Print ISSN: 2321-6379
Online ISSN: 2321-595X
DOI: 10.17354/ijss/2017/512

Original Article

Peptic Ulcer Disease in the Proton Pump Inhibitor Era in Coastal Odisha

Haribhakti Seba Das[1], Chitta Ranjan Panda[2], Sambit Kumar Behera[3]

[1]Assistant Professor, Department of Gastroenterology, Shrirama Chandra Bhanj Medical College, Cuttack, Odisha, India, [2]Associate Professor, Department of Gastroenterology, Shrirama Chandra Bhanj Medical College, Cuttack, Odisha, India, [3]DM Resident, Department of Gastroenterology, Shrirama Chandra Bhanj Medical College, Cuttack, Odisha, India

Abstract

Background and Objective: There is a wide variation in the prevalence of peptic ulcer in India both before and since the use of endoscopy. We studied the risk factors, mode of presentation and treatment outcome in patients with peptic ulcer attending two gastrointestinal (GI) clinics in coastal Odisha, and its relationship with *Helicobacter pylori* infection.

Methods: We investigated patients who underwent a health inspection for upper GI symptoms. Upper GI endoscopy was performed, and biopsy specimens were collected from the stomach of the patients who were found to have peptic ulcer disease (PUD). All patients with peptic ulcer were prospectively followed after *H. pylori* eradication regimen. After a minimum of 4 weeks repeat, upper GI endoscopy was performed to assess healing of ulcers.

Results: Between 2015 and 2017, 3000 patients with peptic ulcer were seen, of whom 1480 (49.33%) had duodenal ulcer, 917 (30.56%) had gastric ulcer, and 603 (20.1%) had both duodenal and gastric ulcer. The mode of presentation was epigastric pain (36%), dyspepsia (26%), GI bleed (24%), and gastric outlet obstructive symptoms (14%). Risk factors were smoking (38%), nonsteroidal anti-inflammatory drugs (NSAID) intake (22%), alcohol intake (13%), and indigenous drug (8%). Among 926 patients, rapid urease test (RUT) could be done. 682 (73.65%) were positive, and 244 (26.35%) were negative for RUT. Among the *H. pylori* positive subjects duodenal ulcer was most common (49.85%) followed by gastric ulcer (30.8%) and both gastric and duodenal ulcer (19.35%). Treatment with *H. pylori* eradication regimen resulted in complete healing 75%, partial healing in 15% but non-healing still persisted in 10% patients.

Conclusions: PUD is very common in coastal eastern Odisha. Among them, duodenal ulcer is the most common variety. Epigastric pain is the most common type of presentation. Smoking is the most common risk factor followed by NSAID intake. *H. pylori* association causes mostly duodenal ulcers. Complete healing of ulcers occurs in two-third cases after *H. pylori* eradication regimen. Further studies required to assess the etiology in remaining partial and non-healing ulcer cases.

Key words: Epigastric pain, *Helicobacter pylori,* Nonsteroidal anti-inflammatory drugs intake, Peptic ulcer

INTRODUCTION

The term peptic ulcer disease (PUD) is used broadly to include ulcerations in the stomach and duodenum from a number of causes. Regardless of the inciting agent, the role of acid and pepsin in the genesis and spread of mucosal injury remains a combining aspect of the pathogenesis of PUD.[1] First isolation of *Helicobacter pylori* in 1982 by Marshall and Warren has revolutionized the pathophysiology and concept of treatment of PUD and also transformed it from a chronic recurrent disease to a curable one.[2-4] PUD results in various complications such as bleeding, perforation, and gastric outlet obstruction.[5,6] *H. pylori* infection and the use of nonsteroidal anti-inflammatory drugs (NSAIDs) are the most well-known causal factors for PUD.[7-11] Although the prevalence of PUD caused by *H. pylori* has been decreasing due to eradication therapy, the prevalence of PUD induced by NSAIDs or aspirin is increasing because of the worldwide increase in the aging population.[12-14] India is a vast country known for its rich history, culture, and food. It is also the typical developing country with a vast rural population living in poverty. The prevalence of *H. pylori* in the Indian subcontinent can be as high as 80% or more in rural areas. Therefore, new

