



Prevalence of Helicobacter Pylori Infection in Patient's of Perforated Peptic Ulcer

Authors

Dr Gajendra Saxena¹, Dr Rambabu Meena², Dr Ruchi Saxena³

¹Senior Surgeon (Ms in Surgery), Dept. of General Surgery, S. P. Medical College & Associated Group of Hospitals, Bikaner, Rajasthan

²Assistant Professor, Dept. of General Surgery, S. P. Medical College & Associated Group of Hospitals, Bikaner, Rajasthan

³Asst. Professor, Dept. of Obstetrics & Gynaecology, S.P Medical College, Bikaner, Rajasthan
Corresponding Author

Dr Gajendra Saxena

Email: drgajendrasaxena@gmail.com, Mobile no.: 09414603668

Abstract

Background: Peptic ulcer disease had a tremendous effect on morbidity and mortality until the last decades of the 20th century when epidemiological trends started to point to an impressive fall in its incidence. The present study to assess the prevalence of Helicobacter Pylori (HP) infection in patients undergoing emergency surgery for repair of complications of peptic ulcer.

Material & Methods: A prospective hospital based study in Dept. of Surgery, S.P. Medical College and P.B.M Hospital, Bikaner, All patients undergoing emergency surgeries within study duration and fulfilling eligibility criteria were included in the study. All patients underwent surgery. Biopsy was taken during operation for HP nearly surgical area. Evaluation of cytology results was based on the following criteria. We subdivided the results of 50 cytology specimens in 3 categories: material unsatisfactory for interpretation, negative results and positive results

Results: In this study a total 50 cases of perforated peptic ulcer were identified during study period. Out of 50 cases 35 (70%) were males and 15 (30%) were females (table 1) with mean age 45.8 ± 10.76 (table 2). The most common symptoms clinically were epigastric / upper abdominal pain 30(60%), heartburn 25 (50%), nausea / vomiting 28(56%) and constipation 25 (50%). The precipitating factors like use of NSAIDs, cigaret smoking, and alcoholism were found in 50%, 38%, and 10% respectively.

Conclusion: Most of the times there are no alarming signs before actual ailment, but seeking proper medical help in time results in favourable results. They should be advised to avoid the common risk factors like too much spicy food, smoking, excess alcohol use, and indiscriminate use of NSAIDs and should seek proper medical advice in time.

Key Words: Helicobacter pylori, NSAIDs, Perforated Peptic ulcer, Laprotomy.

Introduction

Peptic ulcer disease (PUD), also known as a peptic ulcer or stomach ulcer, is a break in the

lining of the stomach, first part of the small intestine, or occasionally the lower esophagus.^{1,2}

An ulcer in the stomach is known as a gastric

ulcer while that in the first part of the intestines is known as a duodenal ulcer. The most common symptoms of a duodenal ulcer are waking at night with upper abdominal pain or upper abdominal pain that improves with eating.¹ With a gastric ulcer the pain may worsen with eating.³ The pain is often described as a burning or dull ache. Other symptoms include belching, vomiting, weight loss, or poor appetite. About a third of older people have no symptoms.¹ Complications may include bleeding, perforation, and blockage of the stomach. Bleeding occurs in as many as 15% of people.⁴

Peptic ulcers are present in around 4% of the population.¹ They newly began in around 53 million persons worldwide in 2013.⁵ About 10% of people develop a peptic ulcer at some point in their life.⁶ They resulted in 301,000 deaths in 2013 down from 327,000 deaths in 1990.⁶ The first description of a perforated peptic ulcer was in 1670 in Princess Henrietta of England.⁴ *H. pylori* was first identified as causing peptic ulcers by Barry Marshall and Robin Warren in the late 20th century,⁷ a discovery for which they received the Nobel Prize in 2005.⁸

Complications

- Gastrointestinal bleeding is the most common complication. Sudden large bleeding can be life-threatening.⁹ It occurs when the ulcer erodes one of the blood vessels, such as the gastroduodenal artery.
- Perforation (a hole in the wall of the gastrointestinal tract) often leads to catastrophic consequences if left untreated. Erosion of the gastro-intestinal wall by the ulcer leads to spillage of stomach or intestinal content into the abdominal cavity. Perforation at the anterior surface of the stomach leads to acute peritonitis, initially chemical and later bacterial peritonitis. The first sign is often sudden intense abdominal pain; an example is Valentino's syndrome, named after the silent-film actor who experienced this pain

before his death. Posterior wall perforation leads to bleeding due to the involvement of gastroduodenal artery that lies posterior to the first part of the duodenum.

