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Prevalence of Peptic Ulcer Disease among Undergraduate Students of Two Universities in Sana'a City

A thesis submitted to the Department of Community Medicine, faculty of Medicine and Health sciences, Emirates international University in partial fulfillment of the requirements for the degree of BBBH in general medicine and surgery.

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الجمهورية اليمنية
نيابة الدراسات العليا والبحث العلمي
الجامعة الإماراتية الدولية
كلية الطب والعلوم الصحية
قسم طب المجتمع

معدل انتشار مرض القرحة الهضمية بين الطلاب الجامعيين في جامعتين في مدينة صنعاء

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Dedication

To

Those whom stood all the way beside us and

Made sure we reach every wish we dreamed

Of our Fathers, Mothers, Brothers and Sisters

And to everyone made us believe in ourselves

And kept supporting us in every way possible,

Our Friends and Family members

Abstract

Background:

Peptic ulcer is one of the most common diseases of the gastrointestinal tract. It affects about 4 million of the world's population annually, with incidence of complications in about 10–20%.Established recent risk factors of PUD such as stress, smoking, and use of coffee and NSAIDs are commonly associated with university students and could impact on the disease prevalence and may result to frequent illness, absenteeism in classes and consequent poor academic performance, also impacts negatively on the health-related quality of life of students .Therefore, determining the prevalence trends of established PUD and associated risk factors in risk populations is essential for clinical and epidemiological decision making.

Objectives:

The current study was designed to study the prevalence, symptoms and risk factors of PUD among medical and non-medical university students.

Method:

Comparative cross-sectional study was conducted in two randomly selected universities in Sana'a city; from April to May 2023 .it included 565 students. A questionnaire was designed for data collection which includes socio-demographic variables, modified risk factors and medical diagnosed peptic ulcer.

Results:

The prevalence of PUD among students in this study was (22.7 %). The results of this study showed a significant relationship between PU and the following factors (specialties, level of educational, smoking status, stress, depression, monthly income), while there was no significant relationship between peptic ulcer with the following (age group, sex, marital status, housing/living, chewing khat, drinking coffee, tea, energy drinks, and taking NSAIDs). The rates of symptoms that suggestive PUD among the students were are change in the color of stool or blood on it (62.5%), nausea or vomiting (56%), heartburn (48.2%), abdominal pain (47%), early satiety (42.4%), abdominal bloating (41.6%), change of appetite (40.5%), and change of weight (36.4%).

Conclusion:

The prevalence of PUD is high among students especially those are related with medical collages. This study also showed that main risk factors for PUD were Stress and smoking. And students of the governmental sector are more prone to get PUD more than privet sector students.

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List of Abbreviations

Ab	**Antibody**
Ag	**Antigen**
BabA	**Blood group antigen binding adhesion**
BPU	**Bleeding peptic ulcer**
CagA	**Cytotoxin associated gene A**
CMV	**Cytomegalovirus**
H. pylori	**Helicobacter pylori**
HSV	**Herpes simplex virus**
IL1B	**Interleukin 1 B**
NSAID	**Non-Steroidal Anti-inflammatory Drugs**
OGDS	**Oesophagogastroduodenoscopy**
OipA	**Outer inflammatory protein adhesion**
OR	**Odds ratio**
***P* value**	**Probability value**
PicB	**Pathogenic island cytotoxin B**
PPU	**Perforated peptic ulcer**
PU	**Peptic ulcer**
PUD	**Peptic ulcer disease**
Pus	**Peptic ulcers**
TLR1	**Toll-like receptor 1**
vacA	**Vacuolating associated cytotoxin A**
χ^2	**Chi-square**

Chapter One

Introduction

1. Introduction

1.1. Background:

Peptic ulcer disease (PUD) is a deep destruction of the stomach lining or mucosa and/or duodenum, reaching beyond the muscularis mucosa, specifically to the muscle layer owing to the environmental gastric acid synthesis (Guerra *et al*, 2022).

PUD is mostly caused by the activities of *Helicobacter pylori* (*H. Pylori*) and/or Non-Steroidal Anti-inflammatory Drugs (NSAIDs) (Russell *et al*, 2001; Rafi *et al*, 2014; Habeeb *et al*, *2020*). There are many established risk factors other than H. pylori and NSAIDs for peptic ulcers (PUs) such as stress, smoking, and corticosteroids use, coffee, spicy foods, fasting, and starvation (Lanza and Chan, 2009; Bashinskaya *et al*, 2011; Etukudo *et al*, 2012; Jemikajah *et al*, 2014; Eniojukan *et al*, 2017). All of these risk factors are commonly associated with undergraduate (Zibima *et al*, 2020).

Globally, PUD is involving 5-10% of the population (Lanas and Chan, 2017). Every year, at least 7 million patients around the world are affected by this disease leads to 100,000 of deaths (Martin *et al*, 2001). The prevalence of both confirmed and unconfirmed cases of PUD on college students at the Nigerian University was 43.2% (Anaemene and Ochogu, 2022); and according to the report of Eniojukan *et al*, 2017(40%) and (60%) prevalence rates of gastric ulcer and duodenal ulcer respectively, in a university community; there are reports supported by (Zibima *et al*, *2020*) increase rates of PUD among university students. Therefore, could impact and may raise the prevalence of PUD. Thus, investigating the prevalence trends and modifiable risk factors of PUD is expedient, especially in a student population; as such information could provide a guide for decision making and development of prevention strategies.

1.2. Study justification

- we have not found studies similar to this one, especially in the world in general and in Yemen in particular. So, our study aims to investigate the prevalence of PUD among undergraduate students of Yemeni universities and identify the risk factors for the affected group of students.
- The risk of PUD and its consequences on the general health of people.

1.3. Research objectives:

The main goal:

To determine the prevalence and risk factors of peptic ulcer disease among undergraduate students.

Sub-objectives:

1. To determine the prevalence rate of PUD among medical students and non-medical students.
2. To determine the main risk factors in the affected group of students.
3. To determine the most important clinical manifestation associated with peptic ulcer disease.

Chapter Two

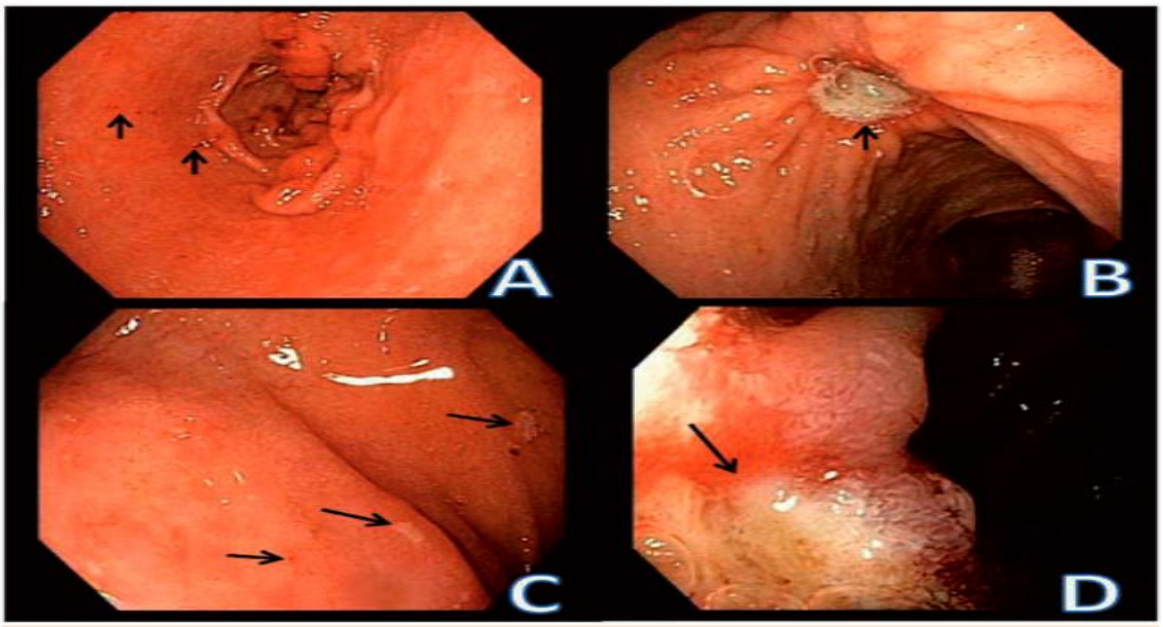
literature review

2. **Literature review:**

2.1. Definition of disease:

Peptic ulcers (PUs) are acid-induced lesions found in the stomach and duodenum characterized by denuded mucosa with the defect extending into the sub mucosa or muscularis propria (Feldman *et al*, 2016). PUD is a common disorder of the digestive system, is defined as digestive tract injury that results in a mucosal break greater than 3–5 mm, a visible depth reaching the submucosa (Lanas & Chan, 2017; Sverdén & Agréus *et al*, 2019). Usually located in the stomach or proximal duodenum, but they can may develop in unusual areas, such as the oesophagus or Meckel's diverticulum (Torpy *et al*, 2012; Del Valle *et al*, 2015; Lanas & Chan, 2017).). In this Seminar, the term PUD refers to PUs located in the stomach or duodenum.

Ulcers in the stomach or duodenum may be acute or chronic; both penetrate the muscularis mucosae but the acute ulcer shows no evidence of fibrosis. Erosions do not penetrate the muscularis mucosae (Davidson's 22th edition). Gastric ulcers are most commonly located on the lesser curvature, whereas duodenal ulcers are most common at the duodenal bulb. The ulcer is round to oval with a smooth base. Acute ulcers have regular borders, while chronic ulcers have elevated borders with inflammation. An ulcer extends beyond the muscularis mucosa **(Fig. 2.1.)** (Malik *et al*, 2023).



A. Small erosion in the gastric antrum. Mucosal breaks with focal hemorrhage are identified by the arrows. **B**. Benign ulcer in the body of the stomach (arrows). **C** duodenal erosions identified by focal areas of adherent exudate (arrows). **D**uodenal ulcer. The mucosal defect has depth and the margin is identified by the arrow. The surrounding mucosa is edematous.

2.2. Incidence and prevalence:

Globally, PUD is involving 5-10% of the population (Lanas and Chan, 2017). The lifetime prevalence of PUD is approximately 10%, affecting about 4.5 million people annually in the United States alone (Prasad & Friedman, 2018; Lanas and Chan, 2017). Peptic ulcer (PU) is a major problem of modern society. In the United States of America, there are about 500,000 new cases per year of PU and 4 million ulcer recurrences. (Stephen *et al*, 2008). Over 40,000 people annually in the USA have surgery each year because of persistent symptoms of problem and 6,000 people die from related complication (CDC, 2001). In the universal counterpart, each year there is at least 7 million patients around the world who are affected by this disease leads to hundreds of thousands of deaths (Martin *et al*, 2001). The number of patients with asymptomatic PUD is unknown. Many patients with uncomplicated PUD are treated empirically without an endoscopically confirmed

diagnosis, which affects epidemiological studies carried out and explains fluctuation in reported results (Malmi, 2018).

The prevalence of endoscopically confirmed PUD was 4.1% in the general adult population in Sweden in the Kalixanda cross-sectional study during 1998-2001 (Aro et al. 2006). Of these patients, 81% reported PUD-related symptoms, whereas the others were asymptomatic. In other population-based studies from Europe and the USA, the one-year prevalence of PUD has ranged from 0.1% to 1.5% (Sung et al. 2009). However, in a recent study from Asia, the prevalence of asymptomatic PUD diagnosed endoscopically in the Taiwanese population was 9.4% (Wang et al. 2011). In another study from China, the prevalence of PUD among a randomly selected cohort of adults was 17% with most patients (72%) being asymptomatic (Li et al. 2010).

Many studies showed that females were more affected of PUD by (72%) than males (Eniojukan *et al*, 2017; Zibima *et al*, 2020). On studise, incidence of PUD in the Men is three times more than women (Gerard *et al*, 2006). These statistics show that the prevalence of PU in the world has different and heterogeneous reports. Therefore the occurrence PUD depends on risk factors not gender. Prevalence of *H.pylori* related PUD was higher among female students of an Iraq University (Hussen et al. 2013). Females were found to be more associated with modifiable risk factors of starvation, stress, and depression than their male counterparts. Though the proportion of females associated with the use of NSAIDS, alcohol and smoking were less than males; the difference in magnitude was minimal and therefore, could play a role in increasing their risk for PUD (Zibima *et al, 2020*).

Incidence of PUD increases with age, with most ulcers occurring between 25 and 64 years of age (Akhtar, Shelton, & Dinh, 2019). Overall, there is a decrease in the incidence of PUD worldwide due to improve d hygienic and sanitary conditions combined with

effective treatment and judicious use of NSAIDs (Lanas *et al*, 2017); Despite reports indicating a decrease in the incidence of PU, especially in countries that are highly developed economically, this disease still remains one of the most important problems, in the practice of both primary health care physicians and gastroenterologists (Bernersen *et al*, 1990; Everhart *et al*, 1998; *Kang et al*, 2006; Nervi *et al*, 2006).

