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Research Paper

Peptic Ulcer Disease: Etiology, Epidemiology, and the Evolving Role of Helicobacter Pylori and NSAIDs

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Abstract
Peptic ulcer disease (PUD) is a common gastrointestinal condition characterized by a break in the lining of the stomach or duodenum, often associated with Helicobacter pylori (H. pylori) infection and nonsteroidal anti-inflammatory drug (NSAID) use. Focusing on the declining prevalence of H. pylori in Western countries and the rising importance of NSAIDs as a contributing factor. Despite improved hygiene and medical interventions, PUD remains a significant health concern due to widespread NSAID use, especially among older populations. The etiology of PUD is multifactorial, involving an imbalance between gastric acid secretion and mucosal defense mechanisms, exacerbated by H. pylori infection, NSAIDs, and genetic predispositions. Lifestyle factors such as smoking and alcohol consumption also increase the risk of ulcer development. Clinically, PUD typically presents with epigastric pain This review explores the epidemiology, etiology, and clinical features of PUD, but asymptomatic cases are not uncommon, leading to severe complications like gastrointestinal bleeding. This review highlights the importance of early diagnosis and treatment to prevent the progression of PUD and its associated complications.

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I. Introduction

Peptic ulcer disease (PUD) is characterized by a break in the stomach lining or duodenum, penetrating through the muscular mucosa, with typical symptoms including epigastric pain relieved by food or antacids (5). PUD is primarily classified as either gastric (along the stomach's lesser curvature) or duodenal (usually in the duodenal bulb). Historically, Helicobacter pylori (H. pylori) has been the main cause of 90% of duodenal and 80% of gastric ulcers (6). However, the prevalence of H. pylori has declined in Western countries, with nonsteroidal anti-inflammatory drugs (NSAIDs) and low-dose aspirin now playing a larger role in PUD. Despite improved management and hygiene, the continued use of NSAIDs keeps PUD a significant concern, with associated medical costs ranging from USD 163-866 per patient(7).

1.1 Epidemiology

PUD remains common worldwide, with an annual incidence of 0.10% to 0.19% for physician-diagnosed cases and 0.03% to 0.17% for hospital-diagnosed cases. Prevalence has decreased in Western populations due to declining H. pylori infections, but in some Asian countries, such as Korea, H. pylori-associated gastric ulcers are on the rise. In Sweden, the overall prevalence of PUD was 4.1%, with asymptomatic cases accounting for 19.5%. Asymptomatic PUD can lead to severe complications such as gastrointestinal bleeding, which is linked to mortality rates approaching 10%.

1.2 Etiology

While H. pylori remain a significant cause of ulcers (95% of duodenal and 70% of gastric ulcers), NSAIDs are responsible for 14-25% of cases. The combined effects of H. pylori infection and NSAID use can be cumulative, leading to more severe ulcers. The primary mechanism involves an imbalance between gastric acid secretion and mucosal defense, exacerbated by histamine release, which stimulates further acid production, creating a cycle that leads to ulcer formation.

1.3 Role of H. pylori Infection

H. pylori-induced ulcers largely depend on the interaction between host and bacterial factors. In cases of high acid production, inflammation is concentrated in the distal stomach and duodenum, leading to duodenal ulcers. Conversely, reduced acid output causes inflammation in the gastric transitional zone, resulting in gastric ulcers. *H. pylori* induces a Th1-polarized immune response, which may contribute to inflammation without clearing the infection. Cytokine gene polymorphisms, such as those involving interleukin-1β, are thought to influence susceptibility to ulceration and gastric cancer.

1.4 Role of NSAIDs

NSAIDs increase the risk of ulceration by inhibiting cyclooxygenase (COX) enzymes, reducing protective prostaglandins in the gastric mucosa. This weakens the gastric barrier, allowing acid and pepsin to cause damage. The risk of complications increases with age, prior ulcer history, and the first few months of NSAID use. Genetic predispositions, such as blood group O and family history, also contribute to the risk of developing PUD.

