

## ORIGINAL RESEARCH

# An observational study of clinic-pathological and microbiological correlation of peptic ulcer perforation at tertiary center

<sup>1</sup>Dr. Ashok Yadav, <sup>2</sup>Dr. Durgesh Tripathi, <sup>3</sup>Dr. Nikhil Shukla, <sup>4</sup>Dr. Abhishek Jina

<sup>1</sup>Professor, <sup>2</sup>Assistant Professor, <sup>3</sup>Final year Resident, <sup>4</sup>Associate Professor, Department of Surgery, BRD Medical College, Gorakhpur, Uttar Pradesh, India

### Corresponding Author

Dr. Abhishek Jina

Associate Professor, Department of Surgery, BRD Medical College, Gorakhpur, Uttar Pradesh, India

Received: 05 November, 2023

Accepted: 09 December, 2023

### ABSTRACT

**Aim:** An observational study of clinic-pathological and microbiological correlation of peptic ulcer perforation at tertiary center. **Material and methods:** An analytical investigation was undertaken at B.R.D Medical College, Gorakhpur to establish a link between the clinical and pathological aspects of peptic ulcer perforation. The study also included a microbiological analysis. Based on the findings, ethical clearance was recommended. In this investigation, the patients were categorised into two categories. This research comprised patients from group 1 who had perforation peritonitis caused by peptic ulcer perforation. The patients included in group 2 had an endoscopic diagnosis of peptic ulcer disease. **Results:** The mean age in pre pyloric gastric perforation was  $51.96 \pm 18.27$  and pyloric ulcer perforation was  $42.25 \pm 6.34$ . Out of 30 patients operated for Peptic ulcer perforation in this study, on histopathological examination were found to be non specific chronic inflammatory lesions. The Sensitivity, specificity, positive predictive value (PPV) and negative predictive value (NPV) of diagnosing Gastritis by Rapid Urease Test are shown in Table 6. The positive Rapid Urease Test had more sensitivity of 87.5%, specificity of 92.9%, PPV of 93.3% and NPV of 86.7% in the diagnosis of Gastritis. These tests were demonstrating the accuracy of risk factors. Comparison of rapid urease test in perforated peptic ulcer diseases and Non perforated peptic ulcer disease following result can be commented on the test, sensitivity 70% specificity 46.67% positive predictive value 56.67% negative predictive value 60.87% and accuracy 58.33%. **Conclusion:** The current investigation has shown that perforated peptic ulcer disease (PUD) is a very dangerous condition that has significant risks of both morbidity and mortality. The prevalent symptoms were stomach pain and distension. The rate of hospitalisation was elevated among the individuals who survived. The death rate rises with advancing age, and delayed presentation and commencement of therapy heighten the risk of fatality.

**Keywords:** Peptic ulcer, Perforation at, Gastroduodenal, Helicobacter pylori

This is an open access journal, and articles are distributed under the terms of the Creative Commons Attribution- Non Commercial- Share Alike 4.0 License, which allows others to remix, tweak, and build upon the work non-commercially, as long as appropriate credit is given and the new creations are licensed under the identical terms.

### INTRODUCTION

Gastroduodenal perforation are third in frequency after acute appendicitis and acute intestinal obstruction among abdominal emergencies. Peptic ulcers were earlier believed to be caused by stress, dietary factors, and increased gastric acid secretion till as late as 1983, when Warren and Marshall identified the correlation between Helicobacter pylori infection and peptic ulcers. It is now well established that most peptic ulcers occur as a result of H. pylori infection. Approximately 50% of the world population is infected with H. pylori. Prevalence varies with geography, age, race, ethnicity, socioeconomic status and is seen to decrease with improved hygiene. In

developing countries, H. pylori infection is usually acquired during childhood, with infection rates ranging from 13.4% to 24%. There have been recent reports of declining H. pylori infections in developed countries[1,2]. The pattern of perforated PUD is said to vary from one geographical area to another, depending on some socio-demographic and perhaps environmental factors. In a developing country such as ours, the patients presenting with perforated PUD are young with a dominant male preponderance. This is in contrast to the developed countries where the patient population with perforated PUD are mainly the elderly with less pronounced incidence differences between sexes. It is probable that the very strong

association with smoking and alcohol among the young male population may account for the high incidence in developing countries. Certainly in the West the high incidence is due to ulcerogenic drug ingestion amongst the elderly population. It is also noted that in the developing countries, the patients with perforated PUD, present late to definitive management centres[3,4]. Many patients first sought medical assistance from traditional healers and unauthorized medical personnel prevalent in developing countries.

