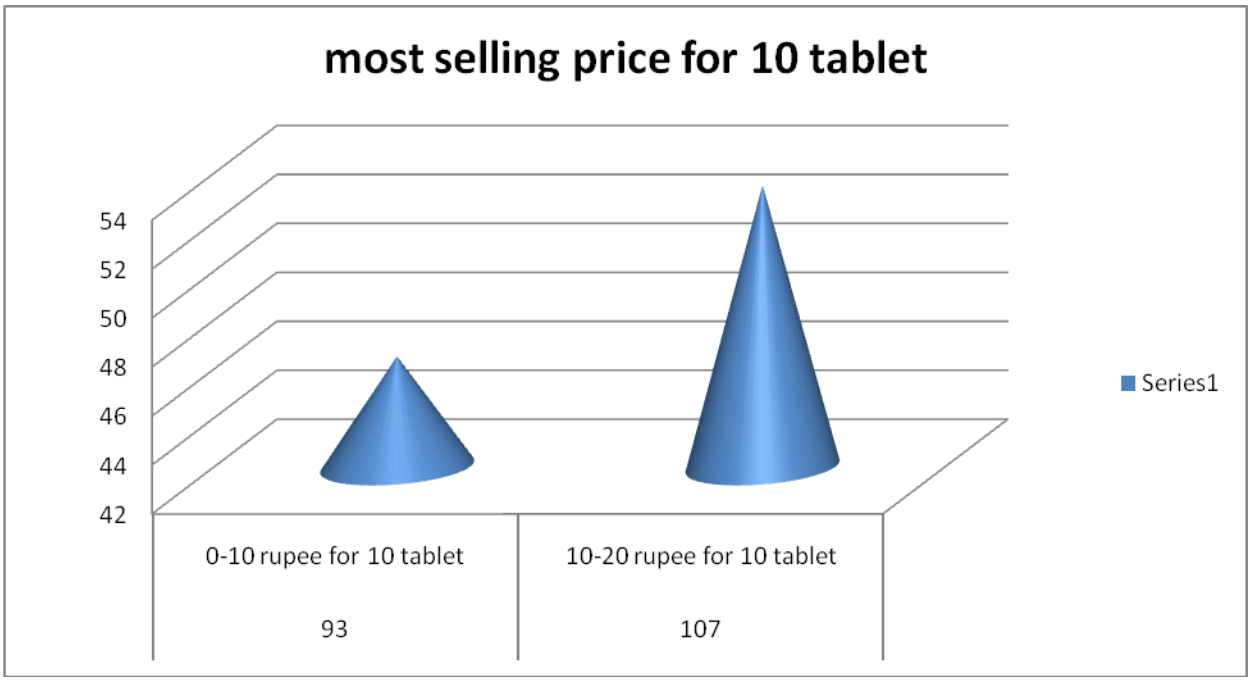


Interpretation –

According to the survey it can be interpreted
56 chemists gave their preference to zintac
39 chemists gave their preference to aciloc
37 chemists gave their preferne to peploc
35 chemists gave their preference to ranitidine
33hemist gave their preference to zoran

2- Most selling price for 10 tablets of strip in selected cities in Uttar Pradesh.