1.5 Smoking and Alcohol

Smoking and alcohol consumption are risk factors for PUD. Smoking decreases epidermal growth factor and increases free radical production, while chronic alcohol use inhibits COX-1 enzymes, reducing protective prostaglandins. However, smoking alone may not significantly contribute to ulcer formation unless combined with *H. pylori* infection.

Table 1. Causes of Gastroduodenal Ulcers (information adapted from reference 4)

CUSE	Comment
COMMON CAUSE	
HELICOBACTER PYLORI INFECTION	A motile, gram-negative spiral rod was discovered in 48% of peptic ulcer patients. Four NSAIDs Peptic ulcer disease affects 5–20% of persons who take NSAIDs for extended periods. Patients who use steroids or anticoagulants, have a history of ulcers or gastrointestinal bleeding, are elderly, or have significant organ dysfunction are more likely to experience NSAID-induced ulcers and problems. potassium chloride, steroids, bisphosphonates, and chemotherapy drugs (such as intravenous fluorouracil)
NSAIDS	A motile, gram-negative spiral rod was discovered in 48% of peptic ulcer patients. Four NSAIDs Peptic ulcer disease affects 5–20% of persons who take NSAIDs for extended periods. Patients who use steroids or anticoagulants, have a history of ulcers or gastrointestinal bleeding, are elderly, or have significant organ dysfunction are more likely to experience NSAID-induced ulcers and problems. potassium chloride, steroids, bisphosphonates, and chemotherapy drugs (such as intravenous fluorouracil)
OTHER MEDICATION	A motile, gram-negative spiral rod was identified in 48% of individuals with peptic ulcers. Four NSAIDs
RARE	
ACID-HYPERSECRETORY STATES	Five to twenty percent of those who take NSAIDs for a long time develop peptic ulcer disease.
MALIGNANCY	NSAID-induced ulcers and difficulties are more common in patients who use steroids or anticoagulants, have a history of ulcers or gastrointestinal bleeding, are old, or have serious organ malfunction.
STRESS	Bisphosphonates, steroids, potassium chloride, and chemotherapy medications (such as intravenous fluorouracil)