Access this article online

www.ijss-sn.com

Month of Submission : 09-2017
Month of Peer Review : 10-2017
Month of Acceptance : 10-2017
Month of Publishing : 11-2017

Corresponding Author: Dr. Chitta Ranjan Panda, Department of Gastroenterology, S. C. B. Medical College and Hospital, Cuttack - 753 007, Odisha, India. Phone: +91-9437013800. E-mail: panda.cr@hotmail.com

strategies for the prevention and cure of PUD in India are important.[15]

It is difficult to detect PUD in asymptomatic individuals. In some cases, it is detected due to serious complications, whereas in others, it is detected on screening endoscopy. As the proportion of the population that receives regular health examination increases, the detection of asymptomatic PUD also appears to increase.

According to the previous studies, PUD has a strong association with cigarette smoking, advanced age, former alcohol use, obesity, and specific chronic diseases.[16] However, the clinical significance and pathogenic factors associated with asymptomatic PUD remain unclear to date.

Therefore, the present study aimed to investigate the prevalence of symptomatic and asymptomatic PUD in individuals receiving regular medical check-ups in coastal Odisha, India, and we attempted to identify risk factors for the development of symptoms in patients with PUD.

METHODS

This prospective observational study was conducted in two gastrointestinal (GI) clinics in Cuttack, an eastern coastal region of Odisha, India, from January 2015 to January 2017. Consecutive patients of both genders, coming to the clinic with the symptoms suggestive of PUD and dyspepsia, i.e., upper abdominal pain, anorexia, vomiting, bloating, belching, GI bleeding, and gastric outlet obstructive symptoms were subjected to endoscopy. Patients are having gastric ulcer, duodenal ulcer or both on upper GI endoscopy were included in the study. In the study, period a total of 3000 patients with endoscopic findings of PUD were finally enrolled.

PUD was defined on the basis of the endoscopy findings as a mucosal break of diameter 5 mm or larger, covered with fibrin. Mucosal breaks smaller than 5 mm were considered as erosions. The endoscopic procedure was conducted using video endoscope (GFI-250, Olympus, Tokyo, and Japan). Total two biopsy specimens were collected from antrum of the stomach of the patients who were found to have PUD. Endoscope and biopsy forceps were disinfected using 2% glutaraldehyde. Instruments were immersed in the solution for 15 min. All subjects provided written consent before the procedure.

Rapid urease tests (RUT) were performed rapidly with one of the four antral specimens using commercially available kit at diagnosis of PUD. Results were available at the end of 10 min and noted in the datasheet.

H. pylori positive patients were treated with 14 days course of triple therapy for *H. pylori* along with PPI and sucralfate and were followed up. Patients were followed up for compliance of drugs and side effects. Follow-up endoscopies were performed at least 4 weeks after completion of therapy.

All analyses were conducted using SPSS version 19.0 and $P < 0.05$ was considered statistically significant.

RESULTS

Between 2015 and 2017, 3000 patients with peptic ulcer were identified by upper GI endoscopy which accounted to 25% of total endoscopy performed. Of them, 2256 patients were male, and 744 patients were female with a male-female ratio of approximately 3:1. Age of the patients ranged from 8 to 95 years with a mean age of 35 years. The patients were mostly of low socioeconomic condition (75 %). The endoscopic findings at enrolment are shown in Table 1. Duodenal ulcer was the most common peptic ulcer.

Most common risk factor was smoking. Different risk factors are shown in Table 2.

Most common mode of presentation was an epigastric pain. Different modes of presentation are shown in Table 3.

On the basis of findings suggestive of PUD at endoscopy, 3000 patients were included in the study. Among 926

Table 1: Endoscopic findings at enrolment

Pepticulcer	Total *n*=3000 (%)
Duodenal ulcer	1480 (49.33)
Gastric ulcer	917 (30.56)
Duodenal ulcer and gastric ulcer	603 (20.1)

Table 2: Risk factors for peptic ulcer

Risk factors	Percentage
Smoking	38
NSAID intake	22
Alcohol	10
Indigenous drug	13
Multiple	8

NSAID: Nonsteroidal anti-inflammatory drugs

Table 3: Mode of presentation for pepticulcer

Mode of presentation	Percentage
Epigastric pain	36
Dyspepsia	26
GI bleed	24
Gastric outlet obstructive symptoms	14

patients, RUT could be done. 682 (73.65%) were positive and 244 (26.35%) were negative for RUT.