Though a lot of work has been done on aetiology of this condition, one specific aetiology after cause can be incriminated in causation of this particular disease especially in our part of the country. Differences in the clinical presentation of gastroduodenal perforations vary from the typical severe acute abdominal pain at one end, to subtle or no symptoms in the hospitalized patients for unrelated illness at the other end. The various atypical presentations that mimic other abdominal conditions throw a real challenge over the diagnosis to the emergency surgeon. *Helicobacter pylori* is a gram -ve helical or curved bacillus. It is about 3 microns long and 0.5 microns in diameter. It is a fastidious, microaerophilic flagellate that has 4 to 6 lophotrichous flagella which are composed of two types of Flagellins[5]. It contains the enzyme 'Hydrogenase' which oxidizes Hydrogen molecules produced by intestinal bacteria[6]. It is also capable of forming Biofilms. It also has the capability to change into a non-culturable coccoid form to offer survival advantage during adverse conditions[7]. A careful medical history, methodical clinical examination and radiological study play a major role in the early diagnosis of this acute abdominal emergency. There are multiple factors that influence the prognosis and outcome of the patient. Preoperative resuscitation, intravenous administration of broad-spectrum antibiotics and good postoperative care are the mainstay in the management of Gastro duodenal Perforations[8,9]. The operative management depends upon the cause of perforations. The mortality rate has been reduced nowadays due to medical attention, quick diagnosis and prompt surgical management. But no significant method of treatment is appropriate for every patient with perforated gastroduodenal ulcer.

The study was conducted with the aim of clinico pathological and microbiological correlation of peptic ulcer perforation in B.R.D Medical College, Gorakhpur and have given the recommendation of ethical clearance

## MATERIAL AND METHODS

An analytical investigation was undertaken at B.R.D Medical College, Gorakhpur to establish a link between the clinical and pathological aspects of peptic ulcer perforation. The study also included a microbiological analysis. Based on the findings, ethical clearance was recommended. In this investigation, the patients were categorised into two categories. This research comprised patients from group 1 who had perforation peritonitis caused by peptic ulcer perforation. The patients included in group 2 had an endoscopic diagnosis of peptic ulcer disease.

## METHODOLOGY

The patient was segregated into two cohorts. Group 1 consists of patients aged 15 years and above who have had surgery for perforated gastric and peptic ulcers. Group 2 consists of individuals who are above 18 years old and have been diagnosed with peptic ulcer disorders by endoscopy. In group 1, two mucosal biopsies were obtained from the perforated site. One biopsy was collected in normal saline for culture, while the other was placed in formalin solution for pathological investigation. Both biopsies were transferred to the microbiological and pathological departments for study. In group 2, after endoscopic identification of peptic ulcer disorders, a mucosal biopsy was obtained from the antrum and submitted for microbiological analysis to perform the Rapid Urease Test. The biopsies from both groups will undergo histological evaluation to determine the presence of *H.pylori*. If the aforesaid test yields positive results, patients will be classified as *h.pylori* positive. The research excluded patients with malignant gastric and duodenal perforation, sealed peptic ulcer perforation who were treated non-surgically, and perforated peptic ulcer in patients under the age of 18.

## RESULTS

The mean age in pre pyloric gastric perforation was  $51.96 \pm 18.27$  and pyloric ulcer perforation was  $42.25 \pm 6.34$ .

**Table 1: Comparisons of age in between Prepyloric and Pyloric**

	Prepyloric (n=26)		Pyloric (n=4)	
	Mean	±SD	Mean	±SD
Age (years)	51.96	18.27	42.25	6.34

Out of 30 patients operated for Peptic ulcer perforation in this study, On histopathological examination were found to be non specific chronic inflammatory lesions.

**Table 2: Distribution of study population according to histopathology**

NSCIL	Number (n)	Percentage (%)
Yes	30	100
No	0	0

**Table 3: Comparisons of Rapid Urease Test and IgG test in between Prepyloric and Pyloric**

H. pylori		Prepyloric (n=26)		Pyloric (n=4)	
		n	%	n	%
Rapid Urease Test	Positive	17	68.00	4	100.00
	negative	9	36.00	0	0.00
IgG	Positive	14	56.00	3	75.00
	negative	12	48.00	1	25.00

In this study, the other group of people who had upper GI endoscopy for dyspepsia about half of the population were found to have gastritis, the other half were non gastritis which include neoplastic growth, ectopic pancreatic tissue and other causes.

**Table 4: Comparisons of age in between Gastritis and non-Gastritis**

	Gastritis (n=15)		Non-Gastritis (n=15)		t	p-Value
	Mean	±SD	Mean	±SD		
Age (years)	49.60	16.51	58.40	12.32	-1.65	0.109

Gastritis is more common in male, this may be because of increased exposure to alcohol, smoking and irregular food habits, etc. Non gastritis are more in females.