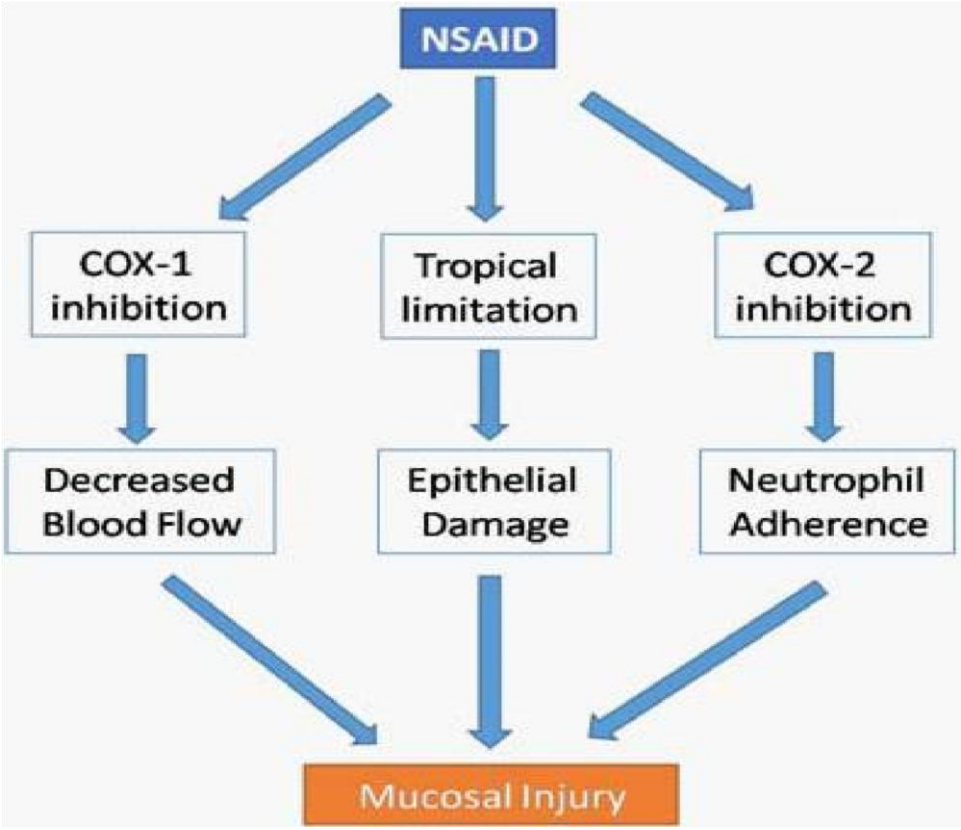

Figure 1: Pathogenesis of NSAID-induced GI injury.

1.6 Clinical Features

Epigastric pain is the hallmark symptom of PUD, described as gnawing, dull, or hunger-like. Pain may be relieved by food or antacids and often returns during fasting or at night. Additional symptoms include fullness, bloating, early satiety, nausea, and, in some cases, weight loss or vomiting. Chronic ulcers, especially those caused by NSAIDs, may be asymptomatic, with complications such as bleeding or perforation being the first sign. Older patients are more likely to present with bleeding, while younger individuals more often report abdominal pain.

II. Materials and Method

This study employed a retrospective literature review approach, using data from previously published research on peptic ulcer disease (PUD) and its association with *Helicobacter pylori* (H. pylori), nonsteroidal anti-inflammatory drugs (NSAIDs), and other risk factors. The methodology involved the following steps:

2.1 Data Sources: A thorough search of electronic databases, such as PubMed, Google Scholar, and Scopus, yielded pertinent studies. Search terms that were used were "peptic ulcer disease," "NSAIDs," "H. pylori," "gastric ulcer," and "duodenal ulcer." Studies were selected based on relevance to PUD etiology, epidemiology, and clinical features.

2.2 Inclusion Criteria: Studies published in English within the last two decades (2000–2023) were included. These studies covered key aspects such as the role of *H. pylori* in ulcer formation, the impact of NSAIDs, genetic predispositions, and the influence of lifestyle factors like smoking and alcohol consumption.

2.3 Exclusion Criteria: Articles that focused on unrelated gastrointestinal conditions, lacked clinical data, or did not meet quality standards (e.g., poorly designed studies or those with small sample sizes) were excluded.

2.4 Data Extraction and Analysis: Data from the selected studies were extracted and synthesized to identify trends in PUD prevalence, etiological factors, and clinical outcomes. Special focus was given to studies examining the interaction between *H. pylori* infection and NSAID use, as well as the role of genetic and environmental factors.

2.4 Epidemiological Trends: The methodology also reviewed epidemiological data to track the decline in *H. pylori* prevalence, particularly in Western populations, and the corresponding changes in PUD incidence. Data from Asian countries were examined to contrast global trends, particularly about the rising incidence of gastric ulcers.

2.5 Limitations: Acknowledged limitations include the variability in study designs and geographic regions, the potential for publication bias, and the reliance on retrospective data, which may underreport asymptomatic cases of PUD and its complications.

III. Results

With a sample size of 65, this descriptive study was carried out in Tripoli, namely in the provinces of Tajour and Ain Zara. Information was gathered using a modified interviewer-administered semi-questionnaire that had been pre-tested. 65 respondents were interviewed to gather data on lifestyle and sociodemographic characteristics. Excel version 2013 was used to enter and evaluate all of the data.