Among these 682 *H. pylori* positive patients, 340 patients had duodenal ulcer, and 220 had gastric ulcers, and 132 patients had both gastric and duodenal ulcer [Table 4].

All patients with PUD treated with anti-*H. pylori* regimen along with PPI and sucralfate including those were *H. pylori* positive. Patients were treated with 14 days course of triple therapy for *H. pylori* and were followed up. Triple therapy contained 2 weeks regimen of amoxicillin (750 mg, bd), clarithromycin (500 mg, bd), and esomeprazole (40 mg, bd). 521 patients returned for follow-up at least 4 weeks after completion of 14 days triple therapy. Endoscopy was performed in the 521 patients who returned for follow-up, 65 patients had duodenal ulcer, 32 had gastric ulcer, and 23 had both duodenal ulcer and gastric ulcer. Complete healing occurred in almost 75% of patients [Table 5].

DISCUSSION

Peptic ulcer is frequently seen in India. In some studies, a higher incidence has been reported from southern India compared to that of northern India.[17-19] Other studies, however, have failed to confirm such regional differences.[20-22] Various factors including differences in diet,[23] socioeconomic status,[18,21] occupation,[21] smoking,[20,24] or alcohol consumption[20] have been incriminated for these differences. We encounter very high number of PUD in this PPI era also. Many patients take some PPI which are available widely before consulting with gastroenterologist. Still then it accounts to 25% of our total endoscopy. Mean age of the patients in this study was 35 years, males were predominant (75%) and most of the patients were from low socioeconomic class (75%). We had 26 children of below 16 years of age. The *H. pylori* positivity in our study is more or less consistent with other studies in our country carried out on patients of PUD due to *H. pylori*.[25,26] According to Maastricht III consensus conference – 2005, diagnosis is confirmed and treatment can be started if RUT is positive.[27] In the current study, *H. pylori* status was considered to be positive if RUT was found to be positive. Epidemiological studies from India have shown 70%,[28] 77.2%,[29] 78%,[30] and 79%[31] prevalence of *H. pylori* infection. Our study also revealed, in accordance with other studies, similar higher association of *H. pylori* with duodenal and gastric ulcer. *H. pylori* eradication rate in this study was 75%. According to Maastricht III Consensus Report, *H. pylori* eradication should be more than 80% for any eradication references regimen to be effective.[27] However, most of published studies in our country failed to attain eradication rate more than 70%.[32-34] Many of these trials used a single test (RUT) to determine clearance of *H. pylori* infection. When rigorous criteria (i.e., a combination of negative urease test, negative histology, and negative urea breath test) were applied, as in a prospective trial from northern India, the eradication rate was considerably lower.[30] Healing rate of peptic ulcer was 75% in this study. Pooled data show that eradication therapy heals >90% of duodenal ulcers and >85% of gastric ulcers, while individual studies repeatedly confirm that it is more effective at healing ulcers than conventional treatment with antisecretory drugs.[20] In a study by Suzuki *et al.*, the eradication rate of *H. pylori* was 84% in the gastric ulcer group and 89% in the duodenal ulcer group.[35] An intimate connection that exists between peptic ulcer and *H. pylori* status; and causal link between the eradication of *H. pylori* and healing of peptic ulcers is well known. However, adequate ulcer healing was achieved in this study despite relatively low eradication rate.

