

Original Research Article

## Prevalence, Risk Factors of Chronic Peptic Ulcer Disease in Khammam, Telangana State

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**Abstract:** There has been a marked decrease in elective surgery for peptic ulcer disease (PUD) following introduction of medical therapies including proton pump inhibitors with or without antibiotics for *H pylori* eradication. By contrast, the numbers of acute complications like ulcer perforation and bleeding requiring emergency surgery have remained quantitatively constant. The present clinical study was carried out at Mamata general hospital surgical ward in Khammam; Telangana state from the period of April 2013 to March 2016 includes 100 patients. The male, female ratio was 56:44. The age wise incidence is more common in the 30-35 years(26%) followed by 50-60 years(20%). In this study 28% male were having the habit of alcohol drinking, smoking and irregular food habits. Most of females (32%) were taking tea/coffee about 4-5 times a day with irregular food habits. Helicobacter pylori infection and NSAIDs usage were more prevalent risk factors. Among these 81% of cases were treated successfully with the medical line of treatment and 18% people went for surgical treatment. Mortality rate was 1%.

**Keywords:** peptic ulcer, chronic, disease, prevalence, risk factors, treatment

### INTRODUCTION

Peptic ulcer is a disease of chronic development, characterized by an imbalance between the factors that damages the mucosa and those for its protection, resulting in a lesion of the lining of the upper digestive tract[1]. It has been one of the most prevalent diseases in the world, including India. Peptic Ulcer Disease (PUD) is the major gastrointestinal disorders causing ulcers in the gastric or duodenal mucosa that extend into the submucosa or deeper. Peptic Ulcer Disease be acute or chronic but are ultimately caused by an imbalance between mucosal defences and acid/peptic injury [2,3]. There are many causes has been found for ulcers. However, it is now clear that an ulcer is the end result of an imbalance between digestive fluids in the stomach and duodenum. Most ulcers are caused by an infection with a type of bacteria called Helicobacter pylori (*H. pylori*).

Risk factors for ulcers include use of painkillers called non steroidal anti-inflammatory drugs (NSAIDs), such as aspirin, naproxen, ibuprofen and many others, excess acid production from gastrinomas, tumours of the acid producing cells of the stomach that increases acid output, excessive drinking of alcohol,

smoking or chewing tobacco, serious illness and radiation treatment. Patients with ulcers may show gnawing or burning pain in the middle or upper stomach between meals or at night bloating, heartburn, nausea and vomiting. In severe cases dark stools due to bleeding, vomiting blood, weight loss and severe pain in the mid to upper abdomen are observed. If ulcers are not treated properly can lead to serious health problems like bleeding, Perforation, gastric outlet obstruction from swelling or scarring that blocks the passage way leading from the stomach to the small intestine.

Every year peptic ulcer disease (PUD) affects 4 million people around the world [4]. Complications are encountered in 10%-20% of these patients and 2%-14% of the ulcers will perforate [5,6]. Perforated peptic ulcer (PPU) is relatively rare, but life-threatening with the mortality varying from 10% to 40% [7-8]. More than half of the cases are female and they are usually older and have more co morbidities than their male counterparts [9]. Main etiologic factors include use of non-steroidal anti-inflammatory drugs (NSAIDs), steroids, smoking, Helicobacter pylori and a diet high in salt [10]. All these factors have in common that they affect acid secretion in the gastric mucosa. Defining the

exact etiological factor in any given patient may often be difficult, as more than one risk factor may be present and they tend to interact [11].

Currently, medical management, usually with a group of drugs called proton pump inhibitors, is the mainstay treatment for uncomplicated peptic ulcers. Ulcer medications can include, Proton pump inhibitors (PPI) Proton reduce acid levels and allow the ulcer to heal.

They include dexlansoprazole, esomeprazole, lansoprazole, omeprazole, pantoprazole, Rabeprazole, and omeprazole/sodium bicarbonate. If patients have H. pylori infection, then antibiotics are also used. Some bleeding ulcers can be treated through an endoscope. Sometimes an operation is needed if the ulcer has created a hole in the wall of the stomach, or if there is serious bleeding that can't be controlled with an endoscope. The alternative to medical treatment for refractory and recurrent peptic ulcer is surgical treatment to decrease the acid secretion in the stomach with the goal of curing the peptic ulcer. It is not known whether medical or surgical management is a better option for people with a refractory or recurrent peptic ulcer.