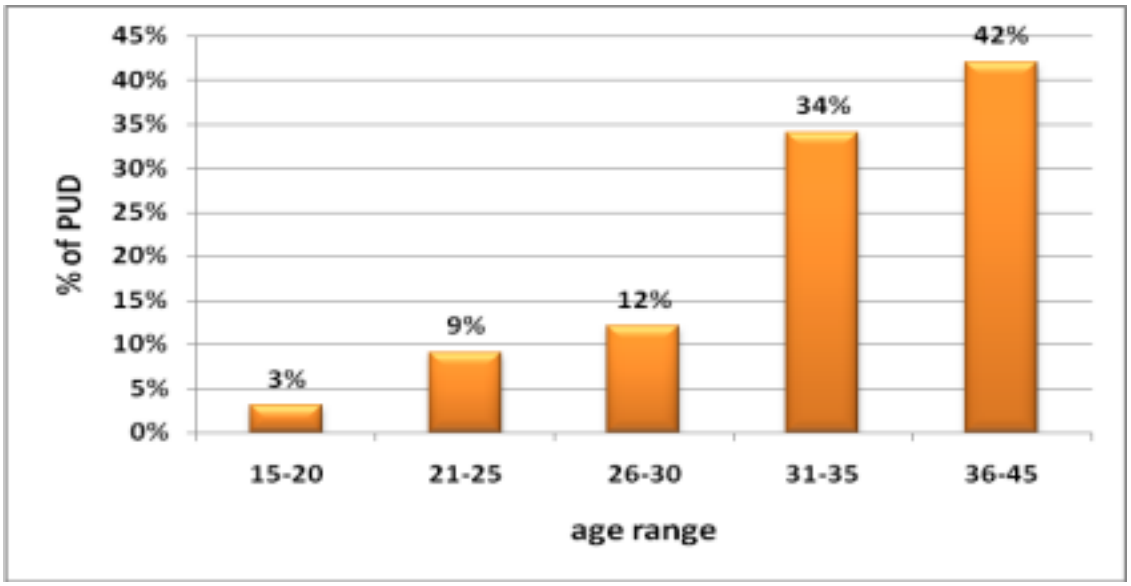

Figure 2. Distribution of the respondents by age (n=65)

According to graph number 2, 3%, 9%, 12%, 33%, and 41% of the respondents are between the ages of 15 and 20 years, 21 and 25 years, 26 and 30 years, 31 and 35 years, and 36 and 45 years.

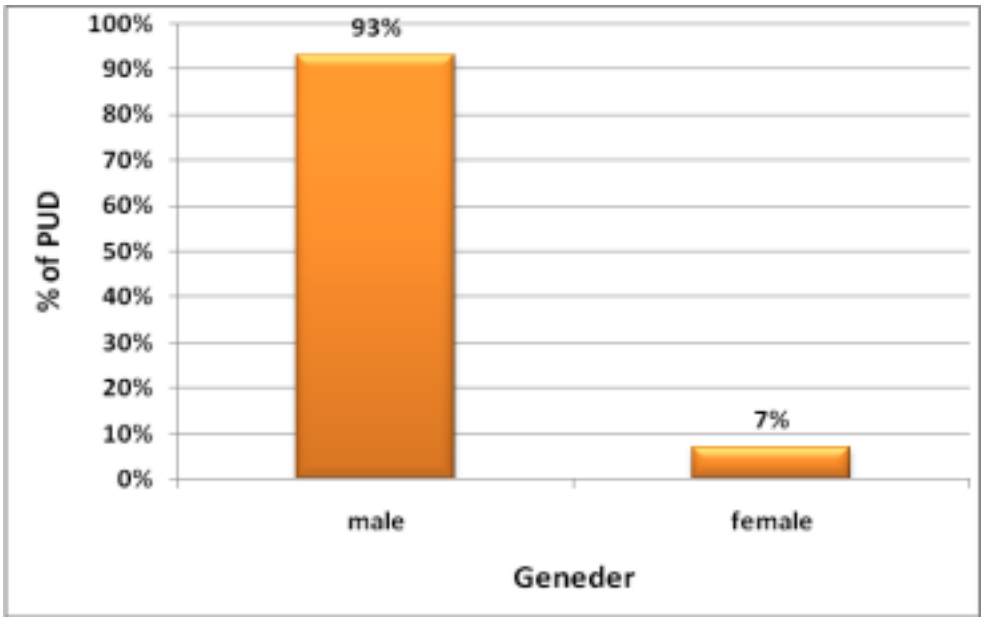

Figure 3 Percentage of respondents by sex

As we can see in graph no 3, the majority of the participants in this survey were male 93% and the rest of the participants were female at 7%