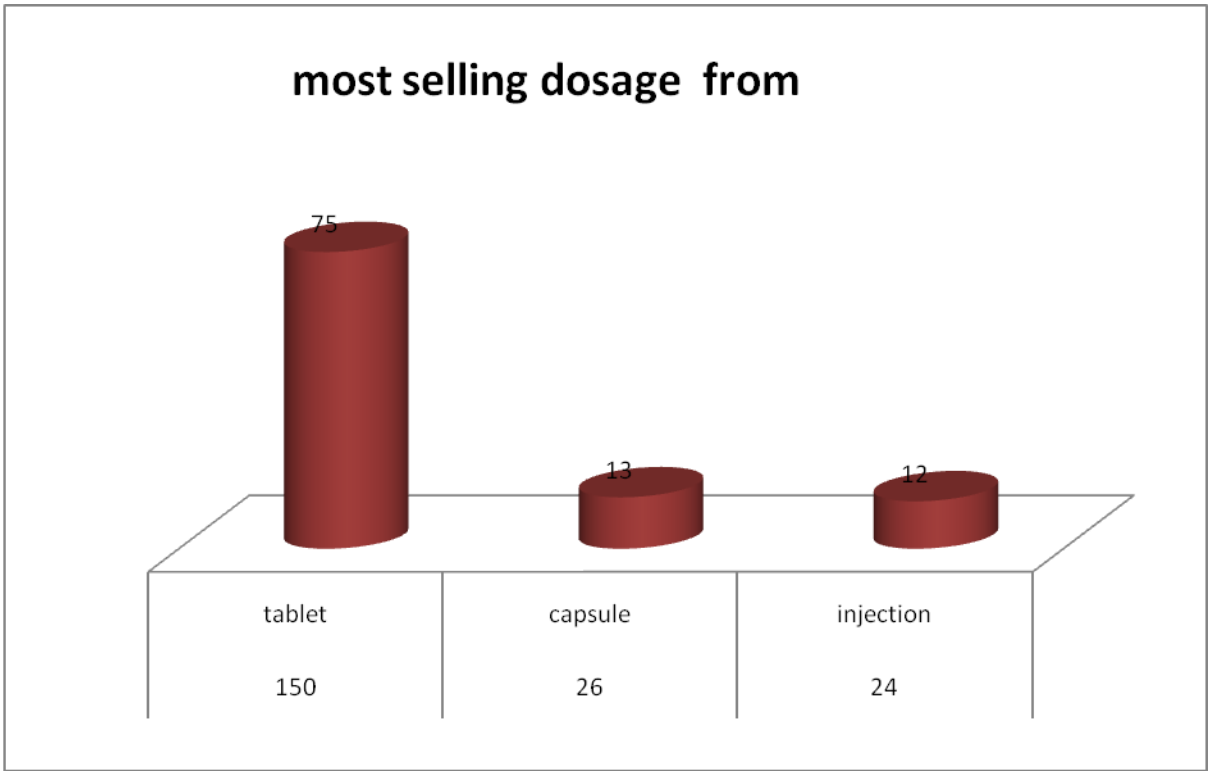
93	7.5-10 rs for ten tablets	46.5
107	10-20 rs for ten tablets	53.5



Interpretation – according to the survey it can be interpretated that the 107 chemist are selling anti ulcerent drugs between 10-20 Rs for ten tablets.

3- The most selling dosage form in selected cities in Uttar Pradesh.