Incidences increased among peoples have stress life's. The prevalence of both confirmed and unconfirmed cases of PUD by cross- sectional survey on Undergraduate Students at the Nigerian University was 43.2%; prevalence of medically diagnosed cases was 7.9% (Anaemene and Ochogu, 2022), While gastric ulcer was 40% and 60% of duodenal ulcer (Eniojukan *et al*, 2017). University education is usually viewed as stressful. This supported by (Zibima *et al*, *2020*) increase rates of PUD among university students

2.3. Pathogenic mechanisms:

Mucosal disruption in patients with acid peptic disease can be due to infection, barrier disruption, or gastric acid hypersecretion. Risk factors for developing PUD include *Helicobacter pylori* (*H. pylori*) infection, alcohol consumption, tobacco use, cocaine and amphetamine use, (NSAIDs), fasting, Zollinger-Ellison syndrome, cancer treatment with angiogenesis inhibitors and Bariatric surgery **(Fig. 2.2)** (Søreide *et al*, 2015).

H. pylori and the use of NSAIDs or aspirin are the main risk factors of both gastric and duodenal ulcers (Huang *et al*, 2002; Sugimoto *et al*, 2009; Zhang *et al*, 2014; Del Valle *et al*, 2015). However, only a few people with *H. pylori* infection or taking NSAIDs or aspirin develop PUD, suggesting that individual susceptibility to bacterial virulence and drug toxicity is essential to the initiation of mucosal damage. The interaction between bacterial and host factors determines the outcome of *H. pylori* infection. The ability of *H. pylori* strains to produce different proteins has been linked to their virulence and to the host immune response (Del Valle *et al*, 2015).

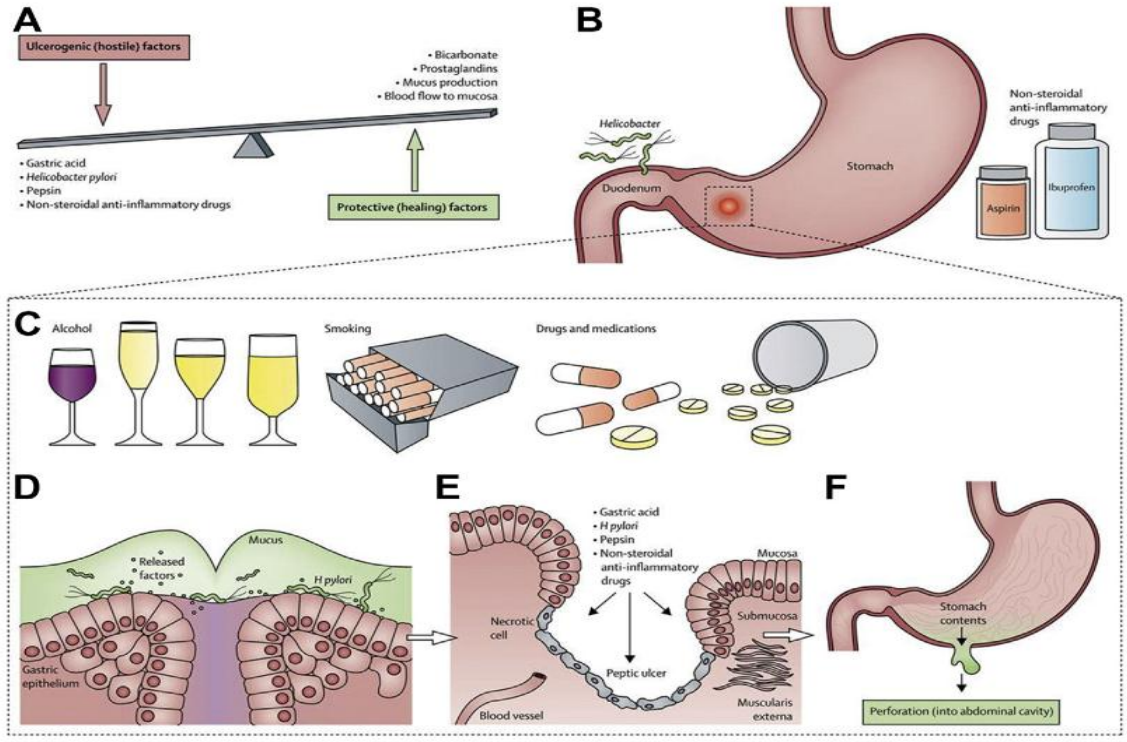


Fig.2.2. **Mechanisms and factors in pathogenesis of perforated peptic ulcer.** *(A) An imbalance between hostile and protective factors start the ulcerogenic process, and (**B**) although man contributors are known, Helicobacter infection and use of NSAIDs appear of importance in disturbing the protective mucosal layer and (**C**) expose the gastric epithelium to acid. Several additional factors (**D**) may augment the ulcerogenic process (such as smoking, alcohol use, and use of several drugs) that leads to erosion (**E**). Eventually, the serosal lining is breached (**F**), and when perforated, the stomach content, including acidic fluid, will enter the abdominal cavity, giving rise to intense pain, local peritonitis that may become generalized, and eventually lead to a systemic inflammatory response syndrome and sepsis with the risk of multiorgan failure and mortality.*

The organism produces urease to create an alkaline environment, which is essential for its survival in the stomach under the mucosal barrier. It also expresses adhesins such as blood group antigen adhesin (BabA) or outer inflammatory protein adhesin (OipA), which facilitate attachment of bacteria to gastric epithelium. A genome pathogenic island encodes the virulent factors CagA and PicB, which—together with other bacterial factors—are thought to interact strongly with host tissue and be linked to gastric mucosal inflammatory cell infiltration and gastric epithelial injury (Yamaoka *et al*, 2014; Datta *et al*, 2015). Almost all *H. pylori* strains contain the vacA gene, which encodes a vacuolating cytotoxin,

although half the strains do not express the protein. The role of VacA protein in disease pathogenesis is unclear. Variations in the vacA gene structure (i.e., a combination of signal sequence allelic types [s1a, 1b, and 2] and mid-region allelic types [m1 and m2] might have functional implications. Most cagA-positive strains carry the vacA-s1 genotypes, whereas almost all cagA-negative strains are classified as vacA s2/m2 strains with low cytokine response and host interaction, which could have clinical consequences (Chen *et al*, 2013; Datta *et al*, 2015). Host interaction and the mucosal inflammatory response to *H. pylori* can be determined, at least in part, genetically and define the outcome of PU and other acid-related diseases (Datta *et al*, 2015). Functional polymorphisms in different cytokine genes have been related to PUD (Robert *et al*, 1991; Furuta *et al*, 2002; Datta *et al*, 2015). Interleukin 1β, encoded by IL1B, is a cytokine associated with the inflammatory response to *H. pylori* infection and with inhibition of gastric secretion. Polymorphisms of IL1B affect mucosal interleukin 1β production in diverse populations, suggesting that these polymorphisms have a role in the pathogenesis of *H. pylori* associated gastroduodenal diseases, including PUD. Genes encoding tumour necrosis factor and lymphotoxin-α have also proved to be associated with duodenal ulcers, gastric ulcers, and antral inflammation caused by *H. pylori* infection (Lanas and Santolaria *et al*, 2001; Zhang *et al*, 2013). A genome-wide association study and meta-analysis identified an association between the locus of Toll-like receptor 1 (TLR1) and *H. pylori* seroprevalence in a white population with European ancestry. Other cytokines have also been linked to the pathogenesis of *H. pylori*-induced diseases, but their role is unclear.

NSAIDs and aspirin are the other major risk factors linked to PUD and its complications. Compared with non-users, NSAID and aspirin use increase the risk of complications of PUD by four times in NSAID users, and by two times in aspirin users. As well as NSAID and aspirin use or H pylori infection, complications are largely driven by

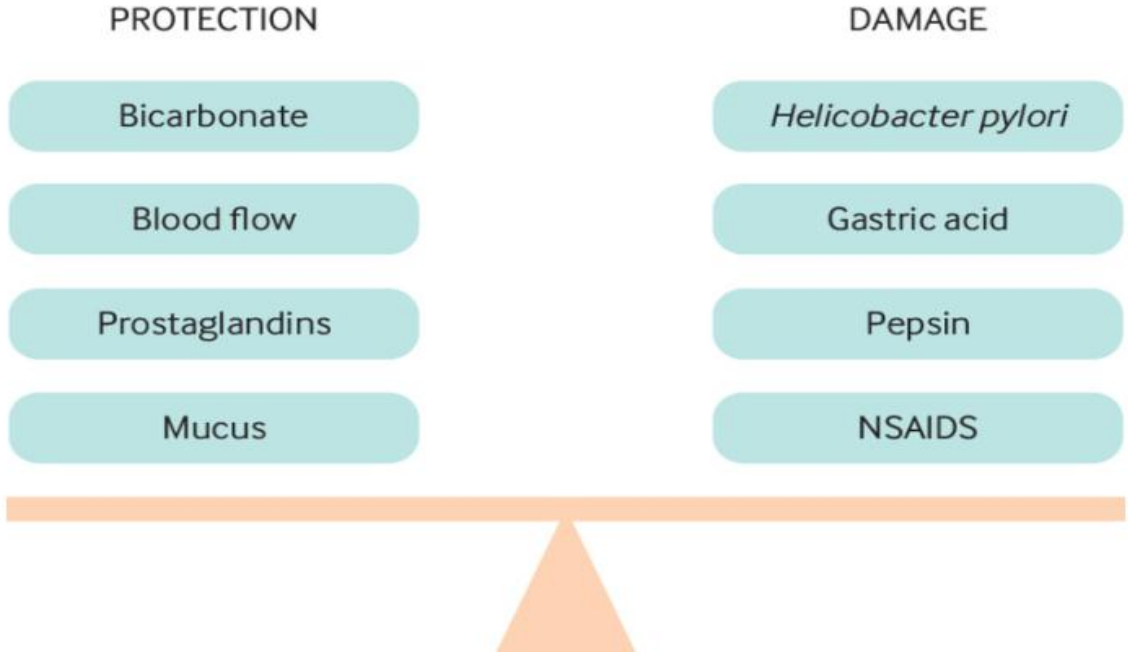
comorbidity and ageing. Concomitant use of NSAIDs or aspirin with selective serotonin-reuptake inhibitors, corticosteroids, aldosterone antagonists, or anticoagulants substantially increases the risk of upper gastrointestinal bleeding. The role of smoking and poor socioeconomic status is unclear. Studies have shown a link between aspirin use and an increased risk of PUD in patients carrying some specific genetic polymorphisms, but the clinical relevance of these studies also remains to be determined (Tanikawa *et al*, 2012; Shiotani *et al*, 2015). Many people who habitually take NSAIDs or aspirin have concurrent *H. pylori* infection. The interaction of these two factors in PUD is controversial. Randomized controlled trials have shown that eradication of *H. pylori* is beneficial in patients who start taking *NSAIDs* but not in those who are on long-term NSAID treatment. A meta-analysis of observational studies found that uncomplicated PUD was more common in *H pylori*-positive patients than in *H. pylori*-negative patients (odds ratio [OR] 2·12, 95% CI 1·68−2·67). The interpretation of this meta-analysis was that both *H. pylori* infection and the use of NSAIDs and aspirin independently increase the risk of PUD, and that the disease was uncommon in patients without *H. pylori* infection who do not take NSAIDs or aspirin (Gisbert et *al*, 2009; Sostres *et al*, 2015). *H. pylori*-negative, NSAID-negative, and aspirin-negative PUD can be diagnosed in at least a fifth of cases. Life-threatening conditions could also induce the disease. For example, soon after the Great East Japan Earthquake in 2011, an unusual increase in cases of *H. pylori*-negative haemorrhagic multiple PUDs were recorded. Accommodation in a refugee shelter was a strong risk factor for peptic ulcer bleeding (PUB) after a large-scale disaster. A Danish study showed that psychological stress was associated with an increased incidence, in part by influencing health risk behaviors, and had similar effects on the disease related or unrelated to either *H. pylori* or NSAIDs. The true prevalence of idiopathic PUD not related to NSAIDs or *H. pylori* infection is unknown because a molecular investigation of patients

with chronic gastritis but negative for *H. pylori* showed that almost half were false negatives (Kiss et *al*, 2016).

2.4. Etiology & risk factors:

PUD develops when the protective mechanisms of the gastrointestinal mucosa, such as mucus and bicarbonate secretion, are overpowered by the damaging effects of gastric acid and pepsin **(Fig. 2.3)** (Debruyne *et al*, 2016; Shah *et al*, 2019). The main risk factors for PUD are *H. pylori* and NSAID use, however not all individuals infected with *H. pylori* or taking NSAIDs develop PUD (Zhang *et al*, 2014; Feldman *et al*, 2016).

