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Perspective

# All Glittering are not Gold and All Peptic Ulcer Disease are not Same Treatment

**Muthukumar Raman[1]*, Manoj Ananthappan[2] and Preethi S[3]**

[1]*Professor, Sri Venkateshwaraa Medical College Hospital and Research Centre, India*

[2]*Senior Resident, Sri Venkateshwaraa Medical College Hospital and Research Centre, India*

[3]*Post Graduate, Sri Venkateshwaraa Medical College Hospital and Research Centre, India*

***Corresponding Author**: Muthukumar Raman, Professor, Sri Venkateshwaraa Medical College Hospital and Research Centre, India.

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## Introduction

In recent years peptic ulcer management has taken a back seat. Prevalence of peptic ulcer and its relapse is at a peak like never before. It makes us wonder the reason behind this current scenario inspite of such advanced technologies and potent drugs. Sitting back to analyse, it all come to the facts that there is lack of awareness in patients regarding self-medication and lack of understanding among medical fraternity in due process of peptic ulcer formation. Many are under a misconception that all peptic ulcer can be treated the same which is not the case and elaborating on it is the main goal of this article.

### Why so many relapses? where we go wrong

The though process of "No gastric acid, No peptic ulcer" is more prevalent among doctors these days. The failure to understand the mechanism leading to ulcer and prescribing the appropriate drug for that particular cause is absent now a days. All patients are being treated in the same way. Moreover ill informed advertisements of drug has led to increase in self-medication. Misuse of anti-ulcer medications as a prophylaxis is also on the rise. For achieving excellent result and no relapse, the treating physician should approach a patient by asking these questions - What caused it? where is the ulcer? Which drug to prescribe?, How to investigate further if persistent? and when to think about surgery? At the end analyzing these questions will give a clear picture of what we are dealing with.

Excessive gastric acid secretion is only one side of the coin. The other side of coin is poor mucosal defence which many of physicians miss. The upper gastrointestinal tract's integrity is in equilibrium, maintained by the "hostile" factors such as gastric acid, *H. pylori*, NSAIDs and pepsin, and "protective" factors such as prostaglandins, mucus, bicarbonate, and blood flow to mucosa affecting gastrointestinal mucosa. Imbalance is caused either by overwhelming hostile factors or deficient protective factors. Any imbalance causes ulcer. *H. pylori* is a major cause and its prevalence is inversely proportional to socio economic status. A small proportion of patients develop ulcer due to the adverse reaction of NSAIDs.

The most important step is acknowledging where the ulcer is. Many physicians fail to do so leading to gross mismanagement. Modified Johnson classification of peptic ulcer has 5 types based on anatomical location of ulcer. Type I ulcer at lesser curvature of stomach, Type II there are 2 ulcers present (gastric, and duodenal or pre pyloric), Type III ulcer at prepyloric region, Type IV Ulcer over proximal gastroesophageal region and Type V Ulcer can occur anywhere mainly due to chronic NSAID use. Why is this so important? All these types differ in the way they are formed. It is essential to understand the due process of their formation as they are not the same. In type I and type IV the gastric acid secretion is either low or normal and are caused due to poor mucosal protection and is not due to gastric acid.

Whereas in type II and type III the gastric acid secretion is very high. The chances of *H. pylori* infection is high in type II when compared to others.

There is a delusion behind which drug to prescribe among many. Systematic approach and clear understanding of each drug properties and its action can eliminate the doubt. It is a well known fact that anti-ulcer drugs are mainly of 3 types based on its action, the ones that decrease intragastric acidity, the ones that increases mucosal protection and finally the ones that eliminate *H. pylori* infection. $H_2$ antagonists, Proton pump inhibitors, and antacids are those agents that decrease intragastric acidity. Sucralfate increases mucosal protection. Some of the unique properties of a drug which is not seen in other drugs are as follows:

- $H_2$ antagonists are the best in nocturnal acid suppression. They are also good against meal stimulated acid production. Patients with night time dyspepsia and heart burn will benefit. Can be used in type II and III ulcers.
- PPIs are the most potent drug. They specifically target $H^+/K^+$ ATPase which is only present in parietal cells. Its duration of action is long so single dose will suffice. They have antibacterial activity against *H. pylori*. Widely used in type II, III and V ulcers and is a part of *H. pylori* eradication regimen.
- Antacids are the drug of choice for immediate pain relief. They interfere with $H_2$ antagonists and should not be given simultaneously. They are effective for 30 minutes when taken in empty stomach and for 2 hours when taken after a meal
- Sucralfate is ulcer protective. They act as a barrier between the ulcer and gastric content. They are best used in Type 1 and IV ulcer, stress ulcer and bile reflux gastritis. Biggest disadvantage is its action only lasts for 6 hours and requires to be taken 4 times a day. Used in critically ill patients to prevent GI bleeding.
- *H. pylori* eradication therapy - current gold standard of therapy is a triple combination of drugs that includes a PPI plus amoxicillin and clarithromycin. All three medicines are to be taken twice per day for 10 - 14 days. PPI should be continued for 4 - 6 weeks for complete cure. For patients who are resistant to these quadruple therapy in which bismuth sub-citrate is given additionally.

Any failure in cure or recurrence after eight weeks of optimal therapy warrants further investigation. Zollinger Ellison syndrome and MEN 1 syndrome needs to be ruled out. Secretin stimulation test and fasting gastrin levels to diagnose Zollinger Ellison syndrome and Serum calcium for MEN 1 syndrome. All these patients should undergo gastric acid analysis to determine the cause of ulcer is either due to gastric acid hypersecretion (basal acid output exceeding 10 mEq/hr) or decreased mucosal protection.

The common question that eludes frequently is when to think about surgery. With advancement in drug therapy, surgery is seldom used now a days and is reserved only for failed medical management and in complications. Surgical procedure varies based on the type of ulcer. Distal gastrectomy with gastrojejunostomy without vagotomy for type I, antrectomy with vagotomy for type II and III, Subtotal gastrectomy with roux en y gastrojejunostomy for type IV and highly selective anterior vagotomy with posterior truncal vagotomy for duodenal ulcer [1-4].

## Conclusion

Despite humungous research and advancements in peptic ulcer management therein lies a problem of mismanagement. At the end of the day the sufferers are patients. Relapses and recurrences can be easily avoided by understanding the basis leading to ulcer formation and the drugs mechanism of action. Emphasis should be given to the fact that peptic ulcer management is purely patient oriented and not the same.

## Bibliography

1. Shiotani A and Graham DY. "Pathogenesis and therapy of gastric and duodenal ulcer disease". *Medical Clinics of North America* 86.6 (2002): 1447-1466.

2. Malfertheiner P., *et al*. "Management of Helicobacter pylori infection-the Maastricht IV/Florence Consensus Report". *Gut* 61.5 (2012): 646-664.

3. Charpignon C., *et al*. "Peptic ulcer disease: one in five is related to neither Helicobacter pylori nor aspirin/NSAID intake". *Alimentary Pharmacology and Therapeutics* 38.8 (2013): 946-954.

4. El-Omar EM., *et al*. "Helicobacter pylori infection and chronic gastric acid hyposecretion". *Gastroenterology* 113.1 (1997): 15-24.

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