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ORIGINAL ARTICLE

Pervasiveness of helicobacter pylori infection in perforated duodenalulcer complication

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ABSTRACT:

Introduction: The aim of this study is to determine the H. Pylori status of patients with PPD so that decision makers will use the information for priority setting and make decisions based on existing evidence. **Materials and Methods:** This Retrospective observational study was conducted in 70 cases of DU perforation with peritonitis who presented to the casualty. All the charts of the patients with perforation peritonitis were analyzed from the medical record department for operative notes. Patient details like age, sex, detailed history with emphasis on previous history of acid peptic disease, past medical history, medical treatment for acid peptic disease and physical examination were recorded. Emergency laparotomy was done in all cases of DU perforation. **Results:** One minuterapid urease test was positive in a total of 45 patients (64.2%) and majority of the cases reported was in the fifth decade of the life followed by fourth decade group. The distribution of cases among gender is shown in table no.2 depicting majority of them being male 84% of cases. **Conclusion:** The present study shows that the risk of developing H. pylori related duodenal ulcer perforation is strongly increased in those patients where the organism is detected by rapid urease test in the duodenum.

Keywords: Duodenal ulcer, perforation, helicobacter pylori

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INTRODUCTION

Worldwide the incidence of peptic ulcer disease is said to have fallen in recent years.[1] However, the incidence of perforated duodenal ulcers has either remained constant or has been increasing due to which there has been increase in the incidence of emergency surgery.

Perforation of the duodenal ulcer is commonly seen in the first part of duodenum and occurs in about 5% to10% of patients with activeulcer disease.[1] Majorityof the patients require surgery except for a very few number of patients who have sealed off perforation. Mortality in perforated DU is around 15% as per literature review. Helicobacter pylori (H. pylori) is a gram-negative bacterium that infects almost 50% of people in developed nations and up to 80% indeveloping countries.[2] Prevalence of Helicobacter pylori is one of the risk factors for peptic ulcer disease.

The average prevalence ofH. pyloriinfection in patients with perforated peptic ulcer is of only about 65–70%, which contrasts with the almost 90–100%. There are many previousstudies showing prevalence of H.pylori in duodenal ulcer perforation patients but none are available in the recent years.[3] Peptic ulcer disease continues to be a significant health and economic burden in low and middle- income countries. Eradication of H. pylori after surgery has been proved to reduce recurrence rate and complications.[4]

H.pylon infection of the gastric antral mucosa plays an important role in the development of duodenal ulcer disease' and is of particular importance in perforation.[5] According to the currently followed model of the pathogenesis of H. pylori related duodenal ulcer, colonization of duodenum by h. pylori is the critical final step responsible for the chain of events leading to the lesion.[6]

Thus, presence of duodenal colonization might represent a very high-risk condition for the development of duodenal ulcer and subsequent perforation. This hypothesis has never been tested until now. This may bedue to the fact that a reliable identification of H. pylori in the duodenum is considered difficult, as a low number of bacteria colonizes cattered are as of duodenal gastric metaplasia.[6] Several methods may be used to diagnose H. Pylori infection. These tests are: serological tests, urea breath test, endoscopy with biopsy (biopsy urease test, histologic identifications of organism, culture of biopsy specimen.

Studies in India on the association of H. Pylori with uncomplicated PUD and outcomes on complicated PUD showed similar results as in other LMIC. In our Hospital, PPD is among the commonest surgical emergencies that require immediate surgical intervention. However there are no studies on status of H. Pylori infection on PPD.

The aim of this study is to determine the H. Pylori status of patients with PPD so that decision makers will use the information for priority setting and make decisions based on existing evidence.

MATERIALS AND METHODS

This Retrospective observational study was conducted in 70 cases of DU perforation with peritonitis who presented to the casualty.

Exclusion Criteria: Patients with gastric ulcer perforation in prepyloric region or perforation in any other part of bowel were excluded from the study

The study was approved by the institutional human ethics committee. Informed written consent was obtained from all the study participants. All the charts of the patients with perforation peritonitis were analyzed from the medical record department for operative notes. Case notes were identified based on diagnosis as well as surgical procedure done. Patients with perforated duodenal ulcer were selected and included in the study.

