Section: Surgery

Original Research Article

PREVALENCE OF HELICOBACTER PYLORI IN PEPTIC ULCER PERFORATION IN MAHARASHTRA POPULATION

Shivaj Thakare[1], Srinivasrao G Shinde[2], Panchalingappa Betageri[3]

[1]Consultant Gastroenterologist Satkrupa Hospital Akola, Maharashtra, India
[2]Assistant Professor, Department of Paediatrics, ESIC Medical College and Hospital Kalaburgi, Karnataka, India
[3]Assistant Professor, Department of Medical Gastroenterology, Kles Dr Prabhakar Kore Hospital and Medical Research Centre, Nehru Nagar, Belgaum, Karnataka, India

Received : 14/09/2023
Received in revised form : 09/10/2023
Accepted : 20/10/2023

Keywords:
Giemsa staining, histopathology, laparotomy, Helicobacter pylori.

Corresponding Author:
Dr. Panchalingappa Betageri,
Email: praveenbetageri@gmail.com

DOI: 10.47009/jamp.2023.5.5.261

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

Int J Acad Med Pharm
2023; 5 (5); 1321-1323

Abstract

Background: Peptic ulcer perforation is quite common due to stress, a strained life, and an irregular diet. H. pylori infection causes inflammation, disrupts the inhibitory control of the gastric, and leads to PPU. **Materials and Methods:** 125 patients with peptic ulcer perforation underwent resuscitation, and a laparotomy was performed in the emergency department of surgery. H. pylori infection was confirmed by histo-pathological examination by Giemsa staining, and H. pylori infection was treated accordingly. **Result:** The distribution of age, site of perforation by H. pylori infection was insignificant, but associated factors smokers, type II DM and tobacco chewers had a significant p value ($p<0.001$). **Conclusion:** It is concluded that the majority of the perforation of the peptic ulcer was due to H. pylori inaction. Hence, every patient undergoing laparotomy for PUP must be investigated for H. pylori infection if a positive H. pylori treatment can be tried to prevent further perforation or limit the perforation.

INTRODUCTION

The word “peptic” is derived from the Greek term "peptikos,” meaning related to digestion. Peptic ulcer perforation was noticed in King Charles I's daughter. Henriette Anne died suddenly in 1670 (at 26 years of age) after a day of abdominal pain and tenderness. Since poisoning was suspected, an autopsy was performed, revealing peritonitis and a small hole in the anterior wall of the stomach. However, the doctors had never heard of perforated peptic ulcers (PPU).[1,2]

The pathogenesis of peptic ulcer disease may best be considered a complex scenario involving an imbalance between the defensive (mucus-bicarbonate) layer, prostaglandins, cellular renovation, and blood flow, and aggressive factors (hydrochloric acid, pepsin, ethanol, bile salt, and some medications).[3] In recent years, Helicobacter pylori infection and NSAID have been identified as the two main causes of peptic ulcers. The use of crack cocaine has also increased in PPU.

Pylori infection causes gastric ulcers and is associated with mortality resulting from haemorrhage, perforation, and obstruction. Hence, the need for surgery for PPU has remained stable or even increased,[4] because the root cause of gastric ulcers is stress, strained life, and junk food; therefore, young adults are more prone to PPU. Hence, an attempt was made to evaluate PPU in different age groups and associated risk factors.

MATERIALS AND METHODS

125 patients admitted to the emergency general surgery department of Sant Krupa Hospital in Akola, Maharashtra, were studied.

Inclusive Criteria

The patient was clinically diagnosed with peptic ulcer perforation. Those who were diagnosed with peptic ulcer perforation, both gastric and duodenal, and gave their consent in writing for surgery were included in the study.

Exclusion Criteria

The patients who had malignancy in the G.I.T. were excluded from the study.

Method: The diagnosis of peptic ulcer perforation was made by history of patients clinical examination, and radiological investigations confirmed at laparotomy.

The investigation included routine blood examinations (CBC, urine analysis, blood urea, serum creatinine, serum electrolytes, radiological examination, ECG, blood grouping, x-ray chest, and USG abdominal). A special attempt was made to look into various precipitating factors that led to

ISSN (O): 2687-5365; ISSN (P): 2753-6556

perforations. A biopsy was taken from the peptic ulcer perforation site with intact mucosa and sent to a histopathology test to detect the organism by Giemsa staining. The perforation was closed and reinforced with an omental patch. Based on the histopathological report, the prevalence of H. pylori infection was treated with proton pump inhibitors and suitable antibiotics.

The duration of the study was from November 2019 to December 2020.