(Fig. 2.3) Factors that protect or harm the gastroduodenal mucosa



H. *pylorus is* a commonest cause of PUD. It is a gram-negative bacillus that is found within the gastric epithelial cells; the organism has a wide spectrum of virulence factors allowing it to adhere to and inflame the gastric mucosa. This results in hypochlorhydria or achlorhydria, leading to gastric ulceration (Malik et *al*, 2023). A unique bacteria that is ideally suited to live in the acidic environment of the human stomach (Peura *et al*, 2010). Person-to-person transmission of bacteria from fecal-oral, oral-oral, or gastric-oral exposure seems the most probable explanation for infection (Brown, 2000; Amieva & El-

Omar, 2008). Especially in developing countries, contaminated water might serve as an environmental source of bacteria because the organism can remain viable for several days in water (Bellack *et al*, 2006). Iatrogenic infection has occurred during the use of a variety of inadequately disinfected gastric devices, endoscopes, and endoscopic accessories (Brown, 2000). Gastroenterologists and nurses appear to be at greater risk for acquiring *H pylori*, presumably because of occupational contact with infected gastric secretions (De Schryver *et al*, 2004), although this is less likely to occur when universal precautions for infection control in the health care setting are strictly enforced. This bacterium is responsible for 90% of duodenal ulcers and 70% to 90% of gastric ulcers. *H. pylori* infection is more prevalent among those with lower socioeconomic status and is commonly acquired during childhood (Malik et *al*, 2023). *H. pylori* are one of the most common chronic bacterial infections in humans, with more than 50% of the world's population infected with these bacteria. (Peura *et al*, 2010) Genetic sequence analysis has proposed that humans have been infected with *H. pylori* for more than 58,000 years (Linz *et al*, 2007), While *H. pylori* have been demonstrated worldwide in individuals of all ages, infection is commonly acquired at an earlier age in developing countries as compared with industrialized nations (Brown, 2000; Kivi *et al*, 2006). In older children and adults, infection persists so that in the developing areas of the world the overall *H. pylori* prevalence can reach more than 80% in individuals older than 50 years (Peura *et al*, 2010).

NSAIDs use is the second most common cause of PUD after *H. pylori* infection (Huang *et al*, 2002; Lanas *et al*, 2015). The use of NSAIDs in Finland has increased significantly from the 1990s, as well as in other countries (Bardhan *et al*, 2004; Pérez-Aisa *et al*, 2005; Kang *et al*, 2006). In developed countries, one fourth of elderly patients use NSAIDs (Barat *et al*, 2000; Turunen *et al*, 2005; Sayer *et al*, 2010).

Other factors implicated include the aspirin (Kainat *et al*, 2020), and blood group O (Ray-Offor and Opusunju, 2020), physical or emotional stress and starvation (Zibima *et al*, 2020), Cigarette smoking was a common factor among those diagnosed with ulcer and alcohol (Eniojukan *et al*, 2017). Smoking also appears to play a role in duodenal ulcers, but the correlation is not linear. Alcohol can irritate the gastric mucosa and induce acidity (Malik *et al*, 2023), smoking is a risk factor for chronic active ulcers or asymptomatic PUD in the United States (Everhart *et al*, 1998), Taiwan (Wang *et al*, 2011), Denmark (Rosenstock *et al*, 2003), and Norway (Bernersen *et al*, 1996), and in American men of Japanese ancestry (Kato *et al*, 1992). Alcohol intake was also associated with PUD (Battaglia *et al*, 1984; Andersen *et al*, 2000; Bayyurt *et al*, 2007; Razvodovsky *et al*, 2007; Salih *et al*, 2007). A study found out that people who had irregular meal timing particularly deviating more than two hours had higher risk of developing gastritis and *H. pylori* infection by thirteen-fold compared to those who ate regularly (Lim *et al*, 2013).
A research on coffee as one of the risk factors of PUD is inconsistent (Zaman *et al*, 2019).

Stress owing to serious health challenges such as those needing management in an intensive care unit is well characterized as an antecedent of PUs, which are termed stress ulcers (Alves *et al*, 2020). When a person affected is by stress he may also smoke more, sleep less and take NSAIDs thereby increasing their susceptibility to ulcer by mechanisms that are related to acidity. Apart from the activities of *H. pylori* and *NSAIDs*, studies have identified risk factors of PUD which are mostly modifiable. The modifiable risk factors include the use of corticosteroids, anticoagulants, coffee, alcohol, smoking, stress, spicy foods, use of unclean water sources and fasting; while non-modifiable risk factors include genetics, age, gender and past history of PUD (Bateson *et al*, 1993; Lanza *et al*, 2009; Bashinskaya *et al*, 2011; Etukudo *et al*, 2012; Jemikajah *et al*, 2014; Eniojukan *et al*, 2017). All of these modifiable risk factors are commonly associated with undergraduate.

University education is usually viewed as stressful. This is because full time students are commonly scheduled for academic activities from 8am-5pm, and have to attend to assignments and personal study during the night. When such activities are routinely observed, there is tendency for stress levels to increase and impact negatively on the gastrointestinal system (Zibima *et al*, 2020).

About a fifth of *PUD* cases are not related to *H. pylori*, *NSAIDs* or aspirin, but the accuracy of this value has been challenged due to false negative *H. pylori* testing or accidental (or underreported) NSAID ingestion (Kanno *et al*, 2015; Kiss *et al*, 2016). This idiopathic PUD may be due to an imbalance between factors that contribute to mucosal integrity and aggressive insults, including a hypersecretory status, Other etiologies for PUD include ischemia causing stress ulcers, medications (steroids, alendronate, potassium chloride, and chemotherapeutic agents), viral infections (CMV, HSV), gastric bypass surgery, metabolic disturbances, radiotherapy, histamine, eosinophilic infltration, and basophilia (McColl, 2009; Lanas & Chan, 2017).

A low socioeconomic status is commonly considered a risk factor for peptic ulceration, although this is, in part, because individuals with low status are more likely to face shift work and stress at work and at home, with a greater probability of smoking, an excessive consumption of alcohol, and *H. pylori* infection. Individuals with low socioeconomic status may still have a physically demanding job but from a young age their leisure activity is substantially lower than that of the upper echelons of society (Shephard *et al*, 1996; Drenowatz *et al*, 2010). Similar to low socioeconomic status, Low education level is associated with PUD because education level was related to living conditions, such as lifestyle, diet, and social stress and these conditions are part of the multifactorial etiology of PUD (Johnsen *et al*, 1994; Wang *et al*, 2011). Numerous studies of sociodemographic characteristics and PUs identified various risk factors, such as age,

household member crowding, unemployment, marital strain, a blue-collar household, , breakfast skipping, high body mass index (BMI), musculoskeletal pain, headache, psychological and physical stress, and previous PUs (Yim *et al*, 2021). Psychological stress and physical stress affect the development of ulcers because stress aggravates gastro duodenal blood flow, reduces acid buffering in the duodenum, and diminishes gastric hypersecretion (Levenstein & Kaplan, 1998; Levenstein *et al*, 1999). Stress tends to be uncontrolled and unpredictable (Koolhaas *et al*, 2011; Overmier *et al*, 2013), promotes the onset of disease (Levenstein *et al*, 1995; Levenstein, 1998), and is one of the most common risk factors for PUD (Bernersen *et al*, 1996).

2.5. Clinical presentation:

A prospective study of patients in Taiwan undergoing a screening upper endoscopy as part of routine health maintenance determined that approximately two-thirds of those found to have PUD are asymptomatic (Lu *et al*, 2004). Among symptomatic patients with PUD, the most common presenting symptom is epigastric pain, which may be associated with dyspepsia, bloating, abdominal fullness, nausea, and early satiety (Malfertheiner *et al*, 2009) as shown in **(Fig. 2.4).** The clinical manifestation can be divided into three phases. In the initial phase within 2 h of onset, epigastric pain, tachycardia and cool extremities are characteristic. In the second phase (within 2 to 12 h), pain becomes generalized and is worse on movement. Typical signs such as abdominal rigidity and right lower quadrant tenderness (as a result of fuid tracking along the right paracolic gutter) may be seen. In the third phase (more than 12 h), abdominal distension, pyrexia and hypotension with acute circulatory collapse may be evident (Silen, 1996). A study involving 84 patients with PPU reported that the commonest presenting symptoms were sudden onset of severe epigastric pain (97.6%), abdominal distention (76.2%) and vomiting (36.9%). Abdominal tenderness and classical signs of peritonitis could be elicited in 88.1% and 66.7% of the patients with

PPU in this study. Other symptoms also included nausea (35.7%), severe dyspepsia (33.3%), constipation (29.8%) and fever (21.4%) (Chalya *et al*, 2011). In our experience of managing 332 patients with PPU, the most common presenting symptom was acute onset of abdominal pain (61.7%) (Anbalakan *et al*, 2015).

Patients who present with warning symptoms or alarm symptoms should be require more invasive forms of evaluation and prompt urgent referral that include: unintentional weight loss, progressive dysphagia, overt gastrointestinal bleeding, iron deficiency anemia, recurrent emesis, and family history of upper gastrointestinal malignancy (Banerjee *et al*, 2010).

Fig.2.4. Symptoms of peptic ulcer disease

2.6. Differential diagnosis:

Acute GI bleeding is one of the most common medical emergencies leading to hospitalization with notable costs (Peery *et al*, 2015). The overall incidence of upper GI bleeding that diagnosed by Oesophagogastroduodenoscopy (OEGD) has varied from 45 per 100 000 inhabitants per year to 160 per 100 000 (Vreeburg *et al*, 1997; Paspatis *et al*,

2000; Ahsberg *et al*, 2010; Button *et al*, 2011). Most patients are referred for acute OEDG for diagnosis **table (2.1).**

Etiology	Vreeburg et al. (1997) %	Leerdam et al. (2003) %	Loperfido et al. (2009) %	Nahon et al. (2012) %	Miilunpoh al. (2017) %
PUD	39	46	53	31	50
Erosive	6	--	10	7	--
Mallory-Weiss	5	--	3	7	8
Variceal bleeding	8	7	12	21	8
Oesophagitis	7	--	4	13	17
Gastroduodenal lesions*	--	20	--	--	--
Neoplasm	3	5	5	3	4
Other	8	8	8	13	--
No finding	24	14	5	5	13

Table 2.1. Etiology (%) of acute upper gastrointestinal bleeding

The patients presenting with acute upper gastrointestinal bleeding with no finding in OEGD the proportion of patients with acute GI bleeding symptoms without a known source in OEGD has varied from 5% (Nahon *et al*, 2012) to 32% (Sengupta *et al*, 2016) in previous studies. The most common sources for acute lower GI bleeding in the previous studies were diverticulosis, ischemic colitis, hemorrhoids and colon cancer (Longstreth 1997; Arroja *et al*, 2011; Hreinsson *et al*, 2013). In those lower GI bleeding studies, no source for bleeding was diagnosed in 8-12% of patients.

A bleeding ulcer can be the first symptom of gastric cancer. The *H. pylori* infection is a risk factor for both PUD and gastric cancer (Tsuda *et al*, 2017).

2.7. Complications:

Gastrointestinal bleeding is the most widely recognized complication. Sudden excess amount of bleeding can be life-threatening (Cullen *et al*, 1997). It is related with 5% to

10% death rate (Lanas *et al*, 2017). BPU occurs in 19 to 57 per 100 000 individuals each year, as per a systematic review (93 studies). Perforation or penetration is relatively less common, occurring in 4 to 14 per 100 000 individuals each year. Mortality is high with these complications. About 8.6% of patients with BPU and 23.5% of patients with perforation die within 30 days (Lau et *al*, 2011).

Perforated peptic ulcer (PPU) is a hole in the wall of the gastrointestinal tract following a gastric ulcer regularly prompts disastrous outcomes whenever left untreated. Perforation is a serious complication of PUD and patients with PPU often present with acute abdomen that carries high risk for morbidity and mortality (Bas *et al*, 2008). The lifetime prevalence of perforation in patients with PUD is about 5% (Vaira *et al*, 1998). PPU carries a mortality ranging from 1.3% to 20% (Boey *et al*, 1987; Hermansson *et al*, 1999; Rajesh *et al*, 2003). Thirty-day mortality rate reaching 20% and 90-day mortality rate of up to 30% have been reported (Buck *et al*, 2014; Søreide *et al*, 2014). Although bleeding is the most common complication (ratio of 6:1), perforation carries the highest mortality risk of up to 30% (Søreide *et al*, 2015). Erosion of the gastrointestinal wall by the ulcer prompts to spillage of the stomach or intestinal substance into the abdominal cavity. Perforation at the anterior surface of the stomach prompts to acute peritonitis, initially chemical and later bacterial peritonitis. The primary sign is often sudden extreme abdominal pain. The death rate in this case is 20%. Penetration is a form of perforation in where in the hole prompts and the ulcer proceed into adjacent organs like as the liver and pancreas.

Cancer is included in the differential diagnosis (explained by biopsy); *H. pylori* as the etiological factor chance it 3 to 6 times more likely to develop stomach cancer from the ulcer (Merck Manuals, 2006).

Gastric outlet obstructions are a narrowing of the pyloric canal by scarring and swelling of the gastric antrum and duodenum because of PUs. The person often presents with serious vomiting (Lanas *et al*, 2017).

The risk of recurrence and complications from idiopathic ulcers is higher than for ulcers with known aetiology, as reported in prospective cohort studies (Hung *et al*, 2005;Wong *et al*,2009).

