ORIGINAL ARTICLE

Frequency of Helicobacter Pylori Infection in Causation of Duodenal Ulcer Perforation

MUHAMMAD MASOOD-UR-RAUF KHAN, MUHAMMAD NAVEED SHAHZED, MUHAMMAD JIBRAN RABBANI, MUHAMMAD JAHANGIR RIAZ

ABSTRACT

Aim: To know the frequency of H.Plori infection in causation of DU Perforation
Place of study: This is descriptive type of study which was done in the Department of Surgery, Nishtar Hospital, Multan.
Material and methods: We included all the patients who have DU perforation. Patients with gastric ulcer, prepyloric ulcer perforation and perforated gastric malignancies were excluded from our study.
Result: During the study period 116 patients proved to have perforated duodenal ulcer. In them 92 were males and remaining 24 were females. We found in our study that perforated DU is more common in age group of 30 to 50 years.
Conclusion: H.Pylori, Stress, smoking and NSAIDs abuse are the factors which cause duodenal ulcer perforation. We suggest randomized controlled study should be carried out to evaluate these factors in causation of duodenal ulcer perforation in our part of world.
Key words: Helicobacter Pylori, D U perforation, infection

INTODUCTION

The incidence of perforated peptic ulcer in the Western countries varies between 7 to 9 cases per 1, 00,000 population per year[1]. Once the diagnosis of perforation has been made, it is generally agreed that emergency surgery should be performed as soon as the patient has been adequately resuscitated[2]. Accepted therapeutic options are either simple closure or immediate definitive operation. Conservative treatment, originally proposed by Wangensteen, is reserved for patients considered to be too ill to stand the stress of surgery[3]. Duodenal ulcer is almost unknown in Helicobacter pylori negative individuals; the organism must make the major contributor to the etiology of the disease and in the maintenance of chronicity of the ulcer[4]. Infection with Helicobacter pylori is the main cause of duodenal ulcer[5]. If this infection is cured, ulcers and their complications rarely occur[6]. In a recent review in the *Journal,* Walsh and Peterson point out that "despite the fact that Helicobacter pylori is necessary for the development of peptic ulcer in most patients, it is far from sufficient"[5]. Others have observed that H. pylori had a limited role in causing disease in surgical patients, and suggested that an "adequate acid reduction procedure will still be the main objective of surgical treatment and prevention of Ulcer recurrence"[7]. Recent literatures suggests that the use of Proton Pump Inhibitors as well as therapy directed against Helicobacter pylori, may reduce the necessity for operation to alleviate recurrent perforated peptic ulcer symptoms[8, 9, 10]. Simple closure followed by H-Pylori eradication may become the optimum treatment for majority of cases of duodenal perforations[11]. There is controversy about eradication therapy; some advocate eradication therapy in all the patients who have sustained perforated DU but this management policy is not recommended by all[12]. Persons infected with H, pylori develop serum antibodies to the organism (both IgG and IgA immunoglobulin classes), and can be used diagnostically. Serological methods of identifying circulating antibodies to H. pylori are cheap, quick and non-invasive. Culturing the organism is probably the gold standard, and has sensitivity of 601-95% and specificity of 100% but the method is slow and expensive. Histology and CLO (campylobacter-like organism) tests, having sensitivity of 80-95% and 90-95% respectively and specificity of 100% and 98-100%, are simpler but depend upon invasive techniques to obtain sample. Breath testing the isotopes of carbon after urea ingestion is non-invasive but has high capital cost[13, 14] and has specificity of 98-100% and sensitivity of 95-100%.

Department of Surgery, Nishtar Medical College/Hospital, Multan
Correspondence to Dr. Muhammad Masood ur Rauf Khan, Assistant Professor Surgery email: drmuhammad_786@yahoo.com

PATIENTS AND METHODS

This is descriptive type of study which was done in Surgical Unit III, Nishtar Hospital, Multan. We included all the patients who have DU perforation. Patients with gastric ulcer, prepyloric ulcer perforation and perforated gastric malignancies were excluded from our study.

Sample size in our study was 116 patients who were admitted in Nishtar Hospital Multan from Jan 2006 to Dec 2008. All the patients had perforation in first part of duodenum and all these patients were operated in A & E Department of Nishtar Hospital, Multan. In all those patients Graham's omental patch was done and no other acid reducing procedure like vagotomy of any type or antectomy was done.