No of chemist	Dosage form	Total %
150	Tablet	75
26	Capsules	13
24	Injection	12

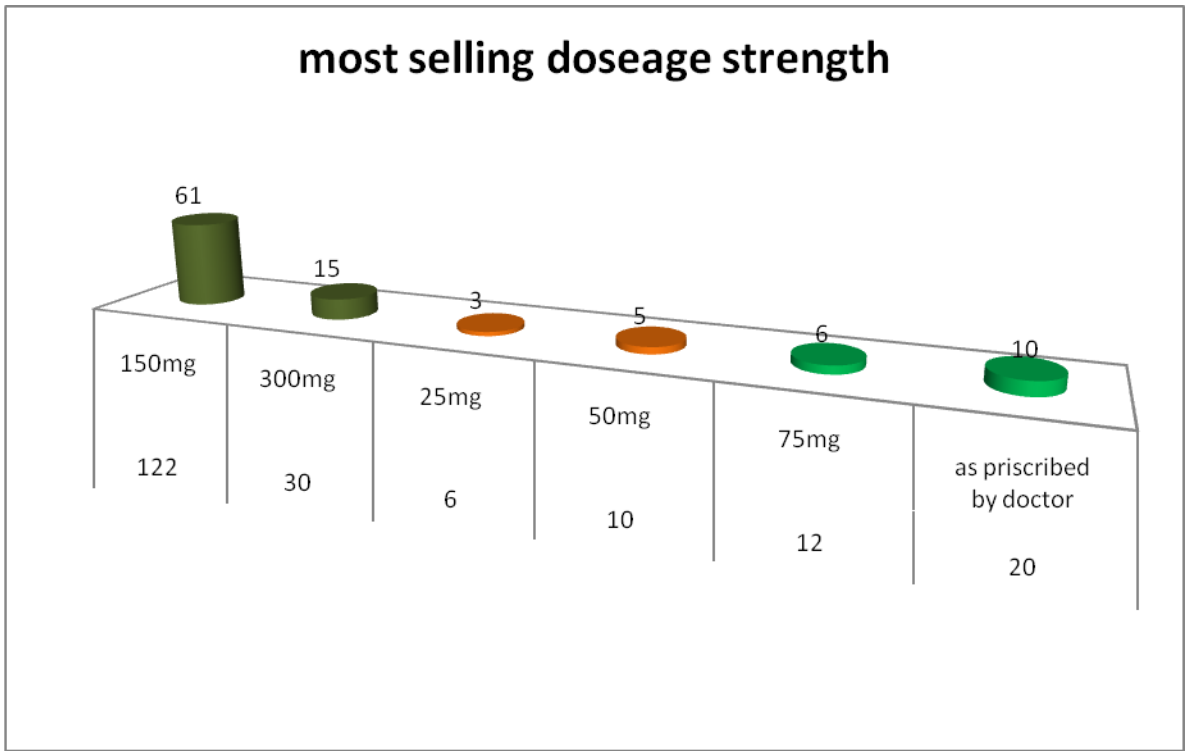


Interpretation –
According to 150 chemists, most sell is on tablet.
According to 26 chemists, most sell is on capsule
According to 24 chemists, most sell is on injection

4- The most selling dosage strength in selected cities in Uttar Pradesh.

No of chemist	Dosage strength	Total %
122	150mg	61
30	300mg	15
6	25mg	3
10	50mg	5
12	75mg	6
20	As prescribed by doctor	10

Interpretation –

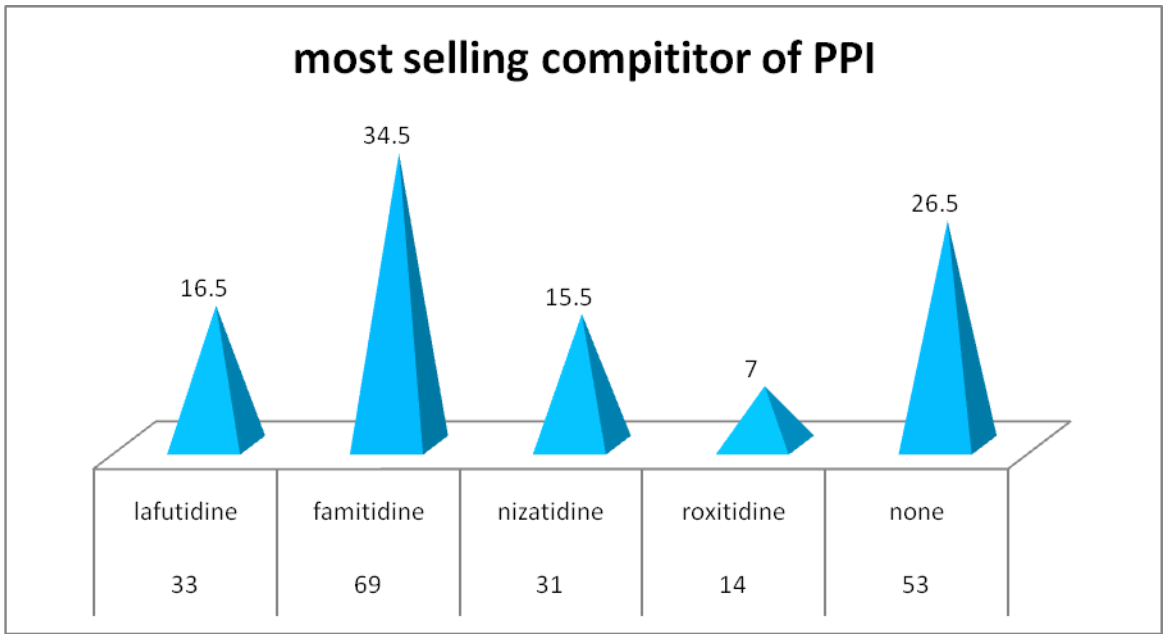


According to 122 chemists, most selling dosage strength, is 150 mg of tablet
According to 30 chemists, most selling dosage strength, is 300 mg of tablet
According to 6 chemists, most selling dosage strength, is 25 mg of capsule
According to 10 chemists, most selling dosage strength, is 50 mg of capsule
According to 12 chemists, most selling dosage strength, is 75 mg of capsule
According to 20 chemists, most selling dosage strength, is as prescribed by doctor

5- Most selling molecules against PPI in selected cities in Uttar Pradesh .