2.8. Diagnostic workup:

Diagnosis of PUD begins with clinical suspicion when patients present with symptoms such as epigastric abdominal pain, burning, post-prandial fullness, or early satiety (Feldman *et al*, 2016). Classically, patients with duodenal ulcers complain of worsening abdominal pain on an empty stomach and describe hunger or abdominal pain two to three hours after meals or at night. In contrast, patients with gastric ulcers report nausea, vomiting, weight loss and post-prandial abdominal pain. Elderly patients are often minimally symptomatic and some patients with untreated PUD may have intermittent symptoms due to spontaneous healing and then relapse due to persistence of risk factors, such as continued NSAIDs use or *H. pylori* infection (Lanas *et al*, 2017). With regard to history of duodenal ulcers it is may occur in any age group. However, they are most commonly diagnosed in patients aged 20 to 45. Most patients will have a history of presenting symptoms consistent with PUD associated with a previous diagnosis of *H. pylori* and/or heavy NSAID use. Other elements of the history to consider include smoking history, daily aspirin use, and history of GI malignancy. On physical examination, patients may have epigastric abdominal tenderness, and if presenting with complications, they may demonstrate signs of anemia such as pale skin and positive fecal occult blood test (Ocasio Quinones & Woolf, 2022).

In most patients with uncomplicated PUD, routine laboratory tests are often unhelpful (Prasad & Friedman, 2018). Although a complete blood cell count can exclude anemia, there is no reliable blood test to confirm PUD. In addition, the following tests may aid in PUD workup: Blood chemistries for assessment of electrolyte levels and liver function; Fecal occult blood test to rule out GI bleeding; Testing for *H. pylori* through stool antigen test, urea breath test, rapid urease test, serology, or histology: Stool antigen is the most accurate test for diagnosing *H. pylori* with 96% sensitivity, 83% specificity, and 91% accuracy compared with serology, which has 50% sensitivity, 54% specificity, and 52% accuracy (Kazemi *et al*, 2011).

Endoscopy is the gold standard for PUD diagnosis and remains the most accurate PUD diagnostic test. Because *H. pylori* infection is the causative factor of most types of PUD, a test-and-treat strategy with a noninvasive test to exclude infection is recommended to rule out infection in patients younger than 55 years (Moayyedi *et al*, 2017), who present with no alarm symptoms in geographical areas where gastric carcinoma is uncommon and *H. pylori* prevalence is greater than 20%. Geographic prevalence of *H. pylori* varies worldwide depending on the socioeconomic status and sanitation conditions, with prevalence of less than 40% in developed countries and more than 80% in developing countries (Kazemi *et al*, 2011). In older patients, endoscopy is recommended to rule out PUD (Lanas & Chan, 2017). Although GI symptoms are common in children, PUD is relatively rare (Fashner & Gitu, 2015). Diagnosis test for *H. pylori* could be noninvasive methods such as stool antigen test and blood antigen test. However, the most accurate methods to detect *H. pylori* are invasive approaches that require histological examination after endoscopic biopsy as well as rapid urease test and microbial culture (Karami *et al*, 2013; Mousavi *et al*, 2013). Upper endoscopy can be used to diagnose PUD and is of particular urgency in those with dyspepsia and concurrent alarm symptoms (e.g. age >60

years, family history of upper gastrointestinal tract malignancy, weight loss, early satiety, dysphagia, gastrointestinal bleeding, iron deficiency anemia, or vomiting) (Talley *et al*, 2005; Allen *et al*, 2015). There are both invasive and non-invasive methods for testing that are summarized in **Table (2.2)**. Of all the noninvasive methods, the urea breath test and stool antigen tests are the most feasible and are more accurate than serologic testing (Chey *et al*, 2007).

Test	Sensitivity	Specificity	Advantages	Disadvantages
Serology	85-92%	79-83%	Only test not influenced by PPI or antibiotic use	Cannot confirm cure
Urea Breath Test	95%	96%	Confirms cure	Accuracy affected by PPI and antibiotic use
Fecal antigen testing	95%	94%	Confirms cure	Accuracy affected by PPI and antibiotic use
Rapid urease test	98%	99%	Inexpensive, confirms cure	Requires endoscopy, less accurate after treatment or after PPI use
Histology	>95%	>95%	Permits visualization, confirms cure	Requires endoscopy, affected by PPI and antibiotic use
Culture	70-90%	100%	Allows determination of antimicrobial sensitivity, confirms cure	Requires endoscopy, result takes several days, affected by PPI and antibiotic use

PPI, proton pump inhibitor

Table 2.2. Diagnostic Tests for *H. pylori*

2.9. Management:

Treatment is usually directed at identifying the factors that lead to PUD. For *H. pylori*-associated PUD, eradication alone will lead to ulcer healing and prevent further mucosal injury. However, due to rising antibiotic resistance in *H. pylori*, treatment has become more difficult. First line therapy for *H. pylori* eradication includes a proton pump inhibitor (PPI), clarithromycin and amoxicillin or metronidazole (for penicillin-allergic patients) for seven to 14 days. Due to increasing antibiotic resistance, the efficacy of triple therapy has fallen below 70% in many countries. As susceptibility testing is often not available in clinical practice, clarithromycin-based regimens should be avoided when local clarithromycin resistance rates are greater than 15% (Malfertheiner *et al*, 2017). PPIs work

synergistically with antibiotics to eradicate *H. pylori* (Strand *et al*, 2017). For areas with high clarithromycin resistance, bismuth-containing quadruple therapy with a PPI, bismuth, tetracycline and a nitroimidazole (metronidazole or tinidazole) for 14 days or PPI, clarithromycin, amoxicillin, and a nitroimidazole for 14 days is the preferred as first line treatment (Chey *et al*, 2017). There have been issues with the cost and availability of tetracycline and the data have been mixed on whether doxycycline can be substituted. The regimens discussed above yield eradication rates greater than 90% (Lanas *et al*, 2017). All patients treated for *H. pylori*, should be tested to confirm eradication at least four weeks after completing therapy. Second line therapy (**Fig.2.5**) should be prescribed if a first line regimen fails and should not include repeating metronidazole or clarithromycin (Chey *et al*, 2017).

In NSAID- or aspirin-associated PUD, ulcers heal more than 85% of the time with 6-8 weeks of PPI therapy if the offending agent is discontinued. Ulcer healing is still attainable but delayed with continued NSAIDs use. Anti-secretory therapy can be started for prevention of PUD in patients on aspirin. Although PPIs, H2 blockers, sucralfate, and misoprostol can all be considered to treat NSAID-associated PUD, PPIs are far more effective than other agents (Strand *et al*, 2017). Sucralfate is effective for treating NSAID-associated duodenal ulcers but not for the treatment or prevention of NSAID-associated gastric ulcers (Lanas *et al*, 1995). BPU account for 40−60% of all causes of acute upper gastrointestinal bleeding (Lanas and Polo-Tomás *et al*, 2011). Some patients may require long term acid suppression if using ulcerogenic drugs for a longer duration (**Box 2.1**). Patients are often uncertain of the reason for long term treatment with PPIs and may not be aware that NSAIDs and aspirin can cause PU. Educate patients about these risks so they are compliant with the treatment.

(Box 2.1): Indications for long term therapy with a PPI in long term users of aspirin or *NSAIDs* (Lanza *et al*, 2009)
• Age >65 years
• A history of PUD, especially with complications
• NSAID use at high doses or in combination with certain other drugs, i.e., aspirin, steroids, selective serotonin reuptake inhibitors, or anticoagulants
• Aspirin use, even at low dosage in elderly patients, particularly in combination with drugs listed above

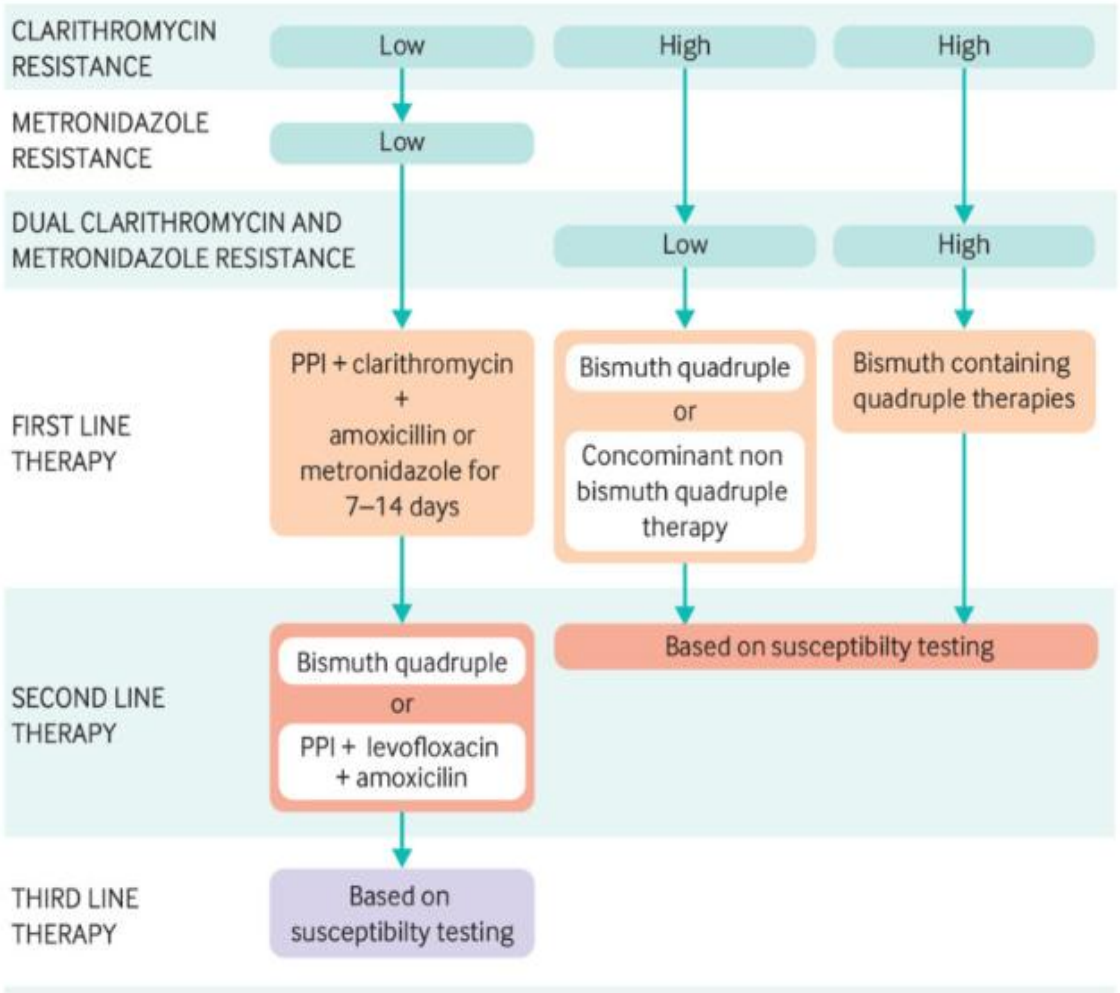
Histamine 2 receptor antagonists are effective in preventing duodenal ulcers among NSAID users, but not gastric ulcers (Rostom *et al*, 2002). These have a shorter duration of action and do not completely suppress postprandial secretion of gastric acid, (Colin-Jones *et al*, 1995) which requires at least twice-daily dosage. Randomised trials and cohort studies have shown that high doses of famotidine (80 g daily) prevent gastric ulcers, although not as effectively as PPIs (Wong *et al*, 2010).

Timely endoscopic treatment and acid suppressive therapy are key for Successful outcomes. Although surgery is the cornerstone for management of patients with uncontrolled or massive recurrent bleeding, radiological intervention has also gained importance in recent years; Patients presenting with upper gastrointestinal bleeding should be assessed promptly and resuscitation should begin with crystalloid solutions. Transfusion policy should be restrictive and aimed to maintain haemoglobin concentrations over 70 g/L, as this approach has been associated with reduced mortality (Villanueva *et al*, 2013). In patients with BPU, endoscopic treatment reduces the risk of re-bleeding, the need for surgery, and mortality (Hearnshaw *et al*, 2007; Gralnek *et al*, 2015). Approximately 10% of patients require urgent angiographic embolisation or surgery for bleeding despite endoscopic intervention (Sverdén *et al*, 2019). The gold standard treatment of ulcer perforation is surgery. Endoscopic stenting plus drainage is a less invasive alternative, but

its role is debated (Chung *et al*, 2017). Pyloric obstruction is typically managed endoscopically with dilatation, although surgery is sometimes required
(Heo *et al*, 2014).

2.10. Follow up:

Ask the patient about improvement in symptoms. Assess outcome of eradication therapy, preferably non-invasively, eg, by a urea breath test or a stool antigen test, at least 2 weeks after finishing the PPI therapy. More than 85% of patients experience eradication with good compliance to treatment when the prescription is appropriate for the local resistance pattern. Discuss elimination of other risk factors mainly NSAIDS and smoking. Patients with a confirmed endoscopic diagnosis of duodenal ulcer do not require follow-up after eradication. Patients with gastric ulcers will need repeat endoscopies and biopsies until confirmed healed, mainly because such ulcers are slower to heal and some may actually be gastric cancers misdiagnosed as an ulcer. Continue PPI treatment after eradication for up to 8 weeks in total or until healing is endoscopically confirmed (Tulassay *et al*, 2008). Of note, a malignant ulcer can also temporarily heal with PPI treatment, so biopsies must also be sampled from any visible scar tissue (Podolsky *et al*, 1988). *H. pylori* eradication may not completely eliminate the risk of gastric cancer. Expert consensus is to offer endoscopic and histological surveillance in patients at risk as defined by the extent and severity of mucosal atrophy on endoscopy. (Sugano *et al*, 2015) If eradication fails, second line therapy should be tried (**Fig.2.5**). If there is no response on second line therapy, or if symptoms persist despite successful eradication, refer the patient to a specialist. Culture from a biopsy of the gastric mucosa can determine potential antibiotic resistance.