Patient details like age, sex, detailed history with emphasis on previous history of acid peptic disease, past medical history, medical treatment for acid peptic disease and physical examination werer ecorded. Plain X ray of the chest and abdomen (erect) and ultra- sonogram of the abdomen findings were noted down into a proforma. Emergency laparotomy was done in all cases of DU perforation.

One minute Rapid Urease test of Arvind et al was done on table in all cases and a specimen was obtained for Histo-pathological examination from the mucosa at the perforation site. The test was performed by adding two drops of 1% phenol red to two ml of freshly prepared 10% w/v urea in deionised water in a test tube at pH6.8.One minute rapid urease test detects the presence of preformed urea produced by H. Pylori in the specimen. The urease present hydrolyses urea in the solution with the production of ammonium ions which raises the ph. This pH change is detected by a pH indicator, phenol red which changes color from yellow at pH 6.8 to pink at pH 8. A color change from yellowto pink at the end of 1 minute is considered a positive reaction. A buffer is present in the medium to increase its stability and reduce false positive reaction. Histopathological staining and modified Giemsa staining was done later from the tissue excised.

RESULTS

As shown in Table No.1 One minute rapid urease test was positive in a total of 45 patients (64.2%) and majority of the cases reported was in the fifth decade of the life followed by fourth decade group. The distribution of cases among gender is shown in table no.2 depicting majority of them being male 84% of cases.

Table No 1: Age wise distribution of DU perforation cases.

	Rapid Urease test		
Age	**Positive**	**Negative**	**Total**
21-30	06	NIL	06
31-40	10	NIL	10
41-50	14	12	26
51-60	05	09	14
61-70	06	02	08
71-80	02	02	04
81-90	02	NIL	02
	45	25	70

It was found that all the 45 cases positive by rapid urease were also positive For H. pylori by histopathological analysis. None of the cases showed any intestinal metaplasia and 25 urease test negative were histopathologically negative too. Thus the sensitivity and specificity of rapid urease test was found to be 100%. 52 patients underwent laparotomy and perforation closure with live omental pedicle patch and five patients were treated with simple closure of the perforation with omental patch. Post operatively the rapid urease positive patients were treated with H.pylori regimens and all the patients responded welland during the follow up period of about 1 year there was no recurrence.

Table No 2: Sex wise distribution of DU perforation for rapid urease test.

	Rapid urease test		
Sex	**Positive**	**Negative**	**Total**
Male	35	17	52
Female	10	08	18
	45	25	70

DISCUSSION

Association of H. pylori and duodenal ulcer perforation has been reported to be as high as 92%, only few studies have evaluated the prevalence of H. pylori in patients suffering from perforated peptic ulcer. There are studies which suggest that other pathogenic factors other than H. pylori are associated with duodenal ulcer perforation.[7]

In our study also about fifteen patients (30%) were tested Rapid urease test negative. The incidence of duodenal ulcer perforation is more in the males (80%) and the incidence of duodenal ulcer perforation is maximum in the 31-50 age group. Ulcer perforation incidence has been studied over an extended period in western Scotland, United Kingdom and Norway who have suggested similar trend of increased incidence rate among males.[8] In men, ulcer perforation increased until about 1950 and declined thereafter. In women the incidence was low and fairly stable until about 1950 from which time it increased. Increasing age among ulcer perforation patients has been observed during this period with declining incidence among the

young and increasing incidence among the elderly.[9] There are conflicting data with regard to association of PPD to H. Pylori infection. Several studies showed socioeconomic status, prevalence of H. Pylori, smoking habits and alcohol are associated with or influence PPU rate.[10] On the other hand, there was no significant association between the incidence of H. Pylori infection in peptic ulcers with smoking and NSAIDS intake. To make these associations even more complex in one study it was found that PPD was associated with H. Pylori but not NSAID use.[11] Unfortunately, in our study, there was incomplete documentation of risk factors in majority of patients and it was impossible to determine association.

