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INCIDENCE OF DUODENAL ULCER PERFORATION CAUSED BY HELICOBACTER PYLORI INFECTION

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Abstract:
Purpose: *To know the frequency of H. Pylori infection causing DU perforation.*
Place of study: *This study is a descriptive study conducted at the Medicine Unit I and Surgical Unit II of Services Hospital Lahore for Six months duration from July 2019 to December 2019.*
Material and methods: We included all patients with DU perforation. Patients with pre-pyloric ulcer, gastric ulcer and perforation and perforated gastric malignancies were excluded from the study.
Results: *116 patients with perforated duodenal ulcer were observed during the study. 92 of them are men and 24 women. In our study, we found that perforated DU is more common in the 30-50 age group.*
Conclusion: *H. Pylori, stress, smoking and abuse of NSAIDs are factors causing perforation of a duodenal ulcer. We suggest conducting a randomized controlled trial to assess those factors that cause perforation of duodenal ulcers in our world.*
Key words: *Helicobacter Pylori, DU perforation, infection.*

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INTRODUCTION:

The incidence of perforated peptic ulcers in Western countries ranges from 7 to 9 cases per 1000 inhabitants per year. After diagnosis of perforation, it is generally accepted that emergency surgery should be performed as soon as the patient is properly resuscitated[1,2]. Accepted therapeutic options are a simple closure or an immediate precise procedure. Conservative treatment originally recommended by Wangensteen is reserved for patients who are considered too sick to endure the stress of surgery. Duodenal ulcer is almost unknown in patients with negative Helicobacter pylori; The body should contribute to the etiology of the disease and maintain the chronicity of the ulcer. Helicobacter pylori infection is the main cause of duodenal ulcers. If this infection heals, ulcers and complications rarely appear. A recent review in the journal Walsh and Peterson emphasize that "Helicobacter pylori is far from sufficient, although it is necessary for the development of peptic ulcer in most patients." Others have found that H. pylori plays a limited role in causing disease in surgical patients and that "an appropriate acid reduction procedure will continue to be the primary goal of surgical treatment and prevention of ulcer recurrence." Recent literature suggests that the use of proton pump inhibitors as well as targeted treatment against Helicobacter pylori may reduce the need for surgery to alleviate the symptoms of recurrent perforated peptic ulcer[3]. A simple closure followed by H. Pylori eradication may become the most appropriate treatment for most cases of duodenal perforation. There is a debate about eradication therapy; Some defensive eradication therapy in all patients with perforated DU, but not all recommend this policy. People infected with H, pylori produce anti-body serum antibodies (immunoglobulin classes IgG and IgA) and can be used for diagnosis[4]. Serological methods for identifying antibodies circulating in H. pylori are not cheap, fast and invasive[5]. Blood culture is probably the gold standard and has a sensitivity of 601–95% and 100% specificity, but this method is time consuming and expensive. Histological and CLO (Campylobacter-like) tests with 80-95% and 90-95%, 100% and 98-100% sensitivity, respectively, are simpler, but their achievement depends on invasive techniques[6,7]. The carbon isotope breath test after urea intake is not invasive, but has a high capital cost and has 98-100% specificity and 95-100% sensitivity.

PATIENTS AND METHODS:

This study is a descriptive study conducted at the Medicine Unit I and Surgical Unit II of Services Hospital Lahore for Six months duration from July 2019 to December 2019. We included all patients with DU perforation. Patients with pre-pyloric ulcer, gastric ulcer and perforation and perforated gastric malignancies were excluded from the study. The sample size in our study was 116 patients. All patients had perforation in the first part of the duodenum. All of these patients underwent Graham's omental patch and no other acid reduction procedure such as vagotomy or antectomy was performed.

All these patients were hospitalized with peritonitis and gas under the diaphragm, the possibility of DU perforation was diagnosed, and the diagnosis was confirmed by surgery. The pre-created Proforma has been completed for all defined patient information, including biological data, preoperative and postoperative results. In our form, we provided complete biological data from our patients who highlighted any of the previous peptic acid diseases, and the patient receives all risk factors such as regular or irregular treatment or smoking, NSAID abuse, alcohol, life. stressful etc. Based on this, we divide patients into four groups:

Group 1: Patients with a history of APD undergoing regular treatment.

Group 2: Patients with a history of APD on irregular treatment.

Group 3: Patients with a history of APD without treatment.

Group 4: Patients without an APD history.

All patients underwent Pylori tests with 89.9% specificity and 95.5% sensitivity.

Detailed history and clinical trial recorded. Preoperative examinations were routine (CBC, CUE, combining and comparison), biochemical (S / E, RFTS, RBS, LFT), radiological (chest radiography, abdominal ultrasound) in all patients. An ECG test was performed on patients over 45 years of age. After the final diagnosis, the patients were operated on as a matter of urgency. All these patients were given GA. An exploratory laprotomy was performed through an incision in the upper midline. After peritoneal lavage containing 3 to 4 liters of normal saline, 2 intraperitoneal drains were placed, one in the under hepatic space and the other in the pelvis. In the immediate postoperative period, patients underwent intravenous third-generation cephalosporins, solid fluid and electrolyte balance, and adequate analgesia. Channels were removed on the second or third day. On the 4th or 5th day, patients could be on a liquid diet. Eradication therapy was used in all patients with H. pylori.