- Penetration is a form of perforation in which the hole leads to and the ulcer continues into adjacent organs such as the liver and pancreas.¹⁰
- Gastric outlet obstruction is a narrowing of the pyloric canal by scarring and swelling of the gastric antrum and duodenum due to peptic ulcers. The person often presents with severe vomiting without bile.
- Cancer is included in the differential diagnosis (elucidated by biopsy), *Helicobacter pylori* as the etiological factor making it 3 to 6 times more likely to develop stomach cancer from the ulcer.¹¹

In Western countries the percentage of people with *Helicobacter pylori* infections roughly matches age (i.e., 20% at age 20, 30% at age 30, 80% at age 80 etc.). Prevalence is higher in third world countries where it is estimated at about 70% of the population, whereas developed countries show a maximum of 40% ratio. Overall, *H. pylori* infections show a worldwide decrease, more so in developed countries. Transmission is by food, contaminated groundwater, and through human saliva (such as from kissing or sharing food utensils).¹²

A minority of cases of *H. pylori* infection will eventually lead to an ulcer and a larger proportion of people will get non-specific discomfort, abdominal pain or gastritis.

Peptic ulcer disease had a tremendous effect on morbidity and mortality until the last decades of the 20th century when epidemiological trends started to point to an impressive fall in its incidence. The reason that the rates of peptic ulcer disease decreased is thought to be the development of new effective medication and acid suppressants and the discovery of the cause of the condition, *H. pylori*.

The incidence of duodenal ulcers has dropped significantly during the last 30 years, while the

incidence of gastric ulcers has shown a small increase, mainly caused by the widespread use of NSAIDs. The drop in incidence for duodenal ulcers is considered to be a cohort-phenomenon independent of the progress in the treatment of the disease. The cohort-phenomenon is probably explained by improved standards of living which has lowered the incidence of *H. pylori* infections. When it comes to treatment of ulcer perforation, modern times have allowed treatment of elderly patients with associated severe diseases, the ulcer perforation patients we usually meet today. However, lethality after ulcer perforation is still relatively high, representing a potential for improvement. The significant evidence suggests the potential role of *Helicobacter pylori* infection in the development of complications of peptic ulcer disease. The present study to assess the prevalence of *Helicobacter Pylori* (HP) infection in patients undergoing emergency surgery for repair of complications of peptic ulcer.

Material & Methods

A prospective hospital based study in Dept. of Surgery, S.P. Medical College and P.B.M Hospital, Bikaner, All patients undergoing emergency surgeries within study duration and fulfilling eligibility criteria were included in the study.

Inclusion Criteria

1. Patients undergoing emergency surgery
2. Patients of all ages, sex & socio economic status.

Exclusion Criteria

1. patients already on *Helicobacter pylori* eradication therapy
2. patients with malignant ulcer with perforation
3. Traumatic perforation
4. Patients who refused to give consent to participate into study.

Procedure of Data Collection

The findings of history, clinical examination, and demographic characteristic were noted for each patient. For all cases baseline investigations such

as blood complete picture, blood sugar and urea, serum electrolytes and X-ray chest were performed; specific investigations such as X-ray abdomen (erect/ supine) to detect free gas under right dome of diaphragm, ultrasound abdomen to detect free fluid in peritoneal cavity and serology (anti *H. pylori* antibodies) to detect the *Helicobacter pylori* infection were also done. For histopathological detection of *H pylori*, biopsy from the edge of ulcer was taken during surgery. Data collection sheets were filled in by the investigator himself. The operation procedure and related preoperative factors were observed directly and recorded in the data collection sheet instantly.

Technique

All patients underwent surgery. Biopsy was taken during operation for HP nearby surgical area. Evaluation of cytology results was based on the following criteria. We subdivided the results of 50 cytology specimens in 3 categories: material unsatisfactory for interpretation, negative results and positive results.

Data Analysis

To collect required information from eligible patients a pre-structured pre-tested Proforma were used. The data presented as number and percent for qualitative variables, while quantitative variables presented as mean and SD \pm . . SPSS version 10.00 used as statistical software.