Perforated peptic ulcer disease is a surgical emergency, patients usually present as an acute abdomen. In this study 95.7% patients were diagnosed to have peritonitis with varying percentages of specific symptoms of peritonitis; diffuse abdominal pain 93.5%, vomiting 82.6% and epigastric pain 65.2%, which is similar to studies in Korea, Nigeria and other studies in India. Intraoperatively, 93.5% patient had perforation on the first part of duodenum, which is similar to other African and Middle Eastern countries.

There were no cases which showed H. pylori by histopathology were negative for the rapid urease test. This suggests that specificity and sensitivity for rapid urease test approaches 100 whichis to the common observation. Marshall et al, Mc Nultyetal have reported sensitivity for biopsy urease test as 98% and 96%. respectively.[9] Intestinal metaplasia was not noticed in any if the rapid urease positive cases, however chronic inflammatory cells were demonstrated in all of them suggesting that association of H. pylori with long standing cases of acid peptic disease. Detection of H. pylori by rapid urease test and histopathology was equally efficient and accurate.

CONCLUSION

The prevalence of H. pylori in DU perforation is 70% and was seen more in those cases with a long duration of APD. The present study shows that the risk of developing H. pylori related duodenal ulcer perforation is strongly increased in those patients where the organism is detected by rapid urease test in the duodenum. This finding may help us to device strategies aimed at prevention of duodenal ulcer, the most common disease caused by H. pylori infection. The sensitivity and specificity of One minute rapid urease test is almost 100%

REFERENCES

1. Saber A. Perforated Duodenal Ulcer in High Risk Patients. InPeptic Ulcer Disease 2011. InTech.
2. Salmanroghani H, Mirvakili M, Baghbanian M et al. Efficacy and Tolerability of Two Quadruple Regimens: Bismuth, Omeprazole, Metronidazole with Amoxicillin or Tetracycline as First-Line Treatment for Eradication of Helicobacter Pylori in Patients with Duodenal Ulcer: A Randomized Clinical Trial. DOI:10.1371/journal. pone. 0197096.
3. Pounder RE, Ng D. The prevalence of Helico bacter pylori infection in different countries. Aliment Pharmacol Ther. 1995;9 Suppl 2:33-9.
4. Ng EK, Chung SC, Sung JJ, et al. High prevalence of Helicobacter pylori infection in duodenal ulcer perforations not caused by non-steroidal anti-inflammatory drugs. Br J Surg. 1996 Dec;83(12):1779-81.
5. Sebastian M, Chandran VP, Elashaal YI, Sim AJ. Helicobacter pylori infection in perforated peptic ulcer disease. Br J Surg. 1995 Mar;82(3):360-2.
6. Pietroiusti A, Luzzi I, Gomez MJ, et al. Helicobacter pylori duodenal colonization is a strong risk factor for the development of duodenal ulcer. DOI:10.1111/j. 1365-2036.2005.02423.x
7. Gisbert JP, Pajares JM. Helicobacter pylori infection and perforated peptic ulcer prevalence of the infection and role of antimicrobial treatment. Helicobacter. 2003 Jun; 8(3):159-67.
8. Svanes C. Trends in perforated peptic ulcer: incidence, etiology, treatment, and prognosis. World J Surg. 2000 Mar;24(3):277-83.
9. Svanes C, Lie RT, Kvåle G, et al. Incidence of perforated ulcer in western Norway, 1935-1990: cohort- or period-dependent time trends? Am J Epidemiol. 1995 May 1;141(9):836-44.
10. Gisbert JP, Pajares JM. Helicobacter pylori infection and perforated peptic ulcer prevalence of the infection and role of antimicrobial treatment. Helicobacter. 2003;8:3159–3167.
11. Metzger J, Styger S, Sieber C, Vogelbach P, Harder F. Prevalence of Helicobacter pylori infection in peptic ulcer perforations. Swiss Med Wkly. 2001;24:99–103.