The main aim and objective of this study is to know the prevalence, risk factors associated with chronic peptic ulcers and to compare conservative medical lines of treatment versus surgical approach in treatment of chronic peptic ulcers.

**MATERIALS AND METHODS**

The present clinical study was carried out at Mamata general hospital surgical ward in Khammam, Telangana state from the period of April 2013 to March 2016 after obtaining permission from hospital ethics committee. A total of 100 patients were included in the study. Inclusion criteria is both male and female patients, aged between 30-50 years, who were consent to participate in study were included. Patients with deformed duodenal cap, pyloric stenosis were excluded from the study. Study included both conservative, elective surgery and emergency laparotomy.

The diagnosis of gastrointestinal ulcers was based on clinical features, blood tests, routine laboratory tests, and radiological findings (i.e. plain abdominal X-ray in all cases and abdominal CT scan in 87 % of patients). Invariably, the definitive diagnosis of PPU was obtained at surgery. Diagnosis of this infection may be achieved through various tests, each with a sensitivity and specificity above 80%. The golden standard test is the upper endoscopy, which allows the physician to collect material to check for the presence of H. pylori besides other therapeutic procedures [12]. Free air under the diaphragm found on an upright chest X-ray is indicative of hollow organ perforation and mandates further work-up and/or exploration. In the setting of an appropriate history and

peritonitis on examination, free air on X-ray is sufficient to justify exploration. However, up to 12% of patients with traumatic perforations may have a normal CT scan. Adding oral contrast and performing triple contrast CT scan may improve diagnostic sensitivity and specificity [13].

After diagnosis patients were subjected to medical line of treatment with a combination of drugs like Omeprazole 40mg, Amoxicillin 1550mg / Clarithromycin 500mg and Tinidazole 1000 mg with antacid gel and bland diet for 7days course in a month repeated for 3 months. The cases were followed up for about 6 months

**RESULTS**

A total of 100 patients were included. The demographic data was given in table1. The male, female ratio was 56:44. The age wise incidence is more common in the 30-35 years (26%) followed by 50-60 years (20%).

**Table.1: Age, Sex wise incidence of peptic ulcers**

Age (year)	Male (No)	Female (No)
30-35	14	12
36-40	10	6
40-45	8	8
46-50	12	8
50-60	10	10

The risk factors for chronic peptic ulcers were given in table 2. Most of males suffering with ulcers were having more than one risk factor. In this study 28% male were having the habit of alcohol drinking, smoking and irregular food habits. Most of females (32%) were taking tea/coffee about 4-5 times a day with irregular food habits. Helicobacter pylori infection and NSAIDs usage were more prevalent risk factors. About 10% of people were suffering with other gastric problems and some were with unknown etiology.

**Table 2: The risk factors for peptic ulcers**

Risk factor	Percentage
Helicobacter pylori infection	42
NSAIDs usage	44
Alcohol drinking	36
Smoking/tobacco chewing	32
Tumors of GIT	2
Drinking of tea/coffee	62
Irregular food habits	56
Others	10
Unkown factors	10

Among these 81% of cases were treated successfully with the medical line of treatment and 18% people went for surgical treatment. Surgical treatment

includes five for cataclysmic haemorrhage, four for persistent haemorrhage, 6 for recurrent bleeding. An analysis of factors leading to the necessity of surgical haemostasis was undertaken by considering the clinical status, endoscopic findings and laboratory results. The size of the ulcer (greater than 2 cm) was the most significant parameter and other criteria shock, endoscopic signs of recent haemorrhage, gastric or duodenal posterior ulcer were considered. Two cases had anterior duodenal ulcer perforation for which emergency laparotomy and closure of perforation was done and one underwent posterior gastrojejunostomy with bilateral truncal vagotomy for peptic ulcer complications like deformed duodenal cap and early signs of pyloric stenosis. Among all one patient was died due to delay in arrival to hospital, delay in diagnosis and management

## DISCUSSION

Peptic ulcer is characterized by a solution of continuity the upper digestive tract mucosa exposed to chloride peptic secretion. It often occurs in the duodenum (5-10% of the population), stomach or esophagus [12]. It is a chronic disease, with activation and remission periods and its pathogenesis is characterized by the imbalance between the factors that damages the mucosa (chloride acid, pepsin, and ulcerogenic drugs) and those that protect it (mucosal barrier, prostaglandins, and mucosal secretion)[14]. Clinical manifestations are characterized by epigastric discomfort, burning or severe and continuous pain, which tends to be worse at night. Pain usually happens one to three hours after eating, and may be followed by nausea, vomiting, and discomfort in the gastrointestinal tract, flatulence, and significant loss of body weight [15].