Bismuth is a salt with bactericidal effect.
Bismuth quadruple is the triple therapy regiments with bismuth added in
Triple therapy: proton pump inhibitor plus two different antibiotics.
Quadruple therapy: proton pump inhibitor plus three different antibiotics
Sequential therapy: e.g. first 7 days proton pump inhibitor plus amoxicillin, next 7 days proton pump inhibitor plus clarithromycin plus metronidazole
Concomitant therapy: e.g. simultaneously proton pump plus amoxicillin, clarithromycin and metronidazole for 10 days

Fig. 2.5. Regimens for eradicating *H. pylori*

Chapter Three

Methodology

3. Methodology:

This chapter included the study area and design, study population, sample size determination, study variables (scope), data collection tools, data analysis and presentation, ethical consideration sand limitations**.**

3.1. Study design:

A comparative cross sectional study

3.2. Study area and duration:

This study was conducted in two universities, (University of 21 September and Emirates International University) during the period from April to May 2023 in Sana'a city.

3.3. Study population and sample

This study Was conducted among Medicine and Business Administration students in various disciplines

3.4. Sample size:

The sample size to be needed in this study was 565 subjects.

3.5. Case definition:

Case definitions are show in (appendix 1).

3.6. Consent form:

Consent form was ensured before collection data and drawing sample (Appendix 2).

3.7. Data collection:

The relative information was collected using a predesigned questionnaire (appendix 3)

3.8. Research tool:

The research tool that is used in this research is the research questionnaire.

A questionnaire is developed in order to collect the required data from Yemeni collages students at universities. The questionnaire contains (Name, Age, Sex, University, Specialty, Educational level, Marital state, Monthly income, Living, Have you been diagnosed for PUD, How have you been diagnosed, Do you suffer of any of the following "Change of appetite , Change of weight, Abdominal bloating, Abdominal pain, Early satiety, heartburn, blood on stool or change of its color, Nausea or Vomiting, No one of those symptoms", when does the symptoms started, Life style "smoking, stress, depression, sedatives, coffee or tea, chewing khat, energy drinks" , does the symptoms increases during exams? .

3.9. Data analysis:

The data were analyzed using a statistical package of social science program (SPSS, version 10). p of < 0.05 were considered statistically significant.

Chapter Four

Results

4. Results:

This comparative cross sectional study was carried out during a period of two months, starting in April and ending in May 2023. It included 565 students: (375) are from undergraduate students in Emirates International University and (189) from 21 September University in Sana'a city as shown in the figure (4.1).

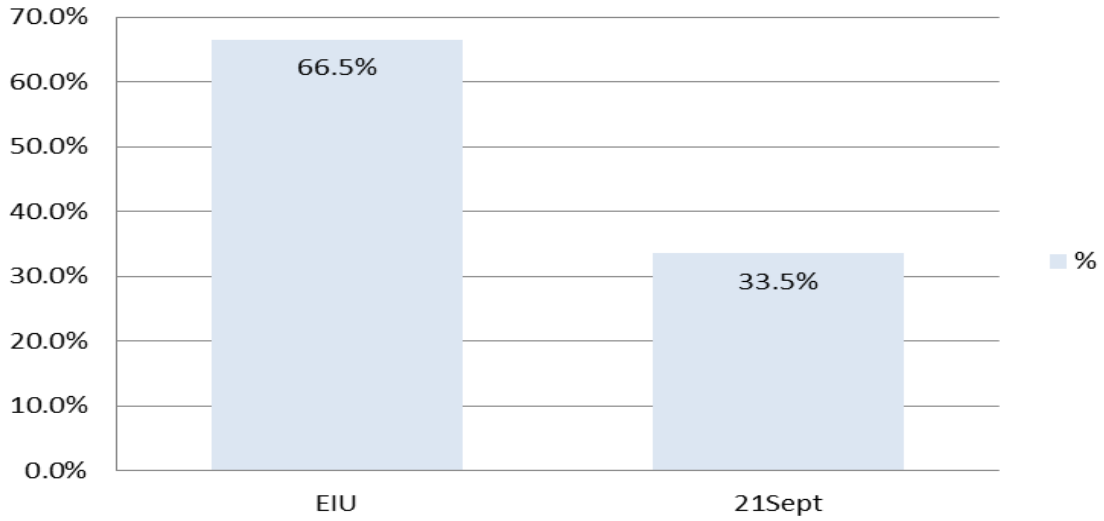


Figure (4.1) Distribution of student response for University

Their age ranged from less than 20 years to more than 30 years old. Out of them (345) 61.1% males and (220) 38.9% females (figure 4.2).

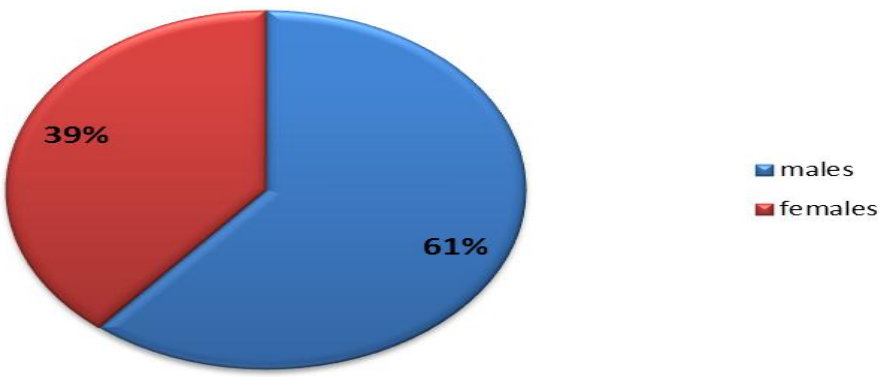


Figure (4.2) distribution of student according to Sex

The detailed results are presented in the following tables and figures:

Figure (4.3): The prevalence rate of reported PUD among the study group

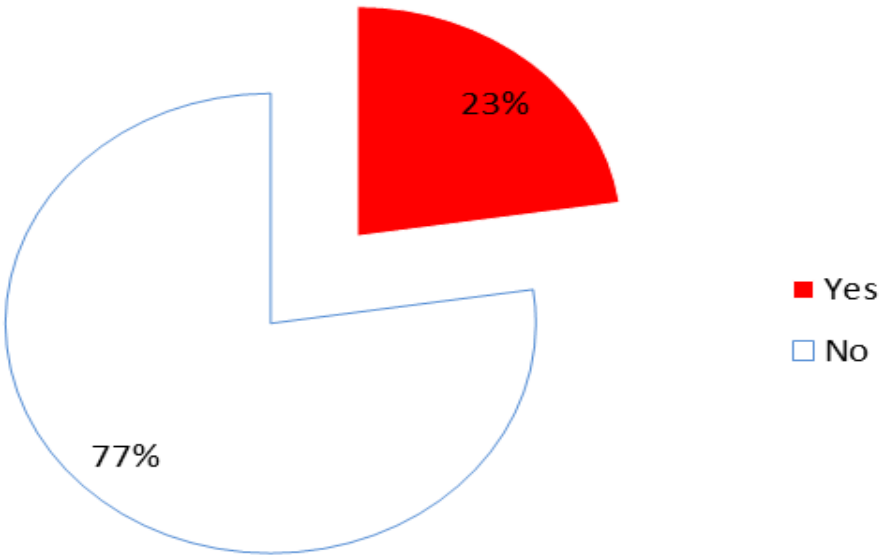


Figure (4.3) shows the prevalence rate of reported PUD among study group was (128/565) 22.7%.

Table (4.1): The prevalence rate of PUD according to age and gender

Property		**Diagnosed with PUD** (n=128)		**Total** (n=565)		***P***
		No.	**%**	**No.**	**%**	
Age groups (Years)	< 20	2	20.9	110	19.5	0.362
	20 — 25	88	22.3	394	69.7	
	25 — 30	17	29.8	57	10.1	
	> 30	0	0.0	4	0.7	
Gender	Male	71	20.6	345	61.1	0.085
	Female	57	25.9	220	38.9	
PUD: Peptic ulcer disease, (n): Number, p: probability. p < 0.05 (significant).						

Table (4.1) summarizes the distribution of PUD according to age and gender among the study group. The highest prevalence of PUD was 29.8% occurring among the age group 25-30 year and the lowest prevalence was 20.9% in the age group < 20 year. The prevalence rate of PUD between male and female students was (20.6 % and 25.9% respectively). However, the prevalence of PUD was not statistically significant according to the gender and age groups.

Figure (4.4) Proportion of students with symptoms suggestive of PUD.

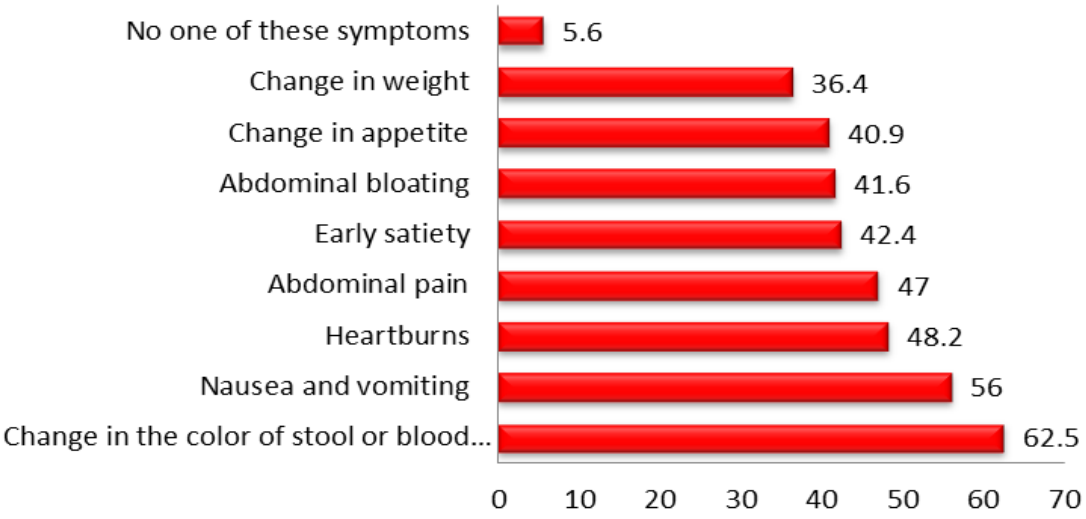


Figure (4.4) shows the proportion of students with symptoms suggestive of PUD. The proportions of clinical manifestations that suggestive PUD among the students suffering of change in the color of stool or blood on it 62.5%, nausea or vomiting 56%, heartburn 48.2%, abdominal pain 47%, early satiety 42.4%, abdominal bloating 41.6%, change of appetite 40.5%, and change of weight 36.4%.

Table (4.2): Timing of the beginning of symptoms with PUD.

Timing of beginning of symptoms	Diagnosed with PUD (n=128)		*P*
	No.	%	
Before university (n= 127)	39	30.7	0.000*
During university (n= 219)	77	35.2	
No symptoms appeared (n=219)	12	5.5	

Table (4.2) shows timing of the beginning of symptoms with the percentages of during university (35.2%), before university (30.7%), and finally no symptoms appeared was 5.5%. These results were statistically significant with values of, p <0.005.

Table (4.3): The prevalence rates of PUD according to sector of university

University	**Diagnosed with PUD** (n=128)		**Total** (n=565)		***P***
	No.	%	No.	%	
Privet	75	20.0	376	66.5	0.021*
Government	53	28.0	189	33.5	

Table (4.3) shows the prevalence rates of PUD according to sector of university. The prevalence rates of students PUD in Government University was (28%) that is higher than in students of Privet University (20%). This shows a significance among the rates of students with PUD who study in government university compering with those who study in privet university (*P*=0.021).

Table (4.4): The prevalence rates of PUD according to different college specialties

Specialty	Diagnosed with PUD (n=128)		Total (n=565)		*P*
	No.	%	No.	%	
Medicine	114	30.3	376	66.5	0.000*
Business administration	14	7.4	189	33.5	

Table (4.4) shows the prevalence rates of PUD according to different college specialties. The prevalence rate of PUD in medicine students was (30.3%) that is higher than in Business administration (7.4%). This results show a significant difference according to different college specialties ($P< 0.05$).

Table (4.5): The distribution of PUD in the different study levels

Grade of study	Diagnosed with PUD (n=128)		Total (n=565)		*P*
	No.	%	No.	%	
Frist level	18	14.8	122	21.6	0.000*
Second level	10	11.8	85	15.0	
Third level	23	25.6	90	15.9	
Fourth level	19	17.1	111	19.6	
Fifth level	58	36.9	157	27.8	

Table (4.5) shows the distribution of PUD between the students in different study levels. The rates of PUD in fifth level was 36.9%, in third level (25.6%), fourth level (17.1%), first level (14.8%), and finally in second level was (11.8%). This shows that fifth year students are the most affected with PUD. There is a statically significant confirmed this results by the $P<0.05$.