Statistical Analysis: distribution of age and site of perforation-associated infections were analysed. Statistically, by chi-square and z tests, significant results were noted. The statistical analysis was carried out in SPSS software. The ratio of males and females was 2:1.

RESULTS

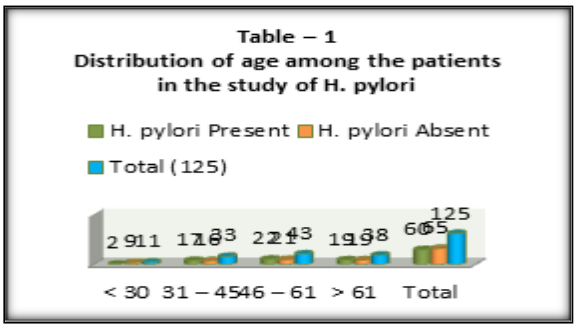


Table 1: Distribution of age among the patients in the study of H. pylori. (Total No. of Patients: 125)

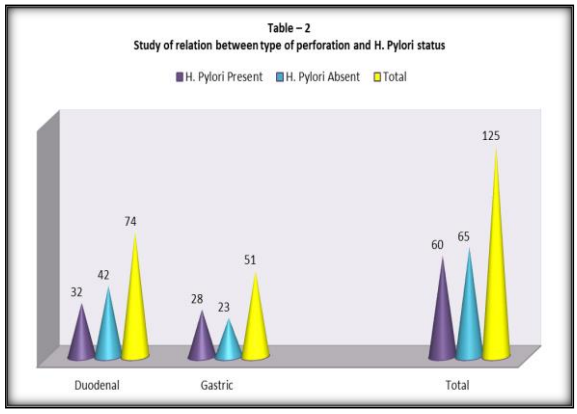
Age	H. pylori Present	H. pylori Absent	Total (125)	Statistical value
< 30	2 (1.6%)	9 (7.2%)	11 (8.8%)	Chi-square test=4.35 DF=3 p>0.05
31 – 45	17 (13.6%)	16 (12.8%)	33 (26.4%)	
46 – 61	22 (17.6%)	21 (16.8%)	43 (34.4%)	
> 61	19 (15.2%)	19 (15.2%)	38 (30.4%)	
Total	60 (48%)	65 (52%)	125 (100%)	

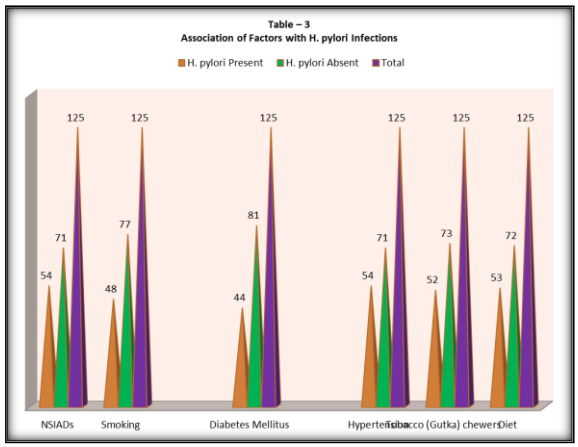
[Table 1]: The distribution of age among the patients in the study was H. pylori. Theage of the patient was <30 to >60 years of age. The chi-square test was 4.35, DF-3, and p > 0.05 (the p value was insignificant).

Table 2: Study of relation between type of perforation and H. Pylori status. (Total No. of Patients: 125)

Site of perforation	H. Pylori Present	H. Pylori Absent	Total	Statistical value
Duodenal	32	42	74	Z=1.28, P>0.05
Gastric	28	23	51	Z=1.28,P>0.05
Total	60	65	125	

[Table 2]: In the study of the relation between type of perforation and H. pylori status, the p value was





insignificant in sites, i.e., duodenal gastric (p > 0.005).

Table 3: Association of Factors with H. pylori Infections. (No. of patients: 125)

Factors	H. pylori Present	H. pylori Absent	Total	Statistical value
NSIADs	54 (43.2%)	71 (56.8%)	125	Z=1.52, P>0.05
Smoking	48 (38.4%)	77 (61.6%)	125	Z=2.59,* P<0.01
Diabetes Mellitus	44 (35.2%)	81 (64.8%)	125	Z=3.30,* P<0.01
Hypertension	54 (43.2%)	71 (56.8%)	125	Z=1.52, P>0.05
Tobacco (Gutka) chewers	52 (41.6%)	73 (58.4%)	125	Z=1.87,*P<0.05
Diet	Regular 53 (42.4%)	Irregular 72 (57.6%)	125	Z=1.69 P<0.05

[Table 3]: In the study of the association of factors smoking type II DM, tobacco chewers had significant factors of H. pyloric perforations (p<0.005).