RESULTS:

116 patients with perforated duodenal ulcers were observed during the study period. 92 of them are men and 24 women. In our study, we found that perforated DU is more common in the 30-50 age group. The distribution of age and gender 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 the remaining 48 patients were from urban areas. In group 1, patients who had an earlier history of APD and were regularly treated were 19. In group 2, 51 patients with APD and receiving irregular treatment had 51. Patients in group 3 had 20 to take medication. The remaining 26 patients were in the group 4 without a history of APD. However, 17 patients had a significant history of steroid or NSAID abuse due to joint disease or obstructive airway disease. The most commonly used drug was PPI 20 mg a night for patients with a regular or irregular history. Only a few patients had H2-blockers in the form of semetidine 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 patients applied within 24 hours of onset of symptoms, and the interval between peritonitis and surgery was 12 to 120 hours. In 2 patients who had both sepsis after 72 hours with comorbidities, we had to pre-insert 2 drains into the bilateral pelvis. One of them died due to multiple organ failure syndrome, and the other survived. Then, after 3 days, we made Graham's omniparent patch on the earring and this patient survived very well.

Table 3: Groups of patients taking medicine

Groups	=n	Medicine take			n
		PPI	%age	H2 Blocker	%age
Group 1	19	10	52.62	9	47.36
Group 2	51	34	66.67	17	33.33

The most important factor we found when assessing history was smoking, H. pylori, stressful lifestyle, family history DU, NSAID abuse, steroid use and alcohol abuse. They are given 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

Patients with a positive H. Pylori result received full eradication therapy, followed by upper gastrointestinal endoscopy. For our patients, the result is that 75 of 116 patients are positive for H. Pylori. This means that in 64% of cases H. Pylori is guilty. Smoking, stressful lifestyle and NSAIDs are other important and important factors for duodenal ulcer perforation.

DISCUSSION:

Despite the overall reduction in the incidence of peptic ulcers, the incidence of perforated duodenal ulcers has not decreased in Western countries. This may be due to the fact that the use of NSAIDs has increased over the past 20 years. Most patients have been reported to have a history suggestive of chronic duodenal ulcer, but about a third of patients have no history of ulcers or indigestion or have a history that only lasts a week or more. H. Pylori is present in 75 (64%) of the 116 patients associated with Reinbach DH et al[8]. (H. Pylori is not associated with the cause of DU perforation). He is an adjunct surgery in Uttaranchal, India. The study

of Somasekhar R. Menakuru led to controversy published in Pak J Med Sci Volume 4, No. 2, 157-163 in April and June 2008. He perfected the relationship between H. Pylori and DU. In addition, "Although the relationship between uncomplicated peptic ulcer disease and H. pylori has been widely supported, it was noted that the association with perforation was not fully accepted, but recently a randomized trial has attempted to determine whether eradication of H. Pylori ulcer occurs in patients undergoing simple perforated closure Duodenal ulceration.[9] The authors concluded that fewer surgeons gained sufficient experience and expertise in performing precise procedures, such as highly selective agronomy, and combining treatment with simple H. pylori closure would be preferable to performing the final surgery. This argument assumes a link between H. pylori and ulcer perforation, but the study showed that 95% of patients with H. pylori positive underwent a simple repair of the ulcer[10]. Duodenal ulcer, followed by combined treatment and eradication, did not cause ulcers per year, if it remains unchanged, the potential for change in traditional perforated ulcer treatment is high, and therefore, further assessment presents the first idea ofanother fact until 1982. The urease-producing organism at the narrow junction between this discovery had a great impact on the treatment of peptic ulcers that transformed recurrent chronic disease into a curable disease. There is no doubt about the importance of H. pylori in peptic ulcer disease[12]. published data confirmed a significant reduction in ulcer recurrence after destruction of this bacterium. The NIH consensus development team at H. pylori came to the conclusion that patients with ulcers with H. pylori infection require treatment with antimicrobials in addition to antisecretory drugs[13]. Elimination of H. pylori also has a positive effect on the treatment of bleeding ulcers. There is currently good evidence that H. pylori eradication can prevent the recurrence of ulcers and bleeding. However, its significance and correlation in perforated peptic ulcer is still an unsolved problem. Reinbach and Chowdhary reported that there was no such relationship. Sebastian, Ng, Chu and Tokunaga, on the other hand, supported the significant relationship between H. pylori infection and perforated peptic ulcer. They suggested that these bacteria would be destroyed to prevent the ulcer from coming back. In our study, 35 of 116 patients (30.17%) had NSAIDs in the past, indicating that NSAIDs are an important factor causing perforation[14]. The literature shows that NSAIDs are one of the main factors causing DU perforation. Our study shows that perforation is more common in men than in other studies. In one study, peak age and ulcers were more common in the fourth decade. In the study, the difference between people living in rural and urban areas is not significant. Since smoking is known to have a number of negative effects on aggressive and mucosal protective factors, smoking has a strong association with perforated DU, especially in men, in this study. In our study, 73.27% of patients with stressful life history occurred in the perforation. 22.41% were asymptomatic[15]. This high frequency of ulcers without history is due to the fact that stress is becoming an important problem due to economic and logistical problems. Alcohol is an important factor that causes perforation of a duodenal ulcer, but it is not caused by religious factors in our society.

CONCLUSIONS:

H.Pylori, stress, smoking and NSAIDs are factors that cause perforation of the duodenal ulcer. We suggest conducting a randomized controlled trial to assess those factors that cause perforation of duodenal ulcers in our world.

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