All these patients were admitted with peritonitis and gas under diaphragm and were diagnosed with the probability of having DU perforation and the diagnosis was confirmed operatively.

A preformed Proforma was filled for all identified information of the patients including biodata and preoperative and postoperative findings.

Our proforma had provision of complete biodata of our patients with emphasis on either previous history of acid peptic disease is present and patient is taking treatment regularly or irregularly or any risk factor like smoking, NSAIDs abuse, drinking alcohol, stressful life etc. On this basis of that we grouped the patients in four groups:

Group 1: patients with previous history of APD on regular treatment.

Group 2: patients with previous history of APD on irregular treatment.

Group 3: patients with previous history of APD taking no treatment.

Group 4: patients with no previous history of APD.

All patients underwent the test for H.pylori by using ACON® one step H.pylori device which is based on H.pylori IgG with of 89.9% specificity and 95.5% sensitivity.

Detailed history and clinical examination was recorded. Preoperative investigations included routine (CBC, CUE, grouping and cross matching), biochemical (S/E, RPM, RBS, LFTs), radiological (x-ray chest, USG abdomen) were done in all patients. ECG of patients above 45yrs was done. After final diagnosis, patients were operated in emergency. All these patients were given GA. Exploratory laprotomy was done through upper midline incision. Peritoneal lavage with 3 to 4 liters of normal saline was followed by placement of 2 intraperitoneal drains, 1 in subhepatic space and 2nd in pelvis. In immediate postoperative period patients were put on intravenous 3rd generation cephalosporins, strict fluid and electrolyte balance and proper analgesia. Drains were removed on 2nd or 3rd day. On 4th or 5th day patients were allowed to take liquid diets. All the patients who had H.pylori were given eradication therapy.

RESULTS

During the study period 116 patients proved to have perforated duodenal ulcer. In them 92 were males and remaining 24 were females. We found in our study that perforated DU is more common in age group of 30 to 50 years. Age and sex distribution of perforated DU is given in table 1.

Table 1: Age and sex distribution in perforated duodenal ulcer

Age (Yrs)	Total	Female	Male
20-29	21	1	20
30-39	34	10	24
40-49	37	11	26
50-59	11	1	10
60-70	8	0	8
>70	5	1	4

Sixty eight patients were from rural areas and remaining 48 were from urban areas. In group 1, the patients who have previous history of APD, who were taking regular treatment were 19. In group 2, patients who were having APD and were on irregular treatment were 51. The patients in group 3 who were taking no medicine were 20. Remaining 26 patients were in group 4 having no history of APD. But significantly there was history of steroid or NSAID abuse either for joint disease or obstructive airway disease in 17 patients. The patients who were having history of treatment either regularly or irregularly, the drug mostly used was PPI with dose of 20mg at night time. Only few patients gave history of taking H_2 blockers either in the form of cemetidine or famotidine.

Table. 2: No. of patients in each group

Groups	=n	%age
Group 1	19/116	16.37
Group2	51/116	43.93
Group 3	20/116	17.24
Group 4	26/116	22.41

Most of the patients presented in 24 hours of onset of symptoms and the time interval between the symptoms of peritonitis and surgery range from 12 hours to 120 hours. 2 of those patients who presented after 72 hours so very much septic with co-morbid diseases, initially we had to put 2 drains in pelvis bilaterally. One of them died because of multiple organ failure syndromes and second survived. Later on, after 3 days we did Graham's omental patch on the perforation and that patient survived very well.

Table . 3: Groups of patients taking medicine

Groups	=n	Medicine taken			
		PPI	%age	H_2 Blocker	%age
Group 1	19	10	52.62	9	47.36
Group 2	51	34	66.67	17	33.33

When we evaluated on history, the most important factors which we found were smoking, H.pylori,

stressful life style, family history of DU, NSAIDs abuse, steroid intake and alcohol abuse. They are tabulated in table 3.

Table 4: No. of patients having observed risk factors

Risk factors	=n	%age
Helicobacter pylori	70/116	60.3
Smoking	65/116	56.03
Stressful life style	85/116	73.27
Family history of proved DU	10/116	8.62
NSAIDs abuse	35/116	30.17
Steroid abuse	17/116	14.66
Alcohol intake	2/116	1.72

All patients underwent the test for H.pylori by using ACON® one step H.pylori device which is based on anti-H.pylori IgG with of 89.9% specificity and 95.5% sensitivity. Patients who were H.Pylori positive were given full eradication therapy and later on we did upper GI endoscopy of these patients. In our patients the result came that 75 patients out of 116 were positive for H.Pylori. It means that H.Pylori was a culprit in 64% of the cases.

