



A PROSPECTIVE STUDY OF PEPTIC PERFORATION IN BUNDELKHAND REGION IN REFERENCE TO PREVALENCE OF H. PYLORI PEPTIC PERFORATION

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ABSTRACT

Purpose: The main aims of study were-Peptic perforation in reference to prevalence of H. pylori infection, age distribution of the patients with peptic perforation, sex distribution of H.pylori in peptic perforation, size and site of perforation, residential living of the patients.

Method: A total of 51 Patients who were diagnosed as a case of peptic perforations, were included in this prospective study conducted in the Department of surgery, Maharani Laxmi Bai Medical College, Jhansi over a period of June 2015 to July. 2017. Proper history, preoperative assessment, intra-operative evaluation and biopsy were taken and Rapid ureas test (RUT) was done.

Results: Highest incidence of perforated peptic ulcer was between 31 to 60 years, 70.58% of patients fell in this group. The mean age of presentation was 40 years. Male to female ratio in peptic perforation was 5:1. 70.58% patients were smokers and (60.78%) patients were drinker. 32 (62.74%) were rural and rest of 19 (37.25%) were urban residents. Size of perforation is less than 1 cm in 41 (80.39%) patients and 1 to 2 cm in 10 (19.60%) patients. In present study patients presenting with acute perforation of peptic ulcer, prevalence of H. pylori is 92.15%.

Conclusion: In this study perforated peptic ulcers are clearly associated with H. pylori infection as a strong etiological factor. Most common peptic perforation was prepyloric perforation. Commonest age of presentation of peptic perforation in Bundelkhand region was 30-60 years. Three decades after the discovery of *Hp*, the etiologies of bleeding peptic ulcers are changing. However, diagnosis of *Hp* infection is still the first priority in these patients. Invasive RUT is most frequently used.

KEYWORDS :Bleeding peptic ulcer, H. pylori, Peptic perforation, peptic ulcer, Rapid ureas test (RUT)

INTRODUCTION

Peptic ulcer disease (PUD) is a break in the lining of the stomach, first part of the small intestine or occasionally the lower esophagus.¹ 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,¹ usually as a result of inflammation caused by the bacteria *H. pylori*, as well as from erosion from stomach acids. Peptic ulcers are a fairly common health problem. There are three types of peptic ulcers:

- Gastric ulcers:
- Esophageal ulcers:
- Duodenal ulcers:

Left untreated, peptic ulcer diseases (PUD) will cause major complications, such as hemorrhage, perforation, or obstruction in 20–25% of patients. Among these complications, upper gastrointestinal (UGI) bleeding is the most frequently encountered, accounting for about 70% of cases^{2,3}. With the discovery of *Helicobacter pylori* (*Hp*)⁴, the pathogenic relationship between PUD and *Hp* infection has come into focus. Worldwide consensus guidelines recommend the mandatory eradication of *Hp* in patient with PUD^{5,6,7}.

Defense of normal gastric mucosa against aggressive factors:

Three basic levels of defense underlie the remarkable ability of normal gastroduodenal mucosa to resist injury from the acid and peptic activity in gastric juice.

1. Surface epithelial cells secrete mucus and bicarbonate, creating a pH gradient in the mucus layer and change the very acidic gastric lumen to the nearly neutral surface of the mucosa⁸.
2. Gastric mucosal cells have a specialized apical surface membrane that resists the diffusion of acid back into the cell.
3. Mucosal cells may directly resist injury by intrinsic mechanisms, such as the extrusion of back-diffused hydrogen ions by means of basolateral carriers (e.g. sodium-hydrogen or sodium bicarbonate exchange)⁹,

Perforation is one of the most catastrophic complications of peptic ulcer. In spite of modern advance in surgical, anesthetic and ancillary facilities, it still assumes life-threatening dimension. Prompt recognition of the condition is of paramount importance, because with diagnosis and treatment it is possible to reduce that still relative high mortality. A perforated ulcer, is a condition where an untreated ulcer can burn through the wall of the stomach (or other areas of the gastrointestinal tract), allowing digestive juices and food to leak into the abdominal cavity. Treatment generally requires immediate surgery.

A diagnosis is made by taking an erect abdominal/chest X-ray (seeking air under the diaphragm). This is in fact one of the very few occasions in modern times where surgery is undertaken to treat an ulcer. Many of the perforated ulcers have been attributed to the bacterium *Helicobacter pylori*. The incidence of perforated ulcer is steadily declining, though there are still incidents where it occurs. Causes include smoking and non-steroidal anti-inflammatory drugs (NSAIDs). A perforated ulcer can be grouped into a stercoral perforation which involves a number of different things that causes perforation of the intestine wall.

The gold standard for diagnosis of *H.pylori* infection has been biopsy obtained during endoscopy. However, no single test has yet emerged as definitive in daily clinical practice for several reasons.