Approximately 1 in 100 to 1 in 800 people have peptic ulcers. The major causes of peptic ulcer are *Helicobacter pylori* infection, non-steroidal anti-inflammatory drug use, and smoking. People who have peptic ulcer have upper abdominal pain, which is sometimes accompanied by dyspepsia. The most serious complications of peptic ulcers are bleeding from the ulcer and perforation of the peptic ulcer, which results in stomach or upper small intestinal contents or both leaking into the tummy. About 1 in 10 people with bleeding peptic ulcer and 1 in 4 people with perforated peptic ulcer die. Peptic ulcers cause approximately 3000 to 4500 deaths per year in the US [16].

It has been one of the most prevalent diseases in the world, and some of its complications have been the major causes of morbidity and mortality [17]. The prevalence differs in the world population between the duodenal and gastric ulcers, and the mean age of people with the disease is between 30 and 60 years, but it can happen in any age. Racial difference has also been

observed, and in Africa duodenal ulcers are found to be rare in black people, but in the United States the incidence is the same for blacks and whites; regarding gender, there is predominance of ulcers in males [18]. Peptic ulcer has a multifactor etiology. Environmental elements such as alcohol and nicotine can inhibit or reduce secretion of mucus and bicarbonate, increasing acid secretion. Genetic factors can influence, and children of parents with duodenal ulcer are three times more likely to have ulcer than the population [19]. In the past decades, the identification of *Helicobacter pylori* and ulcers associated with the chronic use of anti-inflammatory drugs contributed to a better understanding of the events associated to the genesis of peptic ulcers [12].

These results suggested that more than 50% of patients with perforated peptic ulcer respond to conservative treatment without surgery and that the association of few required emergency surgery. Mortality in our study was 1%. When PPU are diagnosed expeditiously and promptly treated, outcomes are excellent. Mortality ranges from 6% to 14% in recent studies [20-21]. Poor outcomes have been associated with increasing age, major medical illness, peri-operative hypotension [22], and delay in diagnosis and management (greater than 24 hours) [23]. With improvements in resuscitation, hypotension may no longer be a significant prognostic indicator [24]. Advanced age (greater than 70 years) is associated with a higher mortality with rates of approximately 41% [25].

## CONCLUSION

The present study concludes that PUD cases can be better managed with medical line of treatment of H2 blockers and PPI's in 80% of patients, despite the introduction of effective medical line of treatment, bleeding is still a frequent complication where surgical treatment should be recommended

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## REFERENCES

1. Nieto YB. Protocolo terapeutico de la úlcera péptica. Medicine-Programa de Formación Médica Continuada Acreditado. 2012 Feb 29; 11(3):179-82.
2. Holle GE. Pathophysiology and modern treatment of ulcer disease (Review). International journal of molecular medicine. 2010 Apr 1; 25(4):483.