Smoking, stressful life style and NSAIDs were other main and big factors in the causation of duodenal ulcer perforation.

DISCUSION

In spite of overall decline in the incidence of peptic ulcer disease, the incidence of perforated duodenal ulcer has not been reduced in western countries. This may be due to the increased use of NSAIDs over the last 20 years[15, 16, 17]. It has been mentioned that the majority of patients have a preceding history suggestive of chronic duodenal ulcer but about one third of the patients have no history of ulcer or dyspepsia or one which extends to only a week or more[18]. H.Pylori is present in 75 out of 116 patients (64%) which co-relates with the study of Reinbach DH, et al[19], (H.Pylori is not associated in causation of DU perforation).

A study by Dr. Somasekhar R Menakuru, Assistant professor of Surgery Uttaranchal, India, published in Pak J Med Sci April-June 2008 Vol 4, No.2, 157-163, has given that there is controversy regarding the relationship between H.Pylori and perforated DU. It is further stated that "Al-though the relationship between uncomplicated peptic ulcer disease and H. pylori is widely supported, the association with perforation is not fully accepted fully. However, a recent randomized study sought to determine if eradication of H. pylori in patients undergoing simple closure of a perforated duodenal ulcer was effective in preventing ulcer recurrence[20]. The authors noted that fewer surgeons are acquiring enough experience and expertise in performing definitive procedures such as highly selective agronomy and argued that combining medical therapy for H. pylori with simple closure would be more desirable than undergoing definitive operation, given at least a similar rate of recurrence."

This argument does assume an association between H. pylori and ulcer perforation, but the study showed that 95% of H. pylori positive patients undergoing simple repair of the perforated duodenal ulcer followed by combined medical therapy and eradication were ulcer-free at one year. If the initial findings of this study continue unchanged, they have great potential to change the traditional management of perforated peptic ulcer disease and therefore, more evaluation is needed.

Until 1982, Marshall and Warren provided the first insight into another important pathogenic factor in peptic ulcer disease. They isolated a spiral urease-producing organism nestled in the narrow interface between the gastric epithelial cell surface and the overlying mucus gel, which was later named Helicobacter pylori. That discovery had a great impact on the treatment of peptic ulcer, changing a chronic relapsing disease into a curable disease. There is no doubt of the importance of H. pylori in uncomplicated peptic ulcer disease. Most of the published data have confirmed significant reduction in ulcer recurrence after eradication of this bacteria[21, 22]. NIH Consensus development panel on H. pylori[23] concluded that ulcer patients with H. pylori infection required treatment, with antimicrobial agents in addition to anti-secretory drugs. H. pylori eradication also shows a positive impact in treating bleeding ulcer. There is now good evidence that recurrent ulceration and bleeding can be prevented by H. pylori eradication[24]. However, its importance and correlation in perforated peptic ulcer remains an unsettled issue. Reinbach[25]and Chowdhary[26] reported the lack of such an association. In contrast Sebastian, Ng, Chu and Tokunaga supported a significant relation between H. pylori infection and perforated peptic ulcer[2728] They recommended eradicating this bacteria to prevent ulcer recurrence.

In our study 35 out of 116 (30.17%) patients gave the history of using NSAIDs indicating that NSAIDs are the important factor in causation of perforation. Literature indicates that NSAIDs is one of the main factors in causation of DU perforation. Our study indicates that perforation is more common in males which agrees with other studies[29]. The peak age in one study, ulcers were more frequent in the fourth decade.

Difference in study in terms of perforation between people living in rural and urban areas is not significant. As smoking is known to have a number of adverse effects on mucosal aggressive and protective factors[30], in this study, cigarette smoking, especially in men, has strong association with

perforated DU. In our study perforation happened in 73.27% patients who gave history of stressful life. 22.41% were asymptomatic. This high incidence of perforation without preceding history is explained on fact that because of economical and logistic problems stress becomes a significant problem. Alcohol is a big factor causing the duodenal ulcer perforation, but not in our society due to religious factors.

CONCLUSIONS

H.Pylori, Stress, smoking and NSAIDs abuse are the factors which cause duodenal ulcer perforation. We suggest randomized controlled study should be carried out to evaluate these factors in causation of duodenal ulcer perforation in our part of world.

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