ISSN (O): 2687-5365; ISSN (P): 2753-6556

DISCUSSION

Present study of the prevalence of H. pylori in peptic ulcer perforation (PUP) in the Maharashtra population. In the distribution of age of the patients in the study, if H. pylori, the age ranged from < 30 years of age to > 60 years of age, the statistical analysis was insignificant ($p > 0.05$). Hence, there is no association between age and H. pylori infection [Table 1]. In the present study, the relationship between site and types of perforation is examined. It was also statistically insignificant ($p > 0.05$) that PUP can occur in duodenal or gastric (either gastric or duodenal) [Table 2]. In the study of co-factors with H. pylori infection smoking, type-II DM and tobacco chewers had a significant p value ($p<0.005$) (Table 3). These findings are more or less in agreement with previous studies.[5-7]

PUP (peptic ulcer perforation) is one of the major surgical emergencies. The optional surgical treatment for duodenal ulcers has been controversial because of their relapse. Hence, simple repair followed by H. pylori eradication therapy for positive cases of H. pylori infection Treatment for H. pylori was more judicious and ideal therapy because H. pylori infection can be held responsible for more than 90% of duodenal ulcers and up to 80% of gastric ulcers.[8] It is reported that H. pylori seems to be acquired in early childhood. In contrast to many other infections, the immune system does not contribute to healing. Another problem with eradicating H. pylori is that it is not only located on the surface of the gastric mucosa but also in the layer of mucous protecting it. It is treated with triple therapy proton pump inhibitor (PPI) clarithromycin plus amoxycillin or metronidiazole because monotherapy of antibiotics was unsuccessful in treating H. pylori infection.[9] Traditionally, peptic ulcers are diagnosed endoscopically, but this is an expensive tool that is well tolerated by patients. the preferred method to diagnose H. pylori by taking perioperative biopsies but to confirm gastric cancer endoscopy is mandatory because PPU can be a symptom of gastric cancer.[10]

Since 1990, laparoscopic closure of PPU has been described. It has a minimally invasive diagnostic tool,[11] the benefits are post-operative pain reduction, less consumption of analgesics, a reduction in hospital stay, and a reduction in wound infections. In rare cases, burst abdomens and incisional hernias due to shorter scars have been noted.

Limitation of Study: Due to tertiary location of research centre, small number of patients and lack of latest techniques, we have limited findings and results.

CONCLUSION

Laparoscopic surgery is a safe and less invasive tool to treat PUP. To prevent recurrence, complete eradication of H. pylori inflection has to be treated along with the healing of post-operative wounds. Changes in lifestyle, diet, smoking, chewing tobacco, and control of type II DM can prevent PUP. Moreover, the present study demands pathophysiological, nutritional, environmental, and genetic studies because the exact pathogeneses of PUP are still unclear.

REFERENCES

1. Baran JH – Paintress, princess, and physician's paramour: poison or perforation? JR Soc. Med. 1998, 91; 213-216.
2. Baran JH – Peptic Ulcer, Sinai J Med. 2000, 67; 58–62.
3. Baron JH, Sonnenberg A – Publication on Peptic Ulcer in Britain, France, Germany, and the US Eur. J. Gastroenterol Hepatol 2002, 14; 711–715.
4. Zittel TT, Jehle EC – Surgical management of peptic ulcer disease today: indication, technique, and outcome Langenbecks Arch. Surg. 2000, 385; 84–96.
5. Gossens H, Glupezynski Y- Evaluation of commercially available second-generation immune globin Genzymes immune assay for detection of H. pylori infection J. Clin. Microbiol. 1992; 30: 176–80.
6. Soreide K, Thorsen K – Perforated peptic ulcer, Lancet 2015, 386; 1288–98.
7. Dogra BB, Panchabai S – Helicobacter pylori in gastro-duodenal perforation 2014, 7 (2); 170-2.
8. Ahmed N – 23 years of the discollery of Helicobacter pylori, is the debate over? Ann. Clin. Microbiol. Antimicrobol. 2005, 4; 17–19.
9. Malfertheiner P, Mergraud F – Current concepts in the management of Helicobacter pylori infection; Gut. 2007, 56; 770–781.
10. Sivri B – Trends in Peptic Ulcer Pharmacotherapy, Fundom Clinic Pharmacol 2004, 18: 23–31
11. Pai D, Sharma A, Jagdish Gupta A – role of abdominal drains in perforated duodenal ulcer patients Aust. Nz J. Surg. 1999, 69; 210–213.

ISSN (O): 2687-5365; ISSN (P): 2753-6556