3. Leong RW. Differences in peptic ulcer between the East and the West. *Gastroenterology Clinics of North America*. 2009 Jun 30; 38(2):363-79.
4. Zelickson MS, Bronder CM, Johnson BL, Camunas JA, Smith DE, Rawlinson D, Von S, Stone HH, Taylor SM. *Helicobacter pylori* is not the predominant etiology for peptic ulcers requiring operation. *The American Surgeon*. 2011 Aug 1; 77(8):1054-60.
5. Bertleff MJ, Lange JF. Perforated peptic ulcer disease: a review of history and treatment. *Digestive surgery*. 2010 Jun 22; 27(3):161-9.
6. Lau JY, Sung J, Hill C, Henderson C, Howden CW, Metz DC. Systematic review of the epidemiology of complicated peptic ulcer disease: incidence, recurrence, risk factors and mortality. *Digestion*. 2011 Apr 14; 84(2):102-13.
7. Svanes C. Trends in perforated peptic ulcer: incidence, etiology, treatment, and prognosis. *World journal of surgery*. 2000 Mar 21; 24(3):277-83.
8. Møller MH, Adamsen S, Wøjdemann M, Møller AM. Perforated peptic ulcer: how to improve outcome?. *Scandinavian journal of gastroenterology*. 2009 Jan 1; 44(1):15-22.
9. Thorsen K, Glomsaker TB, von Meer A, Søreide K, Søreide JA. Trends in diagnosis and surgical management of patients with perforated peptic ulcer. *Journal of Gastrointestinal Surgery*. 2011 Aug 1; 15(8):1329-35.
10. Gisbert JP, Legido J, Garcia-Sanz I, Pajares JM. *Helicobacter pylori* and perforated peptic ulcer. Prevalence of the infection and role of non-steroidal anti-inflammatory drugs. *Digestive and liver disease*. 2004 Feb 29; 36(2):116-20.
11. Kurata JH, Nogawa AN. Meta-analysis of risk factors for peptic ulcer: nonsteroidal antiinflammatory drugs, *Helicobacter pylori*, and smoking. *Journal of clinical gastroenterology*. 1997 Jan 1; 24(1):2-17.
12. Toneto M, Oliveira F, Lopes MH. Evolução histórica da úlcera péptica: da etiologia ao tratamento. *Scientia Medica*. 2011 Mar 1; 21(1):23-30.
13. Malhotra AK, Fabian TC, Katsis SB, Gavant ML, Croce MA. Blunt bowel and mesenteric injuries: the role of screening computed tomography. *Journal of Trauma and Acute Care Surgery*. 2000 Jun 1; 48(6):991-1000.
14. Malhotra AK, Fabian TC, Katsis SB, Gavant ML, Croce MA. Blunt bowel and mesenteric injuries: the role of screening computed tomography. *Journal of Trauma and Acute Care Surgery*. 2000 Jun 1; 48(6):991-1000.
15. Nieto YB. Protocolo terapéutico de la úlcera péptica. *Medicine-Programa de Formación Médica Continuada Acreditado*. 2012 Feb 29; 11(3):179-82.
16. Gurusamy KS, Pallari E. Medical versus surgical treatment for refractory or recurrent peptic ulcer. *The Cochrane Library*. 2016 Jan 1.
17. Sung JJ, Tsoi KK, Ma TK, Yung MY, Lau JY, Chiu PW. Causes of mortality in patients with peptic ulcer bleeding: a prospective cohort study of 10,428 cases. *The American journal of gastroenterology*. 2010 Jan 1; 105(1):84-9.
18. Martins LC, de Oliveira Corvelo TC, Oti HT, dos Santos Barile KA. Soroprevalência de anticorpos contra o antígeno CagA do *Helicobacter pylori* em pacientes com úlcera gástrica na região Norte do Brasil. *Revista da Sociedade Brasileira de Medicina Tropical*. 2002 Jul; 35(4):307-10.
19. Lafortuna CL, Agosti F, Marinone PG, Marazzi N, Sartorio A. The relationship between body composition and muscle power output in men and women with obesity. *J Endocrinol Invest*. 2004 Oct 1; 27(9):854-61.
20. Arici C, Mesci A, Dincer D, Dinckan A, Colak T. Analysis of risk factors predicting (affecting) mortality and morbidity of peptic ulcer perforations. *International surgery*. 2006 Dec; 92(3):147-54.
21. Kocer B, Surmeli S, Solak C, Unal B, Bozkurt B, Yildirim O, Dolapci M, Cengiz O. Factors affecting mortality and morbidity in patients with peptic ulcer perforation. *Journal of gastroenterology and hepatology*. 2007 Apr 1; 22(4):565-70.
22. Bucher PA, Oulhaci W, Morel P, Ris F, Huber O. Results of conservative treatment for perforated gastroduodenal ulcers in patients not eligible for surgical repair. *Swiss medical weekly*. 2007; 137(23-24):337-40.
23. Boey J, Choi SK, Poon A, Alagaratnam TT. Risk stratification in perforated duodenal ulcers. A prospective validation of predictive factors. *Annals of surgery*. 1987 Jan; 205(1):22.
24. Siu WT, Leong HT, Law BK, Chau CH, Li AC, Fung KH, Tai YP, Li MK. Laparoscopic repair for perforated peptic ulcer: a randomized controlled trial. *Annals of surgery*. 2002 Mar 1; 235(3):313-9.
25. Uccheddu A, Floris G, Altana ML, Pisanu A, Cois A, Farci SL. Surgery for perforated peptic ulcer in the elderly. Evaluation of factors influencing prognosis. *Hepatogastroenterology*. 2003 Nov 1; 50(54):1956-8.