Table (4.6): The socioeconomic characters of students and PUD

Property		Diagnosed with PUD (n=128)		*P*
		No.	%	
Marital status	Married (n= 84)	17	20.2	0.338
	Single (n=481)	111	23.1	
Monthly income	Less than 100 thousand (n=373)	97	26.0	0.004*
	Between 100-200 thousand (n=128)	24	18.8	
	More than 200 thousand (n=64)	7	10.9	
Housing/living	With family (n=442)	95	21.5	0.347
	With colleagues (n=77)	22	28.6	
	Own house (n=46)	11	23.9	

***PUD**: Peptic ulcer disease, **p** < 0.05 (significant).*

Table (4.6) shows the socioeconomic characters of students with PUD**.** The main socioeconomic characters associated with PUD was monthly income in which the rates of PUD in less than 100 thousand (26.0%), between 100-200 thousand (18.8 %,) and more than 200 thousand (10.9%). These results were statistically significant with values of, *p* <0.005. The rate of PUD in single students was higher than married (23.1, 20.2% respectively). The rates of PUD in living with colleagues, own house, and with family were (28.6%, 23.9%, 21.5 respectively). These results showed no statistically significant for marital status, Housing/living with PUD.

Table (4.7) A: The modifiable risk factors associated with PUD in study groups.

Risk factors		**Diagnosed with PUD** (n=128)		***P***
		No.	%	
Smoking	NO (n=311)	41	13.2	0.000*
	Rarely (n=79)	18	22.8	
	Sometimes(n=64)	27	42.2	
	Often(n=64)	34	53.1	
	Mostly (n=47)	8	17.0	
Stressed	NO (n=82)	7	8.6	0.000*
	Rarely(n=92)	11	12.0	
	Sometimes(n=201)	47	23.4	
	Often(n=121)	44	36.4	
	Mostly(n=69)	19	27.5	
Having depression	NO(n=228)	33	15.9	0.009*
	Rarely(n=33)	30	21.3	
	Sometimes(n=106)	49	34.8	
	Often(n=140)	11	20.0	
	Mostly(n=58)	5	25.0	

Table (4.7) A: shows the modifiable risk factors associated with PUD in study groups. The main risk factors associated with PUD in study groups were smoking, stressed and having depression. The rates of PUD in often smoking students, sometimes, rarely, and mostly were (53.1%, 42.2%, 22.8%, 17.0% respectively) while the rates of PUD in no smoking students was (13.2%). These results were in a statistically significant difference with values of, p <0.005. The rates of often stressed students, mostly, sometimes, and rarely were (36.4%, 27.5%, 23.4%, 12.0% respectively) while the rates of no stressed students was (8.6%). These results were in a statistically significant difference with values of, p <0.005. The rates of PUD in sometimes having depression (34.8%), mostly (25.0%), rarely (21.3%), often (20.0%), while the rate of PUD in no having depression (15.9%). These results were in a statistically significant difference with values of, p=0.009.

Table (4.7) B: The modifiable risk factors related with PUD in study groups.

Risk factors		**Diagnosed with PUD** (n=128)		***P***
		No.	%	
Drinking soft drinks	NO (n=443)	107	24.2	0.185
	Rarely(n=64)	10	15.6	
	Sometimes(n=29)	7	24.1	
	Often(n=20)	2	10.0	
	Mostly(n=9)	2	22.2	
Drink coffee & tea	NO (n=59)	11	18.6	0.800
	Rarely(n=72)	15	20.8	
	Sometimes(n=153)	42	27.5	
	Often(n=155)	37	23.9	
	Mostly(n=126)	23	18.3	
Taking sedatives	NO(n=306)	57	18.6	0.133
	Rarely(n=126)	37	29.4	
	Sometimes(n=86)	22	25.6	
	Often(n=31)	10	32.3	
	Mostly(n=16)	2	12.5	
Chewing khat	NO(n=226)	57	25.2	0.202
	Rarely(n=63)	16	25.4	
	Sometimes(n=76)	12	15.8	
	Often(n=102)	25	24.5	
	Mostly(n=98)	18	18.4	
Increase symptoms during exams	NO(n=228)	50	21.9	0.693
	Rarely(n=33)	3	9.1	
	Sometimes(n=106)	31	29.2	
	Often(n=140)	32	22.9	
	Mostly(n=58)	12	20.7	

Table (4.7) B: shows the modifiable risk factors associated with PUD in study groups. The rates of other risk factors includes drinking soft drinks, drink coffee & tea, taking sedatives, chewing khat and increase symptoms during exams were not associated with PUD and showed no statistically significant values of, $p > 0.005$.

Chapter Five

Discussion

5. Discussion:

This chapter discusses in detail the major findings and the implications of them. The results put in the context of the previous and recent research in form of comparing our finding with other researchers finding (where applicable) and comparing our findings based on the background variables.

This study was one of the first researches conducted in Yemen that aimed to determine the prevalence of PUD among undergraduate students of Yemeni universities, risk factors for the affected group of students, the more affected college level of students, differentiate between the prevalence of PUD in medical and non-medical students and whom university sector students are the more affected.

In the present study, out of the 565 subjects, the prevalence of PUD was detected in 22.7% of the students as. This result was nearly similar to other one study in neighboring country in Saudi Arabia (21.9%) (Afaf *et al*, 2017). On the other hand, this result was highly compared with three studies performed in Nigeria (5.70%) (Onoh *et al*, 2017), (6.25%) (Zibima *et al*, 2020), and finally (7.9%) (Anaemene and Ochogu, 2022). This height couldn't also be linked to the global reduction in PUD prevalence. These disagreeable findings could be explained by the result of arise of risk factors in the lifestyle of Yemeni student.

In the current study, among (565) students, a high number of them were diagnosed after the beginning of university by (35.2%) as shown in table (3.2). This finding was consistent to other studies in Nigeria (37.5%) (Ogunmodede *et al*, 2016*)* and there are finding supported by that of Bayana *et al*, (2021) which reported that 41.3% of the students of Jimma University in Ethiopia had symptoms suggestive of PUD which developed most after enrolling at the university. This compatibility results may be due to University education is usually viewed as stressful.

In the current study, showed a statistical significance (p =0.021) with high rate of PUD in government university students (28%) compering with privet university students (20%) as shown in table (3.3). To the best of our knowledge, we have not found studies similar to this one.

In our study, found that medical specialty is higher affected of PUD (30.3%) comparing to (7.4%) in business administration as shown in table (3.4). The results showed a statistical significant difference between medical and non-medical specialty with (P <0.0∘). This difference may be due to the full time medicine students are commonly scheduled for academic activities from 8am-5pm and have to attend to assignments and personal study during the night.

Regarding the grader, the results showed that there is a significant difference between the different levels of college in getting affected with PUD in which the study showed that fifth level are the ones at highest danger to get PUD (36.9%) shown in table (3.5), While the other levels are at lower risk of getting affected with PUD. This result is not in agreement with other studies that were conducted in South Nigeria (Zibima *et al, 2020)* who showed that PUD was most prevalent among first-year students (30.4%).

A significant relationship has been found between PUD and the student's monthly income table (3.6). This confirmed by (Stanghellini, 1999) who stated that there was an association between the prevalence of peptic ulcer and monthly income and variables of income in general population of seven international sites.

In this study found that from cases with PU there was a significant relation between peptic ulcer and smoking status (p<0.05). Result of this study was in agreement with a previous study done in china (Li *et al*, 2010), Denmark (Rosenstock *et al*, 2003), Iran (Koroush & Hamed, 2016), USA (Kurata & Nogawa, 1997), Amassoma Bayelsa State, Nigeria (Zibima *et al*, 2020). On other hand, this result was not corresponding to the report

(Assefa *et al*, 2022) in Ethiopia who reported no significant between smoking and PUD. This difference in findings could be explained by the different in geographical areas.

The finding of table (3.7) A revealed that there is a very dangerous link between PUD and intensity of stress which was statistically significant, (P<0.005). Result of this study was corroborated with other studies reported in Saudi Arabia (Afaf *et al*, 2017), Nigeria (Zibima *et al*, 2020), Korea (Yim *et al, 2021).* There are, however, contrasting reports on the relationship between stress and PUD. For example, while Song, Jung and Jung in Korea found no difference in stress level between peptic ulcer patients and controls using the stress severity scale (Song *et al*, 2013). These different results could be by different of stress intensity between studies.

Here the result showed that there is a seriousness between PUD and students who suffer of depression with statistically significant and P=0.009, shown in table (3.7)A. Result of this study was in agreement with a previous study conducted in South Nigeria, (Fang *et al, 2019*; Zibima *et al*, 2020) And other two longitudinal follow-up studies in a Korean. (Kim *et al*, 2020).

In our study, the symptoms that suggestive PUD among the students were suffering of change in the color of stool or blood on it (62.5%), nausea or vomiting (56%), heartburn (48.2%), abdominal bloating (41.6%), and change of appetite (40.5%). These results are coordinated in study of Anaemene and Ochogu (2022) in Nigeria that state the students complained of symptoms suggestive of PUD such as heart burn (46.4%), change of appetite (43.9%), nausea or vomiting (33.8%), and bloody stool (15.8%).

Chapter Six

Conclusions & Recommendations

6. Conclusions and Recommendations

5.1 Conclusions

From the results of the present study, the following can be concluded:

- The present study has proved that the prevalence Of PUD among under graduate students is high with a (22.7%) of the study sample.
- Medical students are more affected with PUD than non-medical students.
- Governmental sector students are more affected with PUD than privet sector students.
- The following factors (age, gender, marital status, housing, drinking soft drinks, coffee and tea, taking sedative, and kat chewing) has no role in the affection with PUD.
- The following factors (Smoking, stress, depresion) has a role in the affection with PUD.

5.2 Recommendations

The following can be recommended from this study:

- Increase awareness and education about PUD among university students in Yemen through health campaigns and educational programs.
- Encourage regular check-ups and early diagnosis of PUD among university students.
- Conduct further research to determine the risk factors associated with PUD among university students in Yemen.
- Conduct further researches about the rule of *H. pylori*, Genetics and Depression on causing PUD
- Collaborate with healthcare providers and universities to develop a comprehensive approach to managing PUD among university students in Yemen.
- Encourage healthy lifestyle habits such as regular exercise, healthy eating, and stress management to prevent the development of PUD.
- Provide counseling services for students diagnosed with PUD to help them manage their symptoms and improve their quality of life.
- Develop guidelines for the management of PUD in university settings, including recommendations for screening, diagnosis, treatment, and follow-up care.
- Finally, it is recommended that policymakers prioritize the prevention and management of PUD among university students in Yemen by allocating resources towards research, education, and healthcare services.

References

- Afaf SB, Nagah M, Reem F, Najah SF, Sara E, Arwa N, *et al*. Profile of peptic ulcer disease and its risk factors in Arar, Northern Saudi Arabia. Electronic Physician, 2017; 9(11): 5740-5745.

- Ahlawat R, Hoilat GJ, Ross AB. Esophagogastroduodenoscopy. [Updated 2022 Aug 14]. In: StatPearls [Internet]. Treasure Island (FL): StatPearls Publishing; 2023 Jan.

- Åhsberg K, Höglund P, Kim W-H, Staël von Holstein C. Impact of aspirin, NSAIDs, warfarin, corticosteroids and SSRIs on the site and outcome of non-variceal upper and lower gastrointestinal bleeding. Scand J Gastroenterol, 2010; 45: 1404-1415.

- Akhtar, F., Shelton, P., & Dinh, A. Peptic ulcer disease. In F. Domino, R. Baldor, J. Golding, & M. Stephens (Eds.), The 5-minute clinical consult, 27th, Philadelphia, PA : Wolters Kluwer, Philadelphia, PA : Wolters Kluwer. 2019: 748– 749.

- Allen JI, Katzka D, Robert M, Leontiadis GI. American Gastroenterological Association Institute technical review on the role of upper gastrointestinal biopsy to evaluate dyspepsia in the adult patient in the absence of visible mucosal lesions. Gastroenterology, 2015; 149(4): 1088–1118.

- Alves-Santos AM, Sugizaki CS, Lima GC, Naves MM. Prebiotic effect of dietary polyphenols: A systematic review. Journal of Functional Foods, 2020 Nov 1; 74: 104169.

- Amieva MR, El-Omar EM. Host-bacterial interactions in H. pylori infection. Gastroenterology, 2008; 134(1): 30 6–23.

- Anaemene D & Ochogu E. Prevalence, symptoms and lifestyle aspect of peptic ulcer disease among students of a Nigerian University. African Journal of Biomedical Research, 2022; 25(2):129–133.

- Anbalakan K, Chua D, Pandya GJ, Shelat VG. Five year experience in management of perforated peptic ulcer and validation of common mortality risk prediction models - are existing models sufficient?. Int J Surg, 2015; 14: 38-44.

- Andersen IB, Jørgensen T, Bonnevie O, Grønbæk, M & Sørensen TI. Smoking and alcohol intake as risk factors for bleeding and perforated peptic ulcers: a population-based cohort study. Epidemiology, 2000; 11: 434–439.