Table 4: Endoscopic findings of *H. pylori* positive patients

Pepticulcer	Percentage
Duodenal ulcer	49.85
Gastric ulcer	30.8
Duodenal ulcer and gastric ulcer	19.35

Table 5: Endoscopic findings *H. pylori* positive patients 4 weeks after completion of triple therapy

Results	Percentage
Complete healing	75
Non-healing	10
Partial healing	15

CONCLUSION

PUD is very common in coastal eastern Odisha. Among them duodenal ulcer is the most common variety. Epigastric pain is the most common type of presentation. Smoking is the most common risk factor followed by NSAID intake. *H. pylori* association causes mostly duodenal ulcers. Complete healing of ulcers occurs in two-third cases after *H. pylori* eradication regimen. Further studies required for complete healing of ulcers in remaining partial and non-healing ulcer cases.

REFERENCES

1. Feldman M, Friedman LS, Brandt LJ, editors. Sleisenger and Fordtran's Gastrointestinal and Liver Disease. 9th ed. Philadelphia, PA: Elsevier; 2010. p. 861.
2. Mejia A, Kraft WK. Acid peptic diseases: Pharmacological approach to treatment. Expert Rev Clin Pharmacol 2009;2:295-314.
3. Marshall BJ, Warren JR. Unidentified curved bacilli in the stomach of