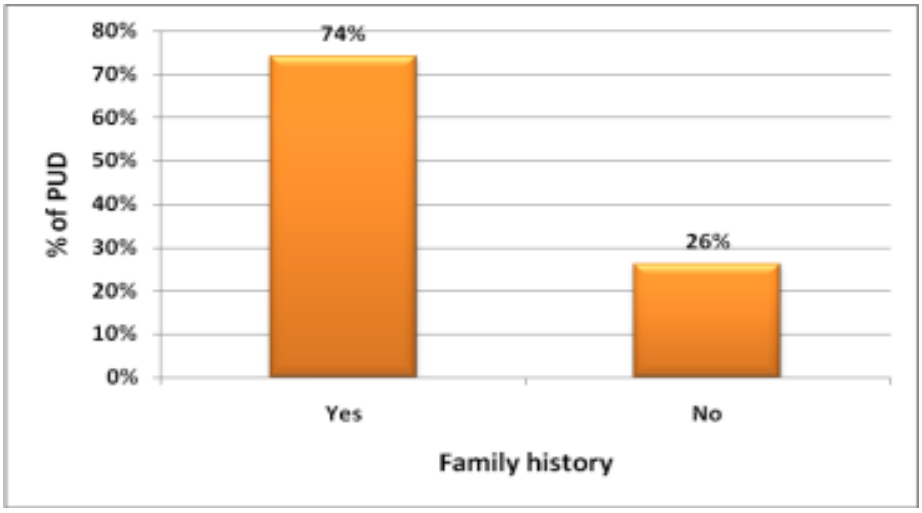

Figure 4, Distribution of respondents by family history of PUD

Figure number 4 demonstrated that 74% of participants answered that there was no family history of PUD while 26% of participants had a family history.

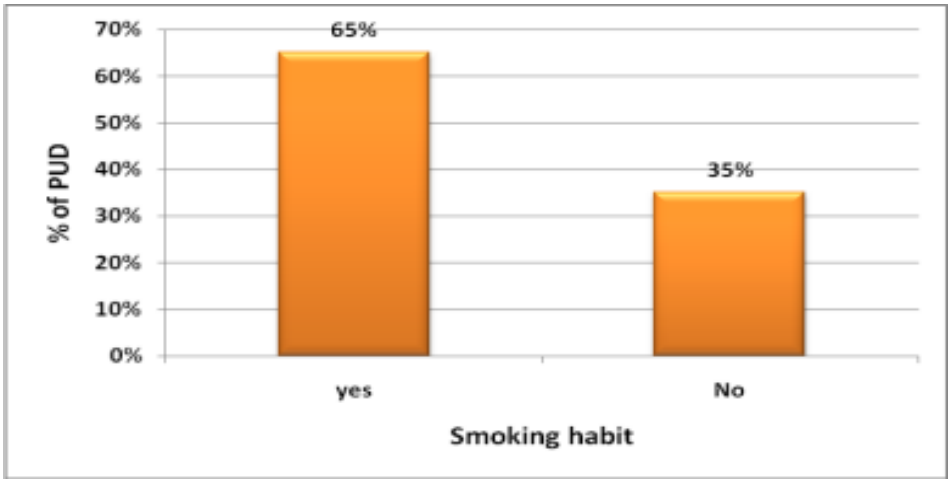

Figure 5 percentage of respondents by smoking habit.

Figure 5 shows that the majority of PUDs involved in this study were smoking (65%) and 35 % did not.