No of chemist	Molecules	Total %
33	Lafutidine	16.5
69	Famitidine	34.5

31	Nizatidine	15.5
14	Roxitidine	7
53	None	26.5

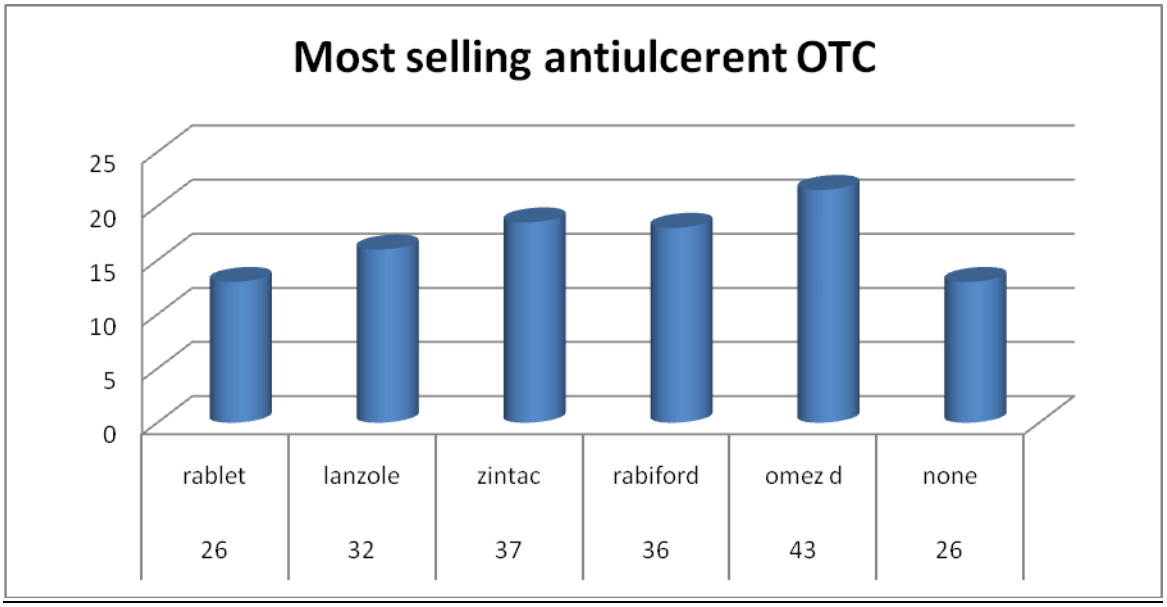


Interpretation –

According to 69 Chemist, most selling molecules against PPI is famitidine
According to 33 chemists, most selling molecules against PPI is lafutidine
According to 31 chemists, most selling molecules against PPI is nizatidine
According to 14 chemists, most selling molecules against PPI is roxitidine
According to 53 chemists, most selling molecules against PPI is none

6- Most selling OTC anti ulcerent in selected cities in Uttar Pradesh

No. of chemist	OTC molecules	Total %
26	Rablet (Rabeprazole sodium)	13
32	Lanzole (Lansoprazole)	7.5
37	Zintac (*Ranitidine)*	18.5
36	Rabiford (Rabeprazole)	9
43	Omez d (Omeprazole)	10.5
26	None	13