Treatment will depend on the underlying cause of patient's ulcer. If tests show that patients have an *H. pylori* infection, we will prescribe a combination of medication, which patients have to take for up to two weeks. The medications include antibiotics to help kill infections, and proton pump inhibitors (PPIs) to help reduce stomach acid.

MATERIAL AND METHOD:

A total of 51 Patients who were diagnosed as a case of peptic

perforators, were included in this prospective study conducted in the Department of surgery, Maharani Laxmi Bai Medical College, Jhansi, Uttar Pradesh, India over a period of June 2015 to July, 2017. The procedures followed were in accordance with the ethical standards committee on human experimentation (institutional or regional) and with the Helsinki Declaration of 1975, as revised in 2000. The necessary permission from the Ethical and Research Committee was obtained for the study.

In case of peptic perforation following plan of work was followed:-

1. Preoperative work up (Clinical and Biochemical) as an Evaluation and analysis of symptoms in order to find out duration of perforation and incidence of each symptoms
2. Past history in order to find out high risk cases and incidence of acid peptic disease as like Diabetes mellitus, Hypertension, Drug intake like NSAIDS & steroids and History suggestive of acid peptic disease.
3. Presence/absence of psychological factor (stress, anxiety)
4. Family history
5. General examination and systemic examination in order to assess general condition of patients suitable for anesthesia and surgery. General condition as like blood pressure (m Hg), pulse rate (per min), respiratory rate (per min), anemia, jaundice, urine Output, cardiovascular examination and respiratory examination.
6. Local examination of abdomen to make clinical diagnosis and analysis of signs in order to evaluate incidence of each sign.
7. Routine investigation like Hemogram (Hb, TLC, DLC etc.), renal function (Blood Urea, Serum Creatinine), serum Elelctrolyte (Serum Na⁺ Serum K⁺), radiological examination such as Radiology (Plain X-ray, abdomen USG finding)

All patients were resuscitated with Intravenous fluids, appropriate antibodies and Nasogastric decompression for Ryles tube. All of the patients were operated under general anesthesia and preferable record was midlines. During operation following point were recorded-

- Size of perforation diameter.
- Site of perforation
- Surrounding wall of duodenum
- Condition of omentum

Any additional findings were noted on exploration. Biopsy taken per operative on the table at the time of operation

Postoperative patients were kept nil orally along with ryles tubes aspiration till bowel sounds were heard and flatus appreciated by the patients. Drains were taken out according to the amount of drainage. Ryles tube was removed after 3-5 days. Patients were called up in follow up.

Biopsy is put on the rapid ureas test kit. The result in form of color changes read. Results are read as positive when color changes to pink in the kit.

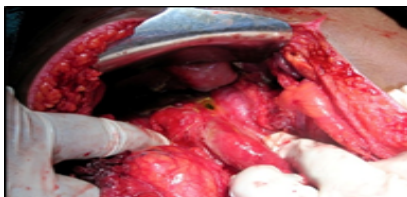


Figure 1: Intraoperative prepyloric perforation



Figure 2: Intraoperative duodenal perforation.



Figure 3: RUT kit with positive result.



Figure 4: RUT kit with negative result.

RESULTS

TABLE 1: AGEWISE DISTRIBUTION

| Age (years) | Total | % |
|-------------|-------|--------|
| 1-10 | 02 | 03.92% |
| 11-20 | 05 | 09.80% |
| 21-30 | 08 | 15.68% |
| 31-40 | 13 | 25.49% |
| 41-50 | 10 | 19.60% |
| 51-60 | 04 | 07.84% |
| >60 | 09 | 17.64% |

TABLE 2: SEX WISE DISTRIBUTION

| Sex | Total | % |
|--------|-------|--------|
| Male | 42 | 82.35% |
| Female | 09 | 17.64% |

TABLE 3: PATIENTS WITH ALCOHOL

| Alcohol | Total | % |
|---------|-------|--------|
| Present | 31 | 60.78% |
| Absent | 20 | 39.21% |

TABLE 4: PATIENTS WITH SMOKING

| Smoking | Total | % |
|---------|-------|--------|
| Present | 36 | 70.58% |
| Absent | 15 | 29.41% |

TABLE 5: PATIENTS WITH RESIDENTIAL LIVING

| Occupation | Total | % |
|------------|-------|--------|
| Rural | 32 | 62.74% |
| Urban | 19 | 37.25% |

TABLE 6: PATIENTS WITH PREVIOUS H/O PEPTIC ULCER

| Previous H/O Peptic Ulcer | Total | % |
|---------------------------|-------|--------|
| Yes | 14 | 27.45% |
| No | 37 | 72.54% |

