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Evaluation of pharmacological treatment and outcome of peptic ulcer in Iraqi patients

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ABSTRACT

Background: Peptic ulcer disease (PUD) is characterized by a disruption of mucosal integrity of stomach, duodenum or both, caused by local inflammation, which leads to well defined mucosal defect. *Aim*: This study aimed to evaluate treatment of PUD and outcome in Iraqi patients, whether controlled or uncontrolled in correlation with treatment. *Methodology*: A total of 520 patients were enrolled from several hospitals in Baghdad. *Results*: Peptic ulcers were most common in patients over 60yrs of age (50%), followed by patients between 40-59yrs of age (27%), then patients between 18-39 years of age (23%). Moreover, PU is predominant in males (73%) of total patients. 65% of patients in study were smokers. *H. pylori* was identified as the causative agent in approximately 15% of cases. Analysis of treatment practices in Iraqi hospitals revealed that 61% of patients received proton pump inhibitors (PPIs), 24% were treated with H_2-receptor antagonists, and only 15% were administered a targeted anti-*H. pylori* regimen. 69% of patients were controlled in correlation with treatment schedule. *Conclusion*: Elderly individuals are at higher risk for development of PU. Smoking is a significant risk factor since it stimulates basal acid output.

KEYWORDS

peptic ulcer, *H. pylori*, PPIs, gastric bleeding, ulceration

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1. INTRODUCTION

Peptic ulcer disease occurs when the corrosive action of aggressive factors such as gastric acid, pepsin, and bile surpasses the protective mechanisms of the gastrointestinal mucosa, resulting in a full-thickness disruption of the gastric or duodenal lining [1].

Stress, smoking, alcohol, NSAIDs (Non-Steroidal Anti-inflammatory Drugs), bisphosphonates, KCl, immunosuppressive agents, and an age related decline in PGs levels have all been shown to contribute to PUD. It was, however, the isolation of *H. pylori* and its identification as the most important cause of peptic ulcer disease that led to exploration of the role of inflammation and its associated cytokine cascade in gastric acid secretion [2].

Duodenal ulcer is essentially an *H. pylori*-related disease and is caused mainly by an increase in acid and pepsin load, and gastric metaplasia in the duodenal cap. Gastric ulcer, at least in Western

countries, is most commonly associated with NSAIDs ingestion, although *H. pylori* infection might also be present [3].

Prevention of gastric ulcers often involves reducing NSAID use and finding different medications or alternative approaches to relieve pain. For example, acetaminophen has not been linked to peptic ulcers. If NSAIDs need to be taken long-term, H_2 blockers or proton pump inhibitors may also be prescribed to prevent the development of peptic ulcers [4].

Doctors recommend eating small portions during meals of foods rich in fiber, especially fruits, vegetables, and whole grains that are also vitamin-rich, which may enhance the body's ability to heal stomach irritation and prevent ulceration [5].

Proton-pump inhibitors (PPIs) are a group of drugs that display pronounced and long-lasting reduction of stomach acid production. H_2 blockers suppress the secretion of HCl by parietal cells by two mechanisms: histamine released by enterochromaffin-like cells in the stomach is blocked; and as a consequence, other substances that promote acid secretion (e.g. gastrin and Ach) have a reduced effect on parietal cells when H_2 receptors are blocked [6].

Anti *H. pylori* regimens include first-line eradication regimens that achieve high rates of both eradication and patient compliance. Two triple therapy day regimens are currently accepted as first-line therapy; they combine PPI with either metronidazole and clarithromycin or amoxicillin and clarithromycin. These regimens generally achieve eradication rates of >80% [7].

The aim of this study is to evaluate the pharmacological treatment of PUD in Iraq and to determine the outcome of PUD in Iraqi patients, whether controlled or uncontrolled in correlation with followed pharmacological treatment.

2. METHODOLOGY

2.1. Study design and participants

This study was conducted at Baghdad teaching hospital, Al-Yarmouk teaching hospital, Al-Karama teaching hospital & Al-Kadhimiya teaching hospital, Baghdad, Iraq, and designed as a cross-sectional study. The study enrolled 520 patients of both sexes. Subjects enrolled in the study are ≥18 years. Women who were pregnant or nursing were excluded from the study.

2.2. Data collection

Data collection based on demographic factors including age, gender, smoking status, NSAIDs history, *H. pylori*, pharmacological treatment, and patient's outcome. The participant's height (m) and body weight (kg) were measured while they were unclothed and without shoes. BMI was calculated by dividing weight in kg by the square of height in meters.

