FREQUENCY OF HELICOBACTER PYLORI IN PATIENTS PRESENTED WITH PERFORATED PEPTIC ULCER

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ABSTRACT

Objective: *To study the frequency of H-pylori infection in patients with perforated peptic ulcer disease (Duodenal and Gastric).*

Material and Methods: *During the study period a total of 85 patients were admitted with acute perforated peptic ulcer to surgical "C" unit Lady Reading Hospital, Peshawar from June 2004 to December 2005. They were operated and post operatively blood sample was taken for identification of antibodies against H-pylori by ELISA method. All patients irrespective of gender and age who were operated for perforated peptic ulcer were included in the study. Patient who gave history of intake of H2 receptor antagonist and Proton Pump Inhibitors up to six weeks prior to their presentation were excluded.*

Results: *Out of 85 patients studied and analyzed for antibodies against H-pylori, 77 (90.59%) were male and 8 (9.41%) were female. Age ranged from 30-75 years. ELISA showed that 56.46 % (n=48) were positive while 43.54 % (n=37) were negative for antibodies against H-pylori. 87% (n=54) patients gave history of chronic dyspepsia. 47 of these were positive for H-pylori. All patients were treated with eradication therapy which consisted of clarithromycin, metronidazole and omeprazole. Six weeks after initial surgery blood samples were analysed for H-Pylori and were found to be negative.*

Conclusion: *Patients who present with perforated peptic ulcer and gave history of chronic dyspepsia should be given eradication therapy post-operatively in order to reduce the incidence of recurrence.*

Key words: *h. Pylori, Perforated Peptic Ulcer, Chronic Dyspepsia.*

INTRODUCTION

Helicobacter pylori is a spiral gram negative rod that resides beneath the gastric mucosal layer adjacent to the gastric epithelial cells. It causes gastric mucosal inflammation with polymorphonuclear neutrophils and lymphocytes and results in ulceration**.** The mechanism of injury may be related in part to the products of two genes Vac A and Cag A.[1,2] The prevalence of H pylori varies amongst countries and population. It is low in developed countries and high in the developing countries.[3,4] There are several diagnostic methods for the detection of H pylori such as non-invasive serological tests which measures the specific anti-H pylori immunoglobulin IgG and or IgA using various antigens and serological tests.[5] Invasive (direct) methods for detection of H pylori in the gastric mucosa include bacterial culture, histological examination of biopsy specimen with different stains, and assays for urease activity. In search of the English literature of the last ten years few reports of association of H-pylori in patient with perforated peptic ulcer were found; high prevalence, however has been shown in association with gastric mucosal atrophy.[2]

The aim of this study was to know the presence of H pylori in patients with perforated peptic ulcer by laboratory based quantitative serologic ELISA test.

MATERIAL AND METHODS

This study was carried out in surgical "C" unit of Postgraduate Medical Institute, Lady Reading Hospital, Peshawar from June 2004 to December 2005. During this period a total of 85 patients were admitted with acute peptic ulcer perforation. All these patients were included in the study. Demographic data, medical history, past history of peptic ulcer disease or dyspepsia and use of NSAIDS were recorded. Patients between the age of 20 and 75 were admitted with

COMPARISON OF THE PATIENTS WITH PERFORATED PEPTIC ULCER FOUND TO BE H-PYLORI POSITIVE OR NEGATIVE

	H-Pylori Positive n=48 (56.46%)	H-Pylori Negative n=37 (43.54%)	Total (n=85)
Male	39 (54.46%)	32 (45.6%)	77
Female	6 (75%)	2 (25%)	8
Chronic dyspepsia	47 (87%)	7 (13%)	54
NSAIDS	1 (9.1%)	10 (90.9%)	11
No history of Chronic dyspepsia or NSAID use	0	20 (100%)	20

Table 1

perforated peptic ulcer irrespective of their gender were included in the study. Patients who either did not agree or who refused to give blood sample for H-pylori estimation were excluded from the study. Patients who gave history of taking acid reducing drugs (H_2 receptor antagonist or PPI) in the last six weeks were also excluded from the study. Patient who showed clinical evidence of septicemia, respiratory failure, congestive heart failure were excluded. Patient who were known diabetic who were taking steroid for some other illnesses, or immune suppressor drugs were not included in the study.

All these patients with acute peptic ulcer perforation were resuscitated and laprotomy was performed in the emergency department. Perforation was closed and re-enforced with an omental patch. Post operatively blood sample was taken from these patients and sent to the laboratory for detection of antibodies against H pylori.