Results

In this study a total 50 cases of perforated peptic ulcer were identified during study period. Out of 50 cases 35 (70%) were males and 15 (30%) were females (table 1) with mean age 45.8 ± 10.76 (table 2). The most common symptoms clinically were epigastric / upper abdominal pain 30(60%), heartburn 25 (50%), nausea / vomiting 28(56%) and constipation 25 (50%). The precipitating factors like use of NSAIDS, cigaret smoking, and alcoholism were found in 50%, 38%, and 10% respectively. The generalized tenderness was present in all cases especially marked in upper abdomen. Abdomen was distended in 42 (84%)

patients. The abdomen of 38 (76%) patients exhibited a board like rigidity and absent bowel sounds.

In this study the operative procedure of perforated duodenal ulcer (first part – anteriorly) was found in 35 (70%) patients and 22(44%) patients had perforated gastric ulcer (antrum anteriorly). The size of perforated duodenal ulcer was less than 1-cm but in three (6%) patients the size exceeded 1 cm. Gastric ulcer was also < 1-cm in size but in four (8%) patients it was 1.5 to 2cm in size. The results of diagnostic tests for *H. pylori* are shown in (table 3).

Table 1: Gender wise incidence

Gender	Patients	Percentage
Male	35	70
Female	15	30

Table 2: Age wise distribution

Age in years	Patients	Percentage
20-30	10	20
31-50	30	60
More than 50	10	20

Table 3: Represent diagnostic test for helicobacter pylori

Test	Patients	Percentage
Serology	50	100
Histology	35	70

Discussion

The results of present study showed that *H. pylori* infection is more prevalent in males (70%), finding is consistent with the study of Oladejo¹³, where 73% male were infected. However Kaffes,¹⁴ found it more common in females. In our study the mean age of patients was 45.8 years, though it ranged from 48-70 years in other studies.^{15,16}

Both *Helicobacter pylori* infection and non steroidal anti-inflammatory drugs (NSAIDs) are independent risk factors for peptic ulcer disease, but the potential synergism between these factors is controversial.¹⁷ In patients with history of ulcer disease, new ulcers are expected to develop irrespective of NSAID use; the histamine H₂-receptor antagonist reduces the rate of recurrence

of *H. pylori*-related ulcers but found completely ineffective for preventing NSAID-related ulcers.¹⁸ A another study in Beninese population, *H. pylori* infection is almost equally distributed in urban and rural population (75% in urban and 72% in rural residents). The important predictor found were, density of family members (more than 3 persons sharing a room), family contact with infected persons and crowded living. Therefore improvement in living conditions can reduce intrafamilial *H. pylori* transmission.¹⁹

Although Incidence of perforated gastroduodenal peptic ulcer has reduced to 50% over the last 6 years due to the increased use of proton pump inhibitors²⁰ yet peptic ulcer when perforate still carries high mortality, delay in surgical management is considered a major determinant.²¹

The sensitivity and specificity of serology assay varies from 52-94.5%, and 60-97.2% respectively.²² The biopsy- based methods, on the other hand have a low sensitivity (83%), but a high specificity (100%). In this study, histological diagnosis of *H. pylori* was made in over 70%. Other similar studies report histological diagnosis varying from 60 and 87%. Such wide difference may be due either to different methods used for culture or to high false negative resulting from indiscriminate use of antibiotics hindering growth of this microorganism. Several studies have been under- taken to define the association and possible etiological role of *H. pylori* in perforated peptic ulcer. We found 70% incidence of perforated peptic ulcer, slightly higher than that reported by Gisbert, et al.²³

The reason for variation in prevalence could be ethnic background, age of patients selected, and sensitivity of the tests done. Remaining of 30% with peptic ulcer perforation in this study were serum anti *H. Pylori* positive but negative on histopathology.

Conclusion

Most of the times there are no alarming signs before actual ailment, but seeking proper medical help in time results in favourable results. They

should be advised to avoid the common risk factors like too much spicy food, smoking, excess alcohol use, and indiscriminate use of NSAIDs and should seek proper medical advice in time.