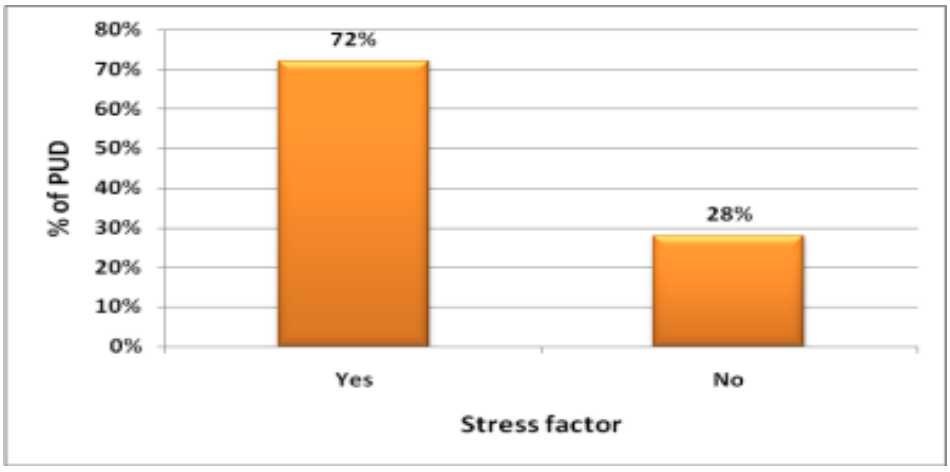

Figure 6, respondents by stress factors

Figure 6 shows that 72% of respondents experienced stress, tension, or anxiety, whereas 28% did not.

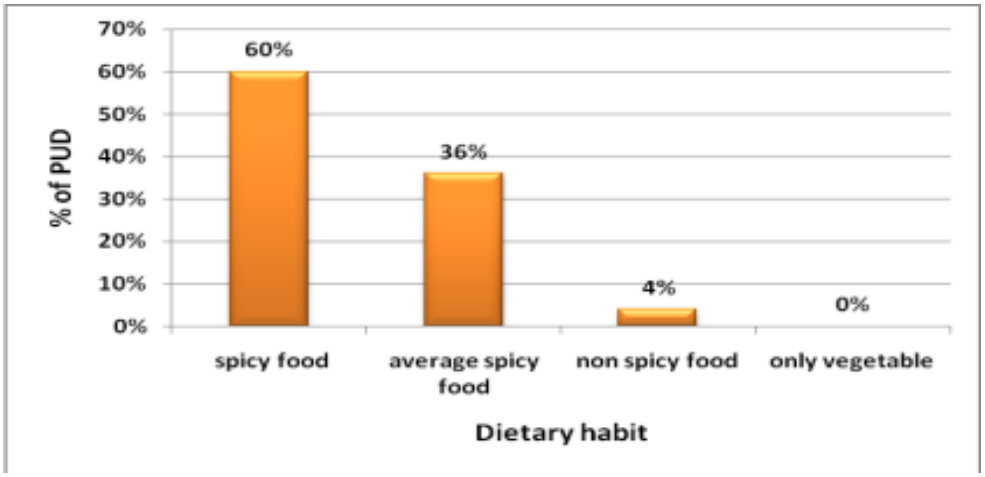

Figure 7, distribution of respondents by dietary habit

Hot food accounted for 60% of the respondents' diets, followed by average hot food (36%), and non-spicy food (3%).