patients with gastritis and peptic ulceration. Lancet 1984;1:1311-5.
4. Marshall BJ, Armstrong JA, McGechie DB, Glancy RJ. Attempt to fulfil koch's postulates for pyloric campylobacter. Med J Aust 1985;142:436-9.
5. Milosavljevic T, Kostić-Milosavljević M, Jovanović I, Krstić M. Complications of peptic ulcer disease. Dig Dis 2011;29:491-3.
6. Al Dhahab H, McNabb-Baltar J, Al-Taweel T, Barkun A. State-of-the-art management of acute bleeding peptic ulcer disease. Saudi J Gastroenterol 2013;19:195-204.
7. Wolfe MM, Lichtenstein DR, Singh G. Gastrointestinal toxicity of nonsteroidalantiinflammatory drugs. N Engl J Med 1999;340:1888-99.
8. Hopkins RJ, Girardi LS, Turney EA. Relationship between *Helicobacter pylori* eradication and reduced duodenal and gastric ulcer recurrence: A review. Gastroenterology 1996;110:1244-52.
9. Atherton JC, Cao P, Peek RM Jr, Tummuru MK, Blaser MJ, Cover TL, *et al.* Mosaicism in vacuolating cytotoxin alleles of *Helicobacter pylori*. Association of specific vacA types with cytotoxin production and peptic ulceration. J Biol Chem 1995;270:17771-7.
10. Hunt RH, Yuan Y. Acid-NSAID/aspirin interaction in peptic ulcer disease. Dig Dis 2011;29:465-8.
11. Yamaoka Y, Ojo O, Fujimoto S, Odenbreit S, Haas R, Gutierrez O, *et al. Helicobacter pylori* outer membrane proteins and gastroduodenal disease. Gut 2006;55:775-81.
12. Potamitis GS, Axon AT. *Helicobacter pylori* and nonmalignant diseases. Helicobacter 2015;20 Suppl 1:26-9.
13. Thorat MA, Cuzick J. Prophylactic use of aspirin: Systematic review of harms and approaches to mitigation in the general population. Eur J Epidemiol 2015;30:5-18.
14. Sasaki H, Nagahara A, Hojo M, Asaoka D, Matsumoto K, Osada T, *et al.* Ten-year trend of the cumulative *Helicobacter pylori* eradication rate for the 'japanese eradication strategy'. Digestion 2013;88:272-8.
15. Kim JH, Kim HY, Kim NY, Kim SW, Kim JG, Kim JJ, *et al.* Seroepidemiological study of *Helicobacter pylori* infection in asymptomatic people in south Korea. J Gastroenterol Hepatol 2001;16:969-75.
16. Lau JY, Sung J, Hill C, Henderson C, Howden CW, Metz DC, *et al.* Systematic review of the epidemiology of complicated peptic ulcer disease: Incidence, recurrence, risk factors and mortality. Digestion 2011;84:102-13.
17. Tovey F. Peptic ulcer in India and Bangladesh. Gut 1979;20:329-47.
18. Dogra JR. Incidence of peptic ulcer in India with special reference to South India. Indian J Med Res 1941;29:665-76.
19. Malhotra SL. Peptic ulcer in India and its etiology. Gut 1964;5:412-6.
20. Sehgal AK, Chhuttani PN, Gupta BB, Malik K, Gupta HD. Epidemiology of peptic ulcer in an urban community in Chandigarh. Indian J Med Res 1971;59:1612-20.
21. Chuttani CS, Wig KL, Chablani TD, Vasudeva YL, Gadekar NG, Chuttani HK. Epidemiology of peptic ulcer. I. Prevalence of peptic ulcer in an urban community of Delhi. II. Significance of various epidemiological factors in the occurrence of peptic ulcer in a community. Indian J Med Res 1967;55:1121-39.
22. Madangopalan N, Balakumar K, Jaishreegajraj A. Epidemiology of peptic ulcer in India. Indian J Gastroenterol 1985;5 Suppl:3-6.
23. Malhotra SL. A comparison of unrefined wheat and rice diets in the management of duodenal ulcer. Postgrad Med J 1978;54:6-9.
24. Doll R, Jones FA, Pygott F. Effect of smoking on the production and maintenance of gastric and duodenal ulcers. Lancet 1958;1:657-62.
25. Abraham P, Bhatia SJ. Position paper on *Helicobacter pylori* in India. Indian society of gastroenterology. Indian J Gastroenterol 1997;16 Suppl 1:S29-33.
26. Singh V, Trikha B, Nain CK, Singh K, Vaiphei K. Epidemiology of *Helicobacter pylori* and peptic ulcer in India. J Gastroenterol Hepatol 2002;17:659-65.
27. Malfertheiner P, Megraud F, O'Morain C, Bazzoli F, El-Omar E, Graham D, *et al.* Current concepts in the management of *Helicobacter pylori* infection: The maastricht III consensus report. Gut 2007;56:772-81.
28. Singh V, Trikha B, Vaiphei K, Nain CK, Thennarasu K, Singh K, *et al.* Helicobacter pylori: Evidence for spouse-to-spouse transmission. J Gastroenterol Hepatol 1999;14:519-22.
29. Katelaris PH, Tippett GH, Norbu P, Lowe DG, Brennan R, Farthing MJ, *et al.* Dyspepsia, helicobacter pylori, and peptic ulcer in a randomly selected population in India. Gut 1992;33:1462-6.
30. Misra V, Misra SP, Dwivedi M, Singh PA. Point prevalence of peptic ulcer and gastric histology in healthy Indians with *Helicobacter pylori* infection. Am J Gastroenterol 1997;92:1487-91.
31. Graham DY, Adam E, Reddy GT, Agarwal JP, Agarwal R, Evans DJ Jr, *et al.* Seroepidemiology of *Helicobacter pylori* infection in India. Comparison of developing and developed countries. Dig Dis Sci 1991;36:1084-8.
32. Dayal VM, Kumar P, Kamal J, Shahi SK, Agrawal BK. Triple-drug therapy of *Helicobacter pylori* infection in duodenal ulcer disease. Indian J Gastroenterol 1997;16:46-8.
33. Bhasin DK, Sharma BC, Sinha SK, Ray P, Vaiphei K, Singh K, *et al. Helicobacter pylori* eradication: Comparison of three treatment regimens in India. J Clin Gastroenterol 1999;28:348-51.
34. Bhatia V, Ahuja V, Das B, Bal C, Sharma MP. Use of imidazole-based eradication regimens for *Helicobacter pylori* should be abandoned in North India regardless of *in vitro* antibiotic sensitivity. J Gastroenterol Hepatol 2004;19:619-25.
35. Suzuki J, Mine T, Kobayasi I, Fujita T. Relationship between the eradication of *Helicobacter pylori* and the healing pattern of peptic ulcer. J Clin Gastroenterol 1998;27 Suppl 1:S159-62.

How to cite this article: Das HS, Panda CR, Behera SK. Peptic Ulcer Disease in the Proton Pump Inhibitor Era in Coastal Odisha. Int J Sci Stud 2017;5(8):8-11.

Source of Support: Nil, **Conflict of Interest:** None declared.