2.3. Ethical approval

Informed consent was obtained from all participants prior to their participation in the study. Participants were first required to read an introductory statement outlining the study's purpose and procedures. Only those who provided consent by selecting the appropriate option were granted access to questionnaire. Individuals who declined to consent were presented with a thanks message and were not permitted to proceed. Ethical approval for the study was obtained from the Ethics Committee of the College of Pharmacy, Al-Bayan University.

2.4. Statistical analysis

In this study, ANOVA was employed to compare the effectiveness of different pharmacological treatment regimens in relation to patient outcomes (controlled vs uncontrolled). The three pharmacological groups analyzed were: PPIs, H_2-receptor antagonists, and Anti-*H. pylori* regimens. The primary dependent variable analyzed was the treatment outcome, categorized as controlled or uncontrolled, reflecting clinical response to therapy. Following the identification of statistically significant differences among treatment groups via ANOVA, Tukey's post hoc test was conducted to determine pairwise comparisons between the treatment regimens. This post hoc analysis was essential to specify which particular treatments differed significantly in their association with improved control of PUD. The statistical analysis was carried out by using SPSS 16.0; the level of significance was set at $p<0.05$.

3. RESULTS

Results shown that PUD was predominantly found in patients aged 60 yrs and older compromising 50% of the study population, then patients aged between 40-59 years (27%), and patients aged between 18-39 years (23%). PUD was predominantly found in males compromising 73% of the study population. This was followed by females (27%). 65% of the study population were smokers. 50% of the study population were using NSAIDs (non-steroidal antiinflammatory drugs). *H. pylori* has 15% involvement as the causative agent of PUD. The

treatment schedule in Iraqi hospitals was the patients were using PPIs compromising 61% of the study population. The patients using H_2-Rc antagonists (24%), and patients using anti *H. pylori* regimen (15%). The controlled patients compromising 69% of the study population in correlation with treatment schedule. The mean BMI was 26.88±2.6 kg/m^2 as shown in (Table 1).

Table 1. Baseline characteristics of the patients (N=520).

Demographic factors		Value
Age (years)	≥ 60 40-59 18-39	260 (50%) 140 (27%) 120 (23%)
Gender	Male Female	380 (73%) 140 (27%)
Smoking	Smoker Non-smoker	338 (65%) 182 (35%)
NSAIDs history	Never Ex/current	260 (50%) 260 (50%)
H. pylori	Positive Negative	78 (15%) 442 (85%)
Pharmacological treatment	PPIs H_2-antagonist anti *H. pylori* regimen	317 (61%) 125 (24%) 78 (15%)
Patient's outcome	Controlled Uncontrolled	359 (69%) 161 (31%)
BMI, kg/m^2	Mean ±SD Range	26.88±2.6 24.27-40.33

4. DISCUSSION

Elderly patients are more susceptible to PUD, largely because of altered gastric microbiota, reduced mucosal protective mechanisms, decreased gastric blood flow, and consequently compromised repair mechanisms-hallmarks of age-related gastric changes [8].

Duodenal and gastric ulcers have historically demonstrated a higher prevalence in males compared to females, with duodenal ulcers occurring more frequently than gastric ulcers. This disparity may be attributed to the protective role of estrogen in females, which has been shown to enhance the expression of tight junction proteins, thereby reinforcing epithelial barrier integrity, reducing mucosal permeability, and promoting bicarbonate ion secretion in the duodenal mucosa. Although PUD was predominantly observed in males in earlier epidemiological data, recent trends in Western countries indicate a shift toward a more balanced gender distribution, with prevalence rates now approaching parity between males and females [9].

Smoking significantly increases the risk of recurrent PUD due to harmful physiological effects of nicotine that include increased gastric acid secretion, impaired mucosal defense, and reduced bicarbonate secretion. These changes contribute to a higher risk of ulceration in both the stomach and duodenum [10].

5. CONCLUSION

Elderly patients are at higher risk for the development of peptic ulcer. Male obviously more prone to PUD than female. Smoking is a significant risk factor for PUD development, since it stimulates the basal acid output which is more pronounced in smokers having duodenal ulcers. The risk of developing PUD is higher among persons with lower socioeconomic status, possibly due to a higher prevalence of *H. pylori* infection. Majority of PUD cases are controlled by pharmacological treatment schedules followed by Iraqi hospitals.

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CONFLICT OF INTEREST STATEMENT
The authors declare no conflicts of interest.

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