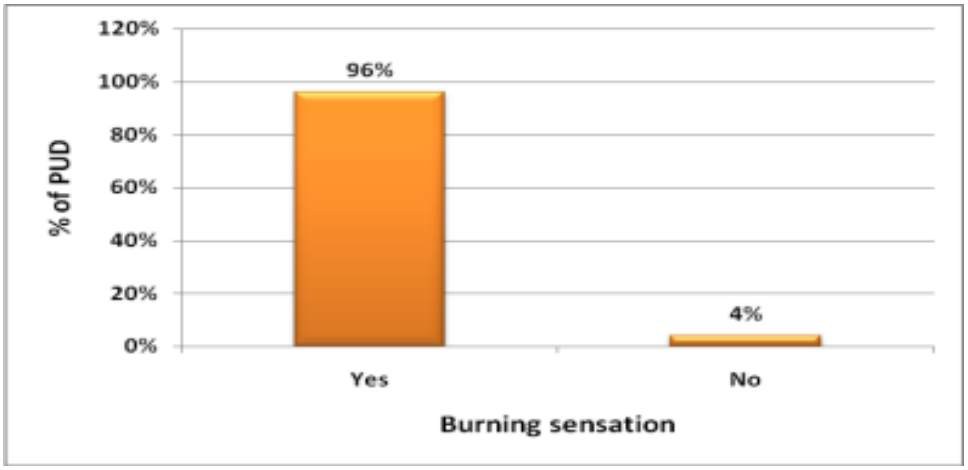

Figure 8, Respondent distribution based on epigastric burning feeling.

As we can see according to the burning sensation 96% of participants have a burning sensationfeeling as compared with 4% who did not.

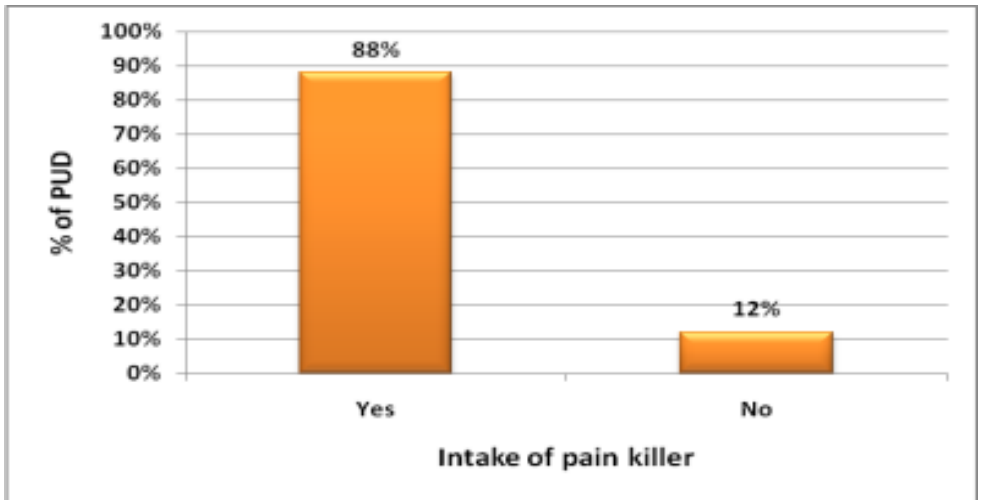

Figure 9, Respondent distribution based on painkiller use.

As we can see, most participants were male with about 88%, while the others were female with 12%.

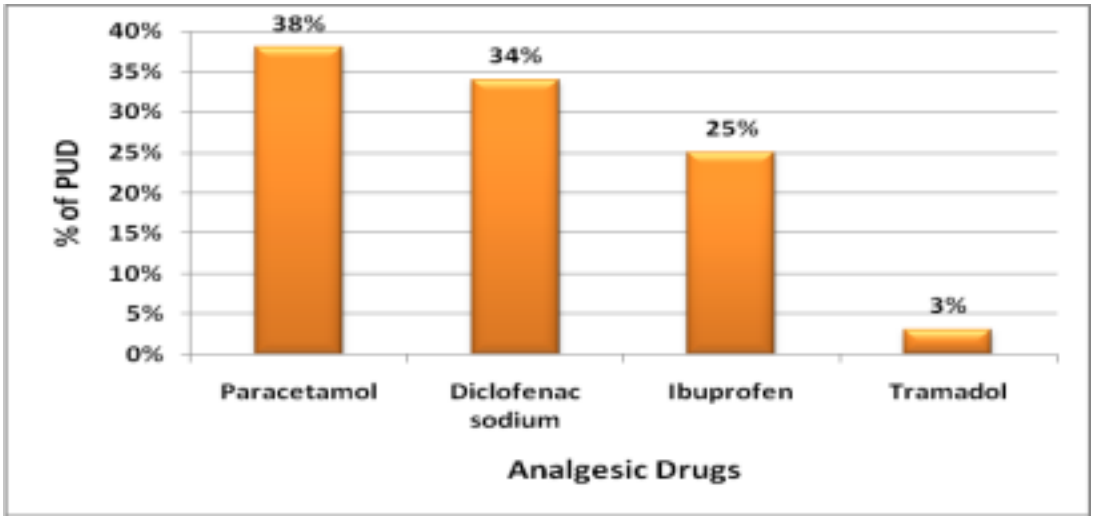

Figure 10, showsthe percentage of analgesics used by the patients.