- Arroja B, Cremers I, Ramos R, Cardoso C, Rego AC, Caldeira A, Eliseu L, Silva JD, Gloria L, Rosa I, Perdosa J. Acute lower gastrointestinal bleeding management in Portugal: a multicentric prospective 1-year survey. Eur J Gastroenterol Hepatol, 2011; 23: 317-322.

- ASGE Standards of Practice Committee; Banerjee S, Cash BD, Dominitz JA, Baron TH, Anderson MA, Ben-Menachem T, Fisher L, Fukami N, Harrison ME, Ikenberry SO, Khan K, Krinsky ML, Maple J, Fanelli RD, Strohmeyer L. The role of endoscopy in the management of patients with peptic ulcer disease. Gastrointest Endosc. 2010 Apr; 71(4):663-8.

- Assefa B, Tadesse A, Abay Z, Abebe A, Tesfaye T, Tadesse M, and Molla A. Peptic ulcer disease among dyspeptic patients at endoscopy unit, University of Gondar hospital, Northwest Ethiopia. BMC Gastroenterology, 2022; 22:164

- Barat I, Andreasen F, Damsgaard EMS. The consumption of drugs by 75yearold individuals living in their own homes. Eur J Clin Pharmacol, 2000; 56: 501-509.

- Bardhan KD, Williamson M, Royston C, Lyon C. Admission rates for peptic ulcer in the Trent region, UK, 1972-2000; Changing pattern, changing disease? Dig Liv Dis, 2004; 36: 577-588.

- Bas G, Eryilmaz R, Okan I, Sahin M. Risk factors of morbidity and mortality in patients with perforated peptic ulcer. Acta Chir Belg, 2008; 108: 424-427.

- Bashinskaya B, Nahed BV, Redjal N, Kahle KT, Walcott BP. Trends in Peptic Ulcer Disease and the Identification of Helicobacter Pylori as a Causative Organism: Population-based Estimates from the US Nationwide Inpatient Sample. Journal of Global Infectious Diseases, 2011: 3(4), 366–370.

- Bashinskaya B, Nahed BV, Redjal N, Kahle KT, Walcott BP. Trends in Peptic Ulcer Disease and the Identification of Helicobacter Pylori as a Causative Organism: Population-based Estimates from the US Nationwide Inpatient Sample. Journal of Global Infectious Diseases, 2011: 3(4): 366–370.

- Bateson MC. Cigarette smoking and Helicobacter pylori infection. Postgrad Med J, 1993; 69: 41-4.

- Battaglia G et al. Recurrence of duodenal ulcer under continuous antisecretory treatment: an approach to the detection of predictive markers. Am. J. Gastroenterol, 1984; 79: 831–834.

- Bayana E, Olani A, Biratu Y, Negash A, Gelan M, disease: a cross-sectional study of symptoms and risk factors among students at Jimma University, Ethiopia. Gastrointestinal Nursing, 2021; 19 (2): 36–40.

- Bayyurt N, Abasiyanik MF, Sander E & Salih BA. Canonical correlation analysis of factors involved in the occurrence of peptic ulcers. Dig. Dis. Sci, 2007; 52: 140–146.

- Bellack NR, Koehoorn MW, MacNab YC, et al. A conceptual model of water's role as a reservoir in Helicobacter pylori transmission: a review of the evidence. Epidemiol Infect, 2006; 134(3): 439–49.

- Bernersen B, Johnsen R & Straume B. Non-ulcer dyspepsia and peptic ulcer: the distribution in a population and their relation to risk factors. Gut, 1990; 38: 822–825.

- Bernersen B, Johnsen R, Straume B, Burhol PG, Jenssen TG, Stakkevold PA. Towards a true prevalence of pepticulcer: the Sørreisa gastrointestinal disorder study. Gut. 1990 Sep; 31(9):989-92.

- Boey J, Choi SK, Poon A, Alagaratnam TT. Risk stratification in perforated duodenal ulcers. A prospective validation of predictive factors. Ann Surg, 1987; 205: 22-26.

- Brown LM. Helicobacter pylori: epidemiology and routes of transmission. Epide-miol Rev, 2000; 22(2): 283–97.

- Buck DL, Møller MH. Influence of body mass index on mortality after surgery for perforated peptic ulcer. Br J Surg, 2014; 101: 993-999.

- Center for Disease Control and Prevention (CDC); Helicobacter pylori and peptic ulcer disease, USA, Atlanta, Georgia, 2001: 1-3.

- Chalya PL, Mabula JB, Koy M, et al. Clinical profile and outcome of surgical treatment of perforated peptic ulcers in Northwestern Tanzania: A tertiary hospital experience. World J Emerg Surg, 2011; 6: 31.

- Chen MY, He CY, Meng X, Yuan Y. Association of Helicobacter pylori babA2 with peptic ulcer disease and gastric cancer. World J Gastroenterol, 2013; 19: 4242-51.

- Chey WD, Leontiadis GI, Howden CW, Moss SF. ACG Clinical Guideline: Treatment of Helicobacter pylori Infection. Am J Gastroenterol, 2017; 112: 212-239.

- Chey WD, Wong BC, Practice Parameters Committee of the American College of G. American College of Gastroenterology guideline on the management of Helicobacter pylori infection. Am J Gastroenterol, 2007; 102: 1808-1825.

- Chung KT, Shelat VG. Perforated peptic ulcer an update. World J Gastrointest Surg, 2017; 9: 1-12.

- Colin-Jones DG. The role and limitations of H2-receptor antagonists in the treatment of gastro-oesophageal reflux disease. Aliment Pharmacol Ther, 1995; 9(l 1): 9-14.

- Cullen DJ, Hawkey GM, Greenwood DC, Humphreys H, Shepherd V, Logan RF, Hawkey CJ. Peptic ulcer bleeding in the elderly: relative roles of Helicobacter pylori and non-steroidal anti-inflammatory drugs. Gut, 1997; 41(4): 459-62.

- Datta D, Roychoidhury S. To be or not to be: the host genetic factor and beyond Helicobacter pylori mediated gastro-duodenal diseases. World J Gastroenterol, 2015; 21: 2883-95.

- De Schryver AA, Van Hooste WL, Van Winckel MA, et al. H. pylori infection: a global occupational risk for healthcare workers? Int J Occup Environ Health, 2004; 10(4): 428–32.

- Debruyne LK, Pinna K and Whitney E. Nutrition and Diet Therapy. 9th, US, Boston, Cengage Learning, 2016: 480.

- Del Valle J. Peptic ulcer disease and related disorders. In: Kasper DL, Fauci AS, Hauser SL, Longo DL, Jameson JL, Loscalzo J, eds. Harrison's Principles of Internal Medicine (19th ed.) New York, NY: McGraw Hill Education, 2015: 1911−32.

- Drenowatz C, Eisenmann JC, Pfeiffer KA, et al. Influence of socioeconomic status on habitual physical activity and sedentary behavior in 8- to 11-year old children. BMC Public Health. 2010; 10: 214.

- Eniojukan JF, Okonkwo OC and Adje UD. Risk Factors, management and other correlates of peptic ulcer disease in a university community in south-south Nigeria. UK Journal of Pharmaceutical and Biosciences Journal, 2017; 5(6): 07–15.

- Etukudo OM, Ikpeme EE, Ekanem EE. Seroepidemiology of Helicobacter pylori infection among children seen in a tertiary hospital in Uyo, Southern Nigeria. Pan Afr Med J, 2012: 12.

- Etukudo OM, Ikpeme EE, Ekanem EE. Seroepidemiology of Helicobacter pylori infection among children seen in a tertiary hospital in Uyo, Southern Nigeria. Pan Afr Med J, 2012; 12:39.

- Everhart JE, Byrd-Holt D & Sonnenberg. A. Incidence and risk factors for self reported peptic ulcer disease in the United States. Am. J. Epidemiol, 1998; 147: 529–536.

- Fang B, Yang S, Lui H, Zhang Y, Xu R, Chen G. Association between depression and subsequent peptic ulcer occurrence among older people living alone: A prospective study investigating the role of change in social engagement. Journal of Psychosomatic Research, 2019; 122: 94-103.

- Fashner J & Gitu A. Diagnosis and treatment of peptic ulcer disease and H. pylori infection. American Family Physician , 2015; 91(4): 236–242.

- Feldman M, Friedman LS, Brandt LJ. Sleisenger and Fordtran's gastrointestinal and liver disease: pathophysiology/diagnosis/management. 10th ed. Philadelphia, PA: Saunders/Elsevier, 2016; 2: 2369− 2389.

- Furuta T, El-Omar EM, Xiao F, Shirai N, Takashima M, Sugimura H. Interleukin 1Î² polymorphisms increase risk of hypochlorhydria and atrophic gastritis and reduce risk of duodenal ulcer recurrence in Japan. Gastroenterology, 2002; 123: 92-105.

- Gerard M. and Lawrence W. Current Surgical diagnosis and treatment, 12th, New York. Raw-Hill Companies, 2006: 513-515.

- Gisbert JP, Calvet X. Review article: Helicobacter pylori-negative duodenal ulcer disease. Aliment Pharmacol Ther 2009; 30: 791-815.

- Gralnek IM, Dumonceau JM, Kuipers EJ, et al. Diagnosis and management of nonvariceal upper gastrointestinal hemorrhage: European Society of Gastrointestinal Endoscopy (ESGE) Guideline. Endoscopy, 2015; 47: 1-46.

- Guerra-Valle M, Orellana-Palma P, Petzold G. Plant-Based Polyphenols: Anti-Helicobacter pylori Effect and Improvement of Gut Microbiota. Antioxidants, 2022 Jan; 11(1): 109.

- Habeeb A, Tiwari SK, Bardia A, Khan S, Vishwakarma SK, Habeeb S, et al. Peptic Ulcer Disease: Descriptive Epidemiology, Risk Factors, Management and Prevention. Accessed on November 9, 2020 from www.smgebooks.com.

- Hearnshaw SA, Logan RF, Lowe D, Travis SP, Murphy MF, Palmer KR. Acute upper gastrointestinal bleeding in the UK: patient characteristics, diagnoses and outcomes in the 2007 UK audit. Gut, 2011; 60: 1327-35.

- Heo J, Jung MK. Safety and efficacy of a partially covered self-expandable metal stent in benign pyloric obstruction. World J Gastroenterol, 2014; 20: 16721-5.

- Hermansson M, Staël von Holstein C, Zilling T. Surgical approach and prognostic factors after peptic ulcer perforation. Eur J Surg, 1999; 165: 566-572.

- Hreinsson JP, Guðmundsson S, Kalaitzakis E, Björnsson ES. Lower gastrointestinal bleeding: incidence, etiology, and outcomes in a population-based setting. Eur J Gastroenterol Hepatol, 2013; 25: 37-43.

- Huang JQ, Sridhar S, Hunt RH. Role of Helicobacter pylori infection and non-steroidal anti-inflammatory drugs in peptic-ulcer disease: a meta-analysis. Lancet, 2002 Jan 05; 359(9300):14-22.

- Hung LC, Ching JY, Sung JJ, et al. Long-term outcome of Helicobacter pylori-negative idiopathic bleeding ulcers: a prospective cohort study. Gastroenterology, 2005; 128: 1845-50.

- Hussen BM, Qader SS, Ahmed HF and Ahmed SH. The prevalence of Helicobacter pylori among university students in Iraq. Indian Journal of Science and Technology, 2013; 6(8): 5019–5023.

- Jemikajah DJ, Okogun GR. Health point prevalence of Helicobacter pylori in central hospital, Warri, Nigeria. Afr J Cell Pathol, 2014; 3: 57-60.

- Jemikajah DJ, Okogun GR. Health point prevalence of Helicobacter pylori in central hospital, Warri, Nigeria. Afr J Cell Pathol, 2014; 3: 57-60.

- Johnsen R, Forde OH, Straume B & Burhol PG. Aetiology of peptic ulcer: a prospective population study in Norway. J. Epidemiol. Commun. Health, 1994; 48: 156–160.

- Kainat S, Bahisht R, Sana N, et al. Determinants of peptic ulcer disease: A systematic review. EAS Journal of Nutrition and Food Sciences, 2020; 2(5): 257–264.

- Kang JY, Elders A, Majeed A, Maxwell JD, Bardhans KD. Recent trends in hospital admissions and mortality rates for peptic ulcer in Scotland 1982-2002. Aliment Pharmacol Ther, 2006; 24: 65-79.

- Kanno T, Iijima K, Koike T, Abe Y, Shimada N, Hoshi T, et al. Accommodation in a refugee shelter as a risk factor for peptic ulcer bleeding after the Great East Japan Earthquake: a case-control study of 329 patients. J Gastroenterol, 2015; 50: 31-40.

- Karami L, Majd A, Mehrabian S, Nabiuni M, Irian S, Salehi M. Antimutagenic effects of ethanol and aqueous extracts of Avicenniamarina leaves on Salmonella typhimurium TA100 bacterium. Arak Med Univ J, 2013; 15: 68-76.

- Kato I, Nomura AM, Stemmermann GN & Chyou PH. A prospective study of gastric and duodenal ulcer and its relation to smoking, alcohol, and diet. Am. J. Epidemiol. 1992; 135: 521–530.