TABLE 7: PATIENTS WITH SITE OF PERFORATION

| Site of Perforation | Total | % |
|---------------------|-------|--------|
| Duodenal | 05 | 09.80% |
| Pre-Pyloric | 43 | 84.31% |
| Stomach body | 03 | 05.80% |

TABLE 8: PATIENTS WITH SIZE OF PERFORATION (MM)

| Size of Perforation (MM) | Total | % |
|--------------------------|-------|--------|
| 3 mm | 14 | 27.45% |
| 5 mm | 21 | 41.17% |
| 6 mm | 01 | 01.96% |
| 8 mm | 05 | 09.80% |
| 10 mm | 7 | 13.72% |
| 15 mm | 3 | 05.88% |

TABLE 9: PATIENTS WITH RAPID UREASTEST

| Rapid ureas test | Total | % |
|------------------|-------|--------|
| Positive | 47 | 92.15% |
| Negative | 04 | 07.84% |

DISCUSSION**Age distribution:**

In the present study of 51 cases the highest incidence of perforated peptic ulcer was between 31 to 60 years, 70.58% of patients fell in this group. The mean age of presentation was 40 years. A study done on 23 patients at EAST BIRMINGHAM HOSPITAL in 1981-82 showed the mean age of presentation of 56 years. This might be due to the different trends of this entity in the Western countries where the elderly patients are the primary victims. Various case series in India and abroad have mean age of presentation between 40-60 years, and in our study between 30-60 years. I.G. Medical College, Shimla in 1983-92, showed the mean age of presentations between 41-50 years, this is consistent with that of our study. A study on perforated peptic ulcer done in JLN Medical College Ajmer RAJASTHAN, showed the maximum age incidence between 30-40 years, this figure is again compatible to our study.

Gender distribution:

In the present study 42 (82.35%) were male and 9 (17.64%) were female. Male to female ratio in peptic perforation was 5:1. Present study shows high incidence of peptic perforation in male. A study in I.G. Medical College Shimla over the period of 10 years from 1983-1992, showed male to female ratio of 17:1. This ratio shows the very high incidence in male patients, which is shown in our study. A prospective study done in JLN Medical College, AJMER (RAJASTHAN), on 43 patients, all the patients in that study were male and none of patient were female. Various other studies conducted abroad confirm the increasing incidence as well as very high incidence of perforated peptic ulcer in male gender.

H/O smoking:

In our study 70.58% patients were smokers were smoking 20 birc/day and 29.41% were non-smoker. Although erratic, it seems that disease incidence is increasing among non-smokers. The association of smoking to peptic disease does not need emphasizing. These data shows the finding of multi factorial etiology of peptic ulcer smoking and peptic perforation.

H/O alcohol intake: Out of 51 patients 31 (60.78%) patients were drinker and 20 (39.21%) patients were non drinker. Although alcohol is always mentioned as a cause of peptic ulcer no study so far shows any definite association between alcohol intake and peptic ulcer disease (Schwartz 8th ed, 958). In our study there is increased incidence of peptic ulcer perforation in alcoholic group than non-alcoholic group.

Residential living:

In our study 32 (62.74%) were rural and rest of 19 (37.25%) were urban residents. So, from above data's can be safely said that in Bundelkhand region the perforation is more common in rural areas.

Size of perforation:

Out of 51 patients, size of perforation is less than 1 cm in 41 (80.39%) patients and 1 to 2 cm in 10 (19.60%) patients.

RELATION BETWEEN H. PYLORI AND PERFORATED PEPTIC ULCER:**Results of biopsy urease test:**

In present study patients presenting with acute perforation of peptic ulcer, prevalence of H. pylori is 92.15%.

Data regarding H. Pylori infection rate in perforated peptic ulcer is highly variable ranging from 0-92% in different studies (see below table)

| Authors | Years | No. of patients | H.P. Positive (%) |
|-----------|-------|-----------------|-------------------|
| Reinbach | 1993 | 80 | 47% |
| Sebastian | 1995 | 29 | 83% |
| Debongnie | 1995 | 36 | 56% |
| Ng | 1996 | 73 | 70% |
| Chowdhary | 1998 | 15 | 0% |
| Chu | 1999 | 163 | 47% |
| Ng | 2000 | 129 | 81% |
| Sharma | 2000 | 44 | 61% |
| Metzger | 2001 | 47 | 73% |
| Kumar | 2004 | 86 | 50% |
| Our Study | 2017 | 47 | 92.15% |

Above mentioned table shows prevalence of H. Pylori infection in patients with perforated peptic ulcer in different studies performed during last 24 years.

Discrepancy between H. pylori infection rate found in different studies may be attributed in part to different population studied. For example,

Sebastian et al. reported an infection rate of 83% in a small group of young male from India with acute peptic ulcer, this result is comparable to our findings.