All patients were given H pylori eradication therapy post operatively at the time of discharge, consisting of Clarithromycin 500mg BD for 7 days, Metronidazole 400mg TDS for 7 days and Omeprazole 20 mg OD for 14 days.

RESULTS

During the 18 months period a total number of 85 patients were included in the study. Their mean age was 45.8 years (Rang 30-75 years), seventy seven (95.59 %) patients were male while only 8 (9.41%) were female. Out of 85 patients, 75 patients (88.24%) had perforated duodenal ulcer while only 10 patients (11.76%) had perforated gastric ulcer. Fifty-four patients (63.54%) had history of chronic dyspepsia. Eleven (12.94%) patients had history of NSAIDS intake while 20 (23.52%) patients had no history of dyspepsia or NSAIDS ingestion. 51 (60%) patients were positive for H pylori while 34 (40%) were negative for H pylori.

Patient with chronic dyspepsia i.e. 47 out of 54 (87%) had high prevalence of H. pylori while patients with NSAIDS (4 out of 11) had low (37%) prevalence. In twenty patients no underlying risk factor could be demonstrated. All these patients were negative for H-pylori. Prevalence was very high (six out of 8) i.e. 75% in females while 54.54% male were positive for H pylori (Table No. 1). All patients recovered postoperatively and no mortality was reported. All patients were given eradication therapy in the form of Clarithromycin 500mg BD for 7 days, Metronidazole 400 mg TDS for 7 days and Omeprazole 20 mg OD for 14 days at the time of discharge. After six week positive patients were called to out patient and their blood sample was taken for H-pylori detection and were found to be negative.

DISCUSSION

H pylori play an important role in the causation of peptic ulcer disease. Although chronic H. pylori infection associated gastritis is present in 30-50% of the population, the majority of the patients are non symptomatic and suffer no sequel. H. pylori infection is strongly associated with peptic ulcer diseases, however only 15% of the people wit chronic infection develop a peptic ulcer disease. Chronic H. pylori gastritis is associated with a 2-4 fold increase risk of gastric adeno-carcinoma and low grade B cell gastric lymphoma. There is little evidence that chronic H. pylori associated gastritis is a cause of dyspeptic symptoms.[2] Studies have shown that eradication therapy not only helps in the prevention of recurrence but it also aids in healing[6]. Eradication of H-pylori has changed the natural history of peptic ulcer specially duodenal ulcer and has become the treatment of choice for duodenal ulcer patients.[7-9] However, its role in perforated duodenal ulcer has not been investigated and the results are conflicting.[10] Some studies have shown that H pylori eradication can prevent complications of peptic ulcer like bleeding and reperforation[11]. Data regarding prevalence of H-pylori infection is very conflicting and shows wide variation from

0-92%.[12-17] This wide variation can be due to the different population group studied in the past. For example Sebugtian et al[14] has reported infection rate of 83%. They studied a small group of young male patients in India who had perforated peptic ulcer. Mehmanli M et al[18] reported an infection rate of 88.8% in a teaching hospital in Istanbul, Turkey. In our study the infection rate was 56.46 %. This was comparable to Sherma et al[12] (61%) and Reinbock et al[15] Although treatment of patients with history of chronic dyspepsia should be individualized, a cost effective initial approach is to test for H. pylori and treat the infection if the test is positive. If the H. pylori test is negative empiric therapy with an acid suppressant or prokinetic agent is recommended.[19] In our study patients having history of dyspepsia had a high prevalence rate of 87% while patients on NSAIDs had a low infection rate of 9.1%. Ng EK et al[16] had the same infection rate of 80% in patients not on NSAIDs. While Aman al[20] had found no difference in H. pylori infection rate between NSAID users and non users. They had also high infection rate of 85%. In our study the infection rate was high i.e 75% in female as compared to the male i.e 54%. This is in contrast to a study in the same hospital by Aman et al.[20] Most of the studies for the detection of H. pylori have relied upon the gastric mucosal biopsies through endoscopy. Since mucosal transmission between patients undergoing endoscopy has been reported.[21] Preference has therefore given to non invasive blood testing for H. pylori detection especially in our setup, where endoscopic facilities in the emergency department are no available. In our study we used indirect methods (Identification H. pylori by ELISA), which has low specificity and sensitivity as compared to other methods, that might be the reason of low infection rate as compare to other studies.

CONCLUSION

This study shows that patients having history of chronic dyspepsia and presenting with perforated peptic ulcer are usually infected with helicobacter pylori and therefore it is recommended that these patients may be treated with eradication therapy.

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