References

1. Najm, WI (September 2011). "Peptic ulcer disease.". *Primary care*. 38 (3): 383–94.
2. "Definition and Facts for Peptic Ulcer Disease". National Institute of Diabetes and Digestive and Kidney Diseases. Retrieved 28 February 2015.
3. Rao, S. Devaji (2014). *Clinical Manual of Surgery*. Elsevier Health Sciences. p. 526.
4. Milosavljevic, T; Kostić-Milosavljević, M; Jovanović, I; Krstić, M (2011). "Complications of peptic ulcer disease.". *Digestive diseases (Basel, Switzerland)*.29(5):491–3.
5. Global Burden of Disease Study 2013, Collaborators (22 August 2015). "Global, regional, and national incidence, prevalence, and years lived with disability for 301 acute and chronic diseases and injuries in 188 countries, 1990-2013: a systematic analysis for the Global Burden of Disease Study 2013.". *Lancet (London, England)*. 386 (9995): 743–800.
6. Snowden FM (October 2008). "Emerging and reemerging diseases: a historical perspective". *Immunol. Rev.*225 (1):9–26.
7. GBD 2013 Mortality and Causes of Death, Collaborators (17 December 2014). "Global, regional, and national age-sex specific all-cause and cause-specific mortality for 240 causes of death, 1990-2013: a systematic analysis for the Global Burden of Disease Study 2013.". *Lancet*. 385: 117–71.
8. Wang, AY; Peura, DA (October 2011). "The prevalence and incidence of *Helicobacter pylori*-associated peptic ulcer disease and upper gastrointestinal bleeding throughout the world.". *Gastrointestinal endoscopy clinics of North America*. 21 (4): 613–35.
9. The Nobel Prize in Physiology or Medicine 2005". nobelprize.org. Nobel Media AB. Retrieved 3 June 2015.
10. Cullen DJ; Hawkey GM; Greenwood DC; et al. (1997). "Peptic ulcer bleeding in the elderly: relative roles of *Helicobacter pylori* and non-steroidal anti-inflammatory drugs". *Gut*. 41 (4): 459–62.
11. "Peptic Ulcer". Home Health Handbook for Patients & Caregivers. Merck Manuals. October 2006.
12. Brown LM (2000). "*Helicobacter pylori*: epidemiology and routes of transmission.". *Epidemiol. Rev.* 22(2): 283–97.
13. Oladejo O. Lawal, Rotimi O, Okeke I. *Helicobacter Pylori* in Gastrointestinal Diseases. *Journal of the National Medical Association* 2007;99(1):31-4.
14. Kaffes A, Cullen J, Mitchell H, Katelaris PH. Effect of *Helicobacter Pylori* Infection and Low-Dose Aspirin Use on Iron Stores in the Elderly 2003. *Gastroenterol Hepatol* 18(9):1024-8.
15. Bjorkman J. H. *pylori*, NSAIDs, and Peptic Ulcer Bleeding 2005. *Clin Gastroenterol Hepatol*;3:859-64.
16. Oliveira AG, Santos A, Guerra GB, Rocha GA, Rocha AM, Oliveira CA, et al. babA2- and cagA Positive *Helicobacter pylori* Strains Are Associated with Duodenal Ulcer and Gastric Carcinoma in Brazil. *Journal of Clinical Microbiology* 2003; 41 (8): 3964-6.
17. Bjorkman DJ. NSAIDs and H. *pylori*: A Dangerous Combination?. *Lancet* 2002; 359:3-4.
18. Graham DY. NSAIDs, *Helicobacter pylori*, and Pandora's Box. *The New England Journal of Medicine* 2002;347 (26):2162-4.
19. Aguemon BD, Struelens MJ, Massoug-bodji A, Ouendo EM. Prevalence and risk-factors for *Helicobacter pylori* infection in urban and rural Beninese populations.

Clinical microbiology and infection
2005;11(8):611-7.

20. Bueno FS, Marín P, Ríos A, Aguayo JL, Robles R, Pinero A, et al. Has the Incidence of Perforated Peptic Ulcer Decreased over the Last Decade? / with Invited Commentary. *Dig Surg* 2001; 18:444-8.
21. Svanes C. Trends in perforated peptic ulcer: incidence, etiology, treatment, and prognosis. *World J Surg* 2000;24(3):277-83.
22. Essa F, Taj Y, Kazmi SU, Abdullah E. Sensitivity and Specificity of various diagnostic tests in the detection of *Helicobacter Pylori*. *J Coll Physicians Surg Pak* 2003;13(2):90-3.
23. Gisbert JP, Legido J, Sanz IG, Pajares M. *Helicobacter pylori* and perforated peptic ulcer. Prevalence of the infection and role of non-steroidal anti-inflammatory drugs. *Digestive and Liver Disease* 2004;36(2): 116-20.