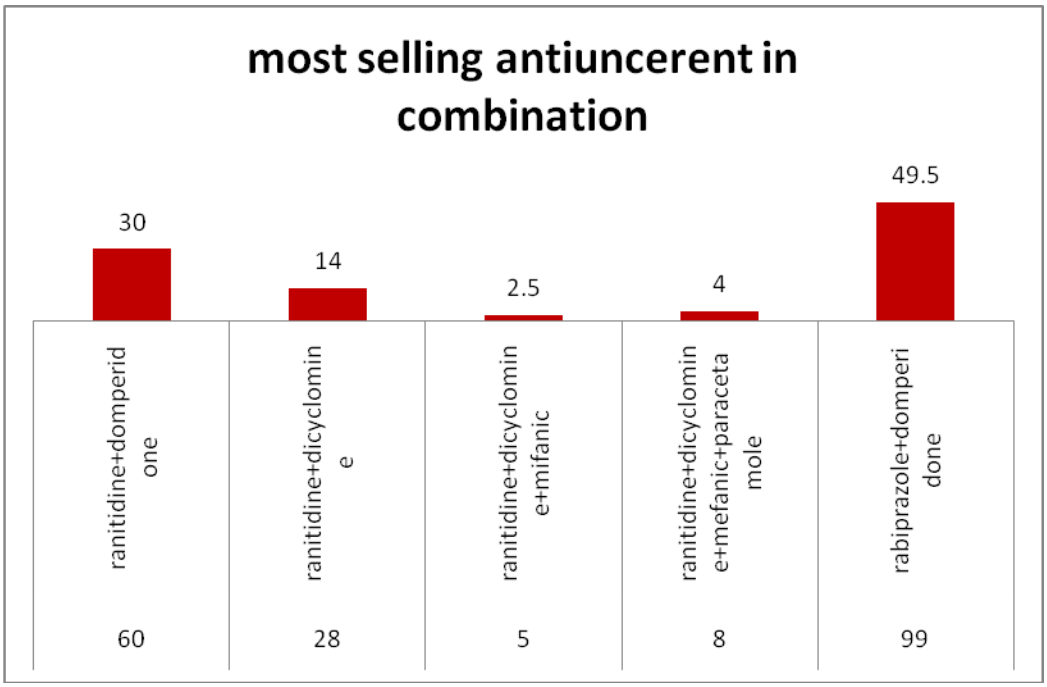


Interpretation-

According to 26 chemists, the most selling anti ulcerent OTC is Rablet
According to 32 chemists, the most selling anti ulcerent OTC is Lanozole
According to 37 chemists, the most selling anti ulcerent OTC is Zintac
According to 36 chemists, the most selling anti ulcerent OTC is Rabiford
According to 43 chemists, the most selling anti ulcerent OTC is Omez d
According to 26chemist, the most selling anti ulcerent OTC is None

7- Most selling antiulcerent in combination in selected cities in Uttar Pradesh

No. of chemist	Combination of antiulcerent	Total %
60	Ranitidine+domperidone	30
28	Ranitidine+dicyclomine	14
5	Ranitidine+ dicyclomine+mifanic	2.5
8	Ranitidine+ dicyclomine+mifanic+paracetamole	4
99	Rabiprazole+domperodone	49.5



Interpretation -

According to 26 chemists, most selling antiulcerent in combination is Ranitidine+domperidone

According to 28 chemists, most selling antiulcerent in combination is Ranitidine+dicyclomine

According to 5 chemists, most selling antiulcerent in combination is Ranitidine+ dicyclomine+mifanic

According to 8 chemists, most selling antiulcerent in combination is Ranitidine+dicyclomine+mifanic+paracetamole

According to 99 chemists, most selling antiulcerent in combination is Rabiprazole+domperodone

Conclusion –

1- The most selling brand of anti ulcerent in selected cities in Uttar Pradesh is zintac
2- The most selling price for 10 tablets of strip in selected cities in Uttar Pradesh is 10-20 Rs.
3- The most selling dosage form in selected cities in Uttar Pradesh is tablet.
4- The Most selling molecules against PPI in in selected cities in Uttar Pradesh is famitidine.
5- The most selling dosage strength in in selected cities in Uttar Pradesh is 150 mg of tablet and 50 mg for capsules.
6- The Most selling OTC anti ulcerent in selected cities in Uttar Pradesh is zintac.
7- The Most selling anti ulcerent in combination in selected cities in Uttar Pradesh is rabiprazole + domeperidone

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