Another study from India with 15 perforated duodenal ulcer patients showed on contrary that all patients were negative from H. pylori while Sharma et al found a prevalence of 61% among 44 patients from Chattishgarh region, India.

Metzger et al study reported a prevalence of 73% of H pylori infection in perforated peptic ulcer.

Papaziogas B, Pavlidis T, et al reported a prevalence of 62.5% of H pylori in perforated peptic ulcer.

Annals of surgery 231 (2); 153-158, Feb. 2000, Ng, Enders K.W.MD, LAM, Y.H.MD et al reported a prevalence of 81% in perforated peptic ulcer.

Department of gastroenterology, PG institute of medical education and research, Chandigarh, India, conducted study on 45 pts, 15 (34%) patients were in group of perforated peptic ulcer, none of them tested positive for H. pylori infection.

According to world journal gastroenterology 2013 a retrospective study was conducted on patients admitted with gastric and duodenal perforation at Stavanger university hospital between January 2001 to December 2010. In this study gastric perforation predominated and accounted for 112 of 172 patients. Prepyloric perforation represented 61 of 112 gastric perforation and 21 of 112 were located in the pylorus. In the corpus/fundus area 12 of 112 perforations were observed while 8 of 112 were located in the antrum. 1 perforation was located in an anastomosis and 9 of 112 perforations were missing but being classified as gastric perforation at operation. In our study 46 (90.19%) patients were of prepyloric perforation and 05 (9.8%) were duodenal.

The no of NSAIDs users was stable during the decade study and were used by 76 of 172 patients. Also NSAIDs use was more common in >60 years age compare to younger patients. In our study 14 (27.45%) patients used NSAIDs. So in our study perforated peptic ulcers are clearly associated with H. pylori infection as a strong etiological factor.

CONCLUSION:

In this study it was concluded that -

1. Most common peptic perforation was prepyloric perforation.
2. Commonest age of presentation of peptic perforation in Bundelkhand region was 30-50 years (45.09%).
3. Most commonly men were more affected than females with the

ratio of 5:1

4. According to study only 70.58% patients were chronic smoker.
5. Most of the patients were alcoholic (60.78%).
6. 62.74% of the patients were from rural areas.
7. 27.45% patients gave history of chronic NSAIDs use.
8. Gas under diaphragm was a significant finding in erect X-ray of abdomen in perforated peptic ulcer.
9. Most of the patients (92.15%) were infected with *H. pylori* detected by rapid urease test.
10. It can be concluded that *H. pylori* may be the causative factor for perforation of peptic ulcer.
11. In all factors studied *H. pylori* was most closely associated factor with perforated peptic ulcer.

Three decades after the discovery of *Hp*, the etiologies of bleeding peptic ulcers are changing. However, diagnosis of *Hp* infection is still the first priority in these patients. Invasive RUT is most frequently used, but this methodology is hampered by a high rate of false-negative results, especially in patients with UGI bleeding. Other delayed tests should be performed if the initial diagnostic test is negative.

REFERENCES:

1. Chey WD, Wong BC. American College of Gastroenterology guideline on the management of Helicobacter pylori infection. *Am J Gastroenterol*. 2007 Aug; 102(8):1808-25.
2. L. Laine and W. L. Peterson, "Bleeding peptic ulcer," *The New England Journal of Medicine*, vol. 331, no. 11, pp. 717-727, 1994.
3. J. S. Barthel, "Bleeding ulcers and Helicobacter pylori," *Gastrointestinal Endoscopy*, vol. 46, no. 4, pp. 371-375, 1997.
4. B. J. Marshall and J. R. Warren, "Unidentified curved bacilli in the stomach of patients with gastritis and peptic ulceration," *The Lancet*, vol. 1, no. 8390, pp. 1311-1314, 1984.
5. NIH Consensus Conference, "NIH Consensus Development Panel on Helicobacter pylori in peptic ulcer disease. Helicobacter pylori in peptic ulcer disease," *The Journal of the American Medical Association*, vol. 272, no. 1, pp. 65-69, 1994.
6. P. Malfertheiner, "Current European concepts in the management of Helicobacter pylori infection. The Maastricht consensus report. European Helicobacter Pylori Study Group," *Gut*, vol. 41, no. 1, pp. 8-13, 1997.
7. S. K. Lam and N. J. Talley, "Report of the 1997 Asia Pacific Consensus Conference on the management of Helicobacter pylori infection," *Journal of Gastroenterology and Hepatology*, vol. 13, no. 1, pp. 1-12, 1998.
8. Quigley EM, Turnberg LA. pH of the microclimate lining human gastric studies in control subjects and in duodenal ulcer patients. *Gastroenterology* 1987; 92: 1876-1884.
9. Sanders MJ, Ayalon A, Roil M, Soil AH. The apical surface of canine chief cell monolayer resists H+ back diffusion. *Nature* 1981; 313:52-54.