**Table 5: Comparisons of gender in between Gastritis and non-Gastritis**

Gender	Gastritis (n=15)		Non-Gastritis (n=15)		Chi-Sq.	p-Value
	n	%	n	%		
Male	10	66.67	6	40.00	1.21	0.272
Female	5	33.33	9	60.00		

**Table 6: Comparisons of Rapid Urease Test in between Gastritis and non-Gastritis**

Rapid Urease Test	Gastritis (n=15)		Non-Gastritis (n=15)		Chi-Sq.	p-Value
	n	%	n	%		
Positive	14	93.33	2	13.33	16.21	<0.001*
Negative	1	6.67	13	86.67		

The Sensitivity, specificity, positive productive value (PPV) and negative productive value (NPV) of diagnosing Gastritis by Rapid Urease Test are shown in Table 6. The positive Rapid Urease Test had more sensitivity of 87.5%, specificity of 92.9%, PPV of 93.3% and NPV of 86.7% in the diagnosis of Gastritis. These tests were demonstrating the accuracy of risk factors.

**Table 7: Sensitivity, specificity positive productive value (PPV) and negative productive value (NPV) of diagnosing of Gastritis by Rapid Urease Test**

Test	Sensitivity	Specificity	PPV	NPV
Rapid Urease Test	87.5%	92.9%	93.3%	86.7%

Comparison of rapid urease test in perforated peptic ulcer diseases and Non perforated peptic ulcer disease following result can be commented on the test, sensitivity 70% specificity 46.67% positive predictive value 56.67% negative predictive value 60.87% and accuracy 58.33%.

**Table.8 Rapid urease test in perforated peptic ulcer diseases and Non perforated peptic ulcer disease**

Rapid urease test	Perforated peptic ulcer	Non perforated peptic ulcer
POSITIVE	21	16
NEGATIVE	9	14

## DISCUSSION

Peptic ulceration is likely caused by an imbalance between the aggressive effects of acid pepsin production and the natural defences of the gastroduodenal mucosa. The primary contributing factor for duodenal ulcer is believed to be the excessive exposure of the duodenal mucosa to acid and pepsin. The primary contributing factor for gastric ulcers seems to be a disruption in the protective mechanisms of the stomach mucosa against acid and pepsin. The hypersecretion is associated with an unusually high overall mass of parietal cells in the

stomach mucosa, perhaps due to either heightened sensitivity of the parietal cells to secretory cues or the absence of normal regulating mechanisms[10]. Gastrointestinal (GI) perforations have been longstanding surgical challenges. Gastrointestinal perforation may manifest at any point along the digestive tract, ranging from the oesophagus to the rectum. Peptic perforation is the primary cause of gastrointestinal (GI) perforation in our nation. Perforated peptic ulcer (PPU) is a frequently occurring surgical emergency in India[11]. The occurrence of hospital hospitalisations and elective

procedures for acid peptic illness has declined in recent years due to the introduction of very effective medical treatment. Nevertheless, the prevalence of PPU remains elevated. Age, gender, and the presence of other illnesses are variables that increase the risk of disease and death in PPU. Preoperative shock, a lag time beyond 24 hours, and the extent of the hole are all variables that increase the risk of postoperative morbidity. Lower digestive system perforations often exhibit mixed contamination, including both aerobes and anaerobes[12].

The distal section of the stomach, namely the prepyloric and pyloric regions, is the most frequent location of perforation in this research. This finding is consistent with a research undertaken by Thirupathaiah et al [13], where the pre pyloric region

was identified as the most frequent location of perforation. The variation in the location of perforation seen in the literature may be partially ascribed to the diverse populations that were researched.

This research establishes a substantial correlation between H.pylori infection and perforated peptic ulcer illness, which is in direct contradiction to the findings of Reinbach et al [14], who claimed the absence of any such connection. According to a research conducted by Kumar et al [15], the fast urease test was shown to be the most sensitive approach for detecting H.pylori, compared to the quick urease test, histology, and culture method. The following research have shown a substantial correlation between H.pylori infection and perforated peptic ulcer.

**Table 9 Prevalence of H.pylori infection in previous study**

Study	Year	Prevalence of H.pylori infection %	Association
Ugochukwu et al [16]	2013	65-70	Significant
Dogra et al [17]	2014	92	Significant
John B et al [18]	2017	47	Significant
Sebastian et al [19]	2001	83.3	Significant

## CONCLUSION

The current investigation has shown that perforated peptic ulcer disease (PUD) is a very dangerous condition that has significant risks of both morbidity and mortality. The prevalent symptoms were stomach pain and distension. The rate of hospitalisation was elevated among the individuals who survived. The death rate rises with advancing age, and delayed presentation and commencement of therapy heighten the risk of fatality.