Figure 10 shows that 38% of respondents used Paracetamol as an analgesic 35% used Diclofenac sodium respectively, 27% used Ibuprofen, and the rest of the participants 2% used Tramadol.

IV. Discussion

Between April 2018 and August 2017, a descriptive study was carried out to determine the prevalence of peptic ulcer disease in PU patients. Patients with abdominal discomfort who were respondents provided detailed information. Data were gathered using a modified, pretested questionnaire. Excel's Statistical Package was used to enter and analyze all of the data.

The higher frequency of risk factors for perforated peptic ulcer illness among our patients may be the cause of the apparent higher incidence of perforated peptic ulcer in the age group between 30 and 45 years, according to the data collected. The increased number of smokers is proof of this.The fact that the majority of our patients are young men and that the risk factors listed above are typical of this age group lends more credence to this; the majority of our patients were in their fourth decade of life and were males (male to female ratio 6:1) [8]. Similar research in poorer nations has also reported this finding [9].A study in Hawaii found that the incidence of duodenal and stomach ulcers rose steadily as pack years of cigarette smoking increased. Sixty-five percent of the participants in this study smoked. Additionally, it was discovered that only 2% of respondents did not exhibit a burning sensation in the epigastric region, whereas the bulk of respondents (98%) did.According to the current study, just 21.4% of respondents experienced worsened pain when their stomach was full, whereas the majority of respondents (78.6%) experienced worsened pain when their stomach was empty. Furthermore, H. Pylori infection, a known cause of peptic ulcers and its consequences, is quite prevalent in Ethiopia. By lowering antral somatostatin, H. pylori infection and the resulting inflammation interfere with the inhibitory regulation of gastrin release; this effect is more pronounced if the cagA-positive strain of the infection is present. One important consequence is the rise in stomach acid output and gastrin release.

The process through which PUD is brought on by an H. pylori infection [10]. Further research is necessary to determine whether the common H. Pylori infection contributes to the higher frequency of perforation and whether it interacts with other known factors for perforation.

The capacity of NSAIDs to induce ulceration and bleeding in the upper gastrointestinal tract was initially noted by Douthwaite and Lintott's 1938 endoscopic investigation [11]. Another study found that NSAIDs, which are frequently used for a range of musculoskeletal issues such as rheumatoid arthritis and the short-term management of pain in osteoarthritis, are linked to both upper and lower gastrointestinal tract disorders [12]. 12.8% of the respondents in this survey did not take painkillers, whereas the majority (87.2%) did.Just 42.3% of respondents did not use aspirin, but the majority (57.7%) did. Additionally, it was discovered that only 3% of respondents did not experience stress, tension, or anxiety, while the majority (97%) did. The majority of study participants (78.6%) reported having had such discomfort in the past, while only 18% reported never experiencing such pain. NSAID use is important in the current investigation. Thus, it is advised to conduct additional research to determine the potential mechanism.

V. Conclusion

The study participants included a diverse range of people with abdominal pain. It was discovered that Libya Hospital's medical facilities were more than capable of offering mass, reasonably priced peptic ulcer disease treatment with impressive success rates. Nonetheless, it was discovered that the disease patients' socioeconomic circumstances and educational attainment were below average. Patients who smoked, had diabetes, high blood pressure, stress, or a family history of the illness were also impacted. These factors, which may include a lack of knowledge about the condition, its effects, and available treatments, maybe the primary causes of the increased prevalence of peptic ulcer disease in developing countries relative to more developed ones.

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