## REFERENCES

- Chung KT, Shelat VG. Perforated peptic ulcer - an update. *World J Gastrointest Surg.* 2017 Jan 27;9(1):1-12. doi: 10.4240/wjgs.v9.i1.1, PMID 28138363, PMCID PMC5237817.
- Gowda DB, Kadambari D, Vijayakumar C, Elamurugan TP, Jagdish S. A clinicomicrobiological profile in patients with perforated peptic ulcer with special reference to anaerobic organisms: a descriptive study. *Int Surg J.* 2017;4:125-30.
- Shreya A, Sahla S, Gurushankari B, Shivakumar M, Rifai KV, Kate V et al. Spectrum of perforated peptic ulcer disease in a tertiary care hospital in South India: predictors of morbidity and mortality. *ANZ J Surg.* 2023 Dec 19. doi: 10.1111/ans.18831 [Epub ahead of print]. PMID 38115644.
- Isselbacher J, Kunt et al. Peptic ulcer, Harrison's Principle of Internal Medicine. 14th ed. Vol. 284; 1596-1609.
- Merich DW et al.: Stomach, Sabiston Textbook of Surgery. 17th ed. 2005;99:1265-321.
- Yogeshwar D et al. The gastrointestinal tract, Robbin's Pathologic Basis of Disease. 4th ed; 1989. p. 827-910.
- Hermansson M, Staël von Holstein C, Zilling T. Surgical approach and prognostic factors after peptic ulcer perforation. *Eur J Surg.* 1999;165(6):566-72. doi: 10.1080/110241599750006479, PMID 10433141.
- David J et al. Duodenal ulcer and peptic ulcer perforation, Maingot,s Abdominal operations. 10th ed; 1997. P. 941-70.
- Palmer KR et al. Diseases of the alimentary tract and pancreas, Davidson,s Principles and Practice of Medicine. 18th ed; 1999. p. 599-682.
- Ali AM, Mohamed AN, Mohamed YG, Keleşoğlu Sİ. Clinical presentation and surgical management of perforated peptic ulcer in a tertiary hospital in Mogadishu, Somalia: a 5-year retrospective study. *World J Emerg Surg.* 2022 May 16;17(1):23. doi: 10.1186/s13017-022-00428-w, PMID 35578285; PMCID.
- Stabile BE, Passaro E. Duodenal ulcer: a disease in evolution. *Curr Probl Surg.* 1984;21(1):1-79. doi: 10.1016/0011-3840(84)90037-6. PMID 6317293.
- Türkdoğan MK, Hekim H, Tuncer I, Aksoy H. The epidemiological and endoscopic aspects of peptic ulcer disease in Van region. *East J Med.* 1999;4(1):6-9.
- Thirupathaiah K, Jayapal L, Amaranathan A, Vijayakumar C, Goneppanavar M, Nelamangala Ramakrishnaiah VPN. The Association Between Helicobacter Pylori and Perforated Gastroduodenal Ulcer. *Cureus.* doi: 10.7759/cureus.7406.
- Reinbach DH, Cruickshank G, McColl KE. Acute perforated duodenal ulcer is not associated with Helicobacter pylori infection. *Gut.* 1993;34(10):1344-7. doi: 10.1136/gut.34.10.1344, PMID 8244099.
- Kumar D, Sinha AN. Helicobacter pylori infection delays ulcer healing in patients operated on for perforated duodenal ulcer. *Indian J Gastroenterol.* 2002;21(1):19-22. PMID 11871831.
- Ugochukwu AI, Amu OC, Nzegwu MA, Dilibe UC. Acute perforated peptic ulcer: on clinical experience in an urban tertiary hospital in south east Nigeria. *Int J Surg.* 2013;11(3):223-7. doi: 10.1016/j.ijssu.2013.01.015, PMID 23403213.
- Dogra BB, Panchabhai S, Rejinthal S, Kalyan S, Priyadarshi S, Kandari A. Helicobacter pylori in gastroduodenal perforation. *Med J DY Patil Univ.*

- 2014;7(2). doi: 10.4103/0975-2870.126331.
18. John B, Mathew BP, Chandran VP. Prevalence of Helicobacter pylori in peptic ulcer perforation. *Int Surg J.* 2017;4(10):3350-3. doi: 10.18203/2349-2902.isj20174494.
19. Sebastian M, Chandran VP, Elashal YI, Sim AJ. Helicobacter pylori infection in perforated peptic ulcer disease. *Br J Surg.* 1995;82(3):360-2. doi: 10.1002/bjs.1800820325, PMID 7796009.