- Kazemi, S, Tavakkoli H, Habizadeh MR and Emami MH. Diagnostic values of Helicobacter pylori diagnostic tests: Stool antigen test, urea breath test, rapid urease test,

serology and histology. Journal of Research in Medical Sciences, 2011; 16(9): 1097–1104.

- Kim SY, Min C, Oh DJ, Choi HG. Reciprocal association between depression and peptic ulcers: Two longitudinal follow-up studies using a national sample cohort. Scientific Reports, 2020; 10: 1749.

- Kiss S, Zsikla V, Frank A, Willi N, Cathomas G. Helicobacter-negative gastritis: polymerase chain reaction for Â HelicobacterÂ DNA is a valuable tool to elucidate the diagnosis. Aliment Pharmacol Ther 2016; 43: 924-32.

- Kivi M, Tindberg Y. Helicobacter pylori occurrence and transmission: a family affair? Scand J Infect Dis, 2006; 38(6/7): 407–17.

- Koolhaas JM, et al. Stress revisited: a critical evaluation of the stress concept. Neurosci. Biobehav. Rev, 2011; 35: 1291–1301.

- Koroush S, Hamed T. Systematic review and meta-analysis methods prevalence of peptic ulcer in Iran. Govaresh, 2016; 20(4): 258.

- Kurata JH, Nogawa AN. Meta-analysis of risk factors for peptic ulcer. Nonsteroidal antiinflammatory drugs, Helicobacter pylori, and smoking. J Clin Gastroenterol, 1997; 24: 2–17.

- Lanas Á, Carrera-Lasfuentes P, Arguedas Y, et al. Risk of upper and lower gastrointestinal bleeding in patients taking nonsteroidal anti-inflammatory drugs, antiplatelet agents, or anticoagulants. Clin Gastroenterol Hepatol, 2015 May; 13(5):906-12.

- Lanas A, Chan FKL. Peptic ulcer disease. Lancet, 2017; 390(10094): 613-624.

- Lanas A, GarcÃ-a-GonzÃ¡lez MA, Santolaria S, et al. TNF and LTA gene polymorphisms reveal different risk in gastric and duodenal ulcer patients. Genes Immun, 2001; 2: 415-21.

- Lanas A, García-Rodríguez LA, Polo-Tomás M, et al. The changing face of hospitalization due to gastrointestinal bleeding and perforation. Aliment Pharmacol Ther, 2011; 33: 585−91.

- Lanas AI, Remacha B, Esteva F, Sainz R. Risk factors associated with refractory peptic ulcers. Gastroenterology, 1995; 109: 1124-1133.

- Langman MJS. Peptic ulcer epidemiology. Clin Surg Int, 1983; 7: 1-13.

- Lanza FL, Chan FK, Quigley EMPractice Parameters Committee of the American College of Gastroenterology. Guidelines for prevention of NSAID-related ulcer complications. AmJ Gastroenterol, 2009; 104: 728-38.

- Lau JY, Sung J, Hill C, Henderson C, Howden CW, Metz DC. Systematic review of the epidemiology of complicated peptic ulcer disease: incidence, recurrence, risk factors and mortality. Digestion, 2011; 84: 102-13.

- Levenstein S & Kaplan GA. Socioeconomic status and ulcer. A prospective study of contributory risk factors. J. Clin. Gastroen- terol, 1998; 26: 14–17.

- Levenstein S. et al. Patterns of biologic and psychologic risk factors in duodenal ulcer patients. J. Clin. Gastroenterol, 1995; 21: 110–117

- Levenstein S. Peptic ulcer at the end of the 20th century: biological and psychological risk factors. Can. J. Gastroenterol, 1999; 13: 753–759.

- Levenstein, S. Stress and peptic ulcer: life beyond helicobacter. BMJ, 1998: 316, 538.

- Li Z, ou D, Ma X et al. Epidemiology of peptic ulcer disease: endoscopic results of the systematic investigation of gastrointestinal disease in China. Am. J. Gastroenterol, 2010; 105: 2570–2577.

- Lim SL, Canavarro C, Zaw MH, et al. Irregular meal timing is associated with Helicobacter pylori infection and gastritis. ISRN Nutrition, 2013 Article ID 714970.

- Linz B, Balloux F, Moodley Y, et al. An African origin for the intimate association between humans and Helicobacter pylori. Nature, 2007; 445(7130): 915–8.

- Longstreth GF. Epidemiology and outcome of patients hospitalized with acute lower gastrointestinal hemorrhage: a population-based study. Am J Gastroenterolm, 1997; 92: 419-424.

- Lu CL, Chang SS, Wang SS, Chang FY, Lee SD. Silent peptic ulcer disease: frequency, factors leading to "silence," and implications regarding the pathogenesis of visceral symptoms Gastrointest Endosc, 2004;60(1): 34–38.

- Malfertheiner P, Chan FK, McColl KE. Peptic ulcer disease. Lancet, 2009; 374(9699): 1449–1461.

- Malfertheiner P, Megraud F, O'Morain CA, Gisbert JP, Kuipers EJ, et al. Management of Helicobacter pylori infection-the Maastricht V/ Florence Consensus Report. Gut, 2017; 66: 6-30.

- Malhotra SL. Epidemiological study of peptic ulcer in the South India. Gut, 1967; 8: 180-8.

- Malik TF, Gnanapandithan K, Singh K. Peptic Ulcer Disease. 2023 Feb 12. In: StatPearls [Internet]. Treasure Island (FL): StatPearls Publishing; 2023 Jan.

- Malmi Hanna. Peptic ulcer disease: incidence, associated morbidity and mortality. Helsinki, 2018: 90.

- Martin J Blaster and Douglas E. Helicobacter pylori genetic diversity and risk of human disease. The Journal of clinical investigation, 2001; (107): 767-773.

- McColl KE. H. pylori-negative nonsteroidal anti-inflammatory drug-negative ulcer. Gastroenterol Clin North AM, 2009; 38: 353-361.

- Merck Manuals 2006. "Peptic Ulcer". Home Health Handbook for Patients & Caregivers. (Archived from the original on 28 December 2011).

- Moayyedi PM, Lacy BE, Andrews CN, Enns RA, Howden CW & Vakil N. ACG and CAG clinical guideline: Management of dyspepsia. The American Journal of Gastroenterology, 2017; 112(7): 988–1013.

- Morra ME, Elshafay A, Kansakar AR, Mehyar GM, Dang NPH, et al. Definition of "persistent vomiting" in current medical literature: A systematic review. Medicine (Baltimore), 2017 Nov; 96(45): e8025.

- Mousavi F, Rahnema M, Heydarieh N, Tajabadi Ebrahimi M. The effect of Iranian native Lactobacillus pentosus on healing of gastric ulcers in male Wister rats. Arak Med Univ J, 2013; 16: 81-90.

- Nahon S, Hagège H, Latrive JP, Rosa I, Nalet B, Bour B, et al. Epidemiological and prognostic factors involved in upper gastrointestinal bleeding: results of a French prospective multicenter study. Endoscopy, 2012; 44: 998-1006.

- Nervi G, Liatopoulou S, Cavallaro LG, Gnocchi A, Dal-Bo N, et al. Does H. pylori infection eradication modify peptic ulcer prevalence? A 10 years endoscopical survey. World J Gastroenterol, 2006 Apr 21; 12(15): 2398-401.

- Ng FH, Wong SY, Lam KF, et al. Famotidine is inferior to pantoprazole in preventing recurrence of aspirin-related peptic ulcers or erosions. Gastroenterology, 2010; 138: 82-88.

- Ocasio Quinones GA, Woolf A. Duodenal Ulcer. [Updated 2022 May 7]. In: StatPearls [Internet]. Treasure Island (FL): StatPearls Publishing; 2023 Jan.

- Ogunmodede JA, Bojuwoye MO, Olokoba AB, Bolariwa OA and Kolo PM. Self-reported symptoms of univestigated dyspepsia among university staff in Ilorin, Nigeria. Res J of Health Sci, 2016; 4 (4): 296-303.

- Onoh CC, Aguocha JA, Osuagwu IK, Amadi CO, Akalonu GI and Nwabueze RN. Magnitude and pattern of disease presentation among students in Federal University of Technology Owerri Nigeria. IOSR Journal of Dental and Medical Sciences, 2017; 16 (3): 116-121.

- Overmier JB & Murison R. Restoring psychology's role in peptic ulcer. Appl. Psychol. Health Well Being, 2013; 5: 5–27.

- Papatheodoridis G, Sougioultzis S, Archimandritis A. Effects of Helicobacter Pylori and Nonsteroidal Anti Inflammatory Drugs on Peptic Ulcer Disease: A Systematic Review. Journal Clinical Gastroenterology and Hepatology, 2006; 4 (2):130-42.

- Paspatis GA, Matrella E, Kapsoritakis A, Leontithis C, Papanikolaou N, Chlouverakis GJ, Kouroumalis E. 2000. An epidemiological study of acute upper gastrointestinal bleeding in Crete, Greece. Eur J Gastoenterol Hepatol, 2000; 12: 1215-1220.

- Peery AF, Crockett SD, Barritt AS, Dellon ES, Eluri S, Gangarosa LM, Jensen ET, Lund JL, Pasricha S, Runge T, Schmidt M, Shaheen NJ, Sandler RS. 2015. Burden of

Gastrointestinal, Liver, and Pancreatic Diseases in the United Staes. Gastroenterology, 2015; 149: 1731-41.

- Pérez-Aisa MA, del Pino D, Siles M, Lanas A. 2005. Clinical trens in ulcer diagnosis in a population with high prevalence of Helicobacter pylori infection. Aliment Pharmacol Ther, 2005; 21: 65-72.

- Peura DA, Crowe SE. Helicobacter pylori. In: Feldman M, Friedman LS, Brandt LJ, editors. Sleisenger and Fordtran's gastrointestinal and liver disease. 9th. Philadelphia, Saunders; 2010: 833–45.

- Podolsky I, Storms PR, Richardson CT, Peterson WL, Fordtran JS. Gastric adenocarcinoma masquerading endoscopically as benign gastric ulcer. A five-year experience. Dig Dis Sci, 1988; 33: 1057-63.

- Prasad M & Friedman L. Peptic ulcer disease. In S. Srinivasan & L. Friedman (Eds.), Essentials of gastroenterology 2nd, Hoboken, NJ: Wiley, 2018: 35– 44.

- Rafi AHS. Peptic Ulcer Disease among the Patients with Abdominal Pain Attending the Department 0f Medicine in Dhaka Medical College Hospital, Bangladesh. IOSR Journal of Dental and Medical Sciences, 2014; 13 (1): 05-20.

- Rajesh V, Chandra SS, Smile SR. Risk factors predicting operative mortality in perforated peptic ulcer disease. Trop Gastroenterol, 2003; 24: 148-150.

- Ray-Offor E and Opusunju KA. Current status of peptic ulcer disease in Port Harcourt metropolis, Nigeria. African Health Science, 2020; 20(3): 1446–1451.

- Razvodovsky YE. Alcohol poisoning and duodenum ulcer mortality in Russia, 1965–2005. Adicciones, 2007; 19: 423–431.

- Robert A, Olafsson AS, Lancaster C, Zhang WR. Interleukin-1 is cytoprotective, antisecretory, stimulates PGE2 synthesis by the stomach, and retards gastric emptying. Life Sci, 1991; 48: 123-34.

- Rosenstock S, Jorgensen T, Bonnevie O & Andersen L. Risk factors for peptic ulcer disease: a population based prospective cohort study comprising 2416 Danish adults. Gut, 2003; 52: 186–193.

- Rostom A, Dube C, Wells G, et al. Prevention of NSAID induced gastroduodenal ulcers. Cochrane Database Syst Rev, 2002; (4).

- Russell RI. Non-steroidal anti-inflammatory drugs and gastrointestinal damage-problems and solutions. Postgrad Med J, 2001; 77: 82-88.

- S Rosenstock, T Jørgensen, O Bonnevie, L Andersen. Risk factors for peptic ulcer disease: a population based prospective cohort study comprising 2416 Danish adults Gut, 2003; 52: 186–193.

- Salih BA, Abasiyanik MF, Bayyurt N & Sander E. H. pylori infection and other risk factors associated with peptic ulcers in Turkish patients: a retrospective study. World J. Gastroenterol. WJG, 2007; 13: 3245.

- Sengupta N, Tapper EB, Patwardhan VR, Ketwaroo GA, Thaker AM, Leffler associated with recurrent bleeding after discharge for patients hospitalized with upper gastrointestinal bleeding. Endoscopy, 2016; 48: 9-15.

- Shah A., Tayyaba U. and Rabia, A. Global incidence and prevalence of peptic ulcer disease: A systematic review study. Indo. Am. J. P. Sci, 2019; 6(6): 11267-11273.

- Shephard RJ, Bouchard C. Associations between health behaviours and health related fitness. Br J Sports Med, 1996; 30(2): 94–101.

- Shiotani A, Fujita Y, Nishio K. Low-dose aspirin-associated upper and mid gastrointestinal tract damage and gene polymorphism. Curr Pharm Des, 2015; 21: 5066-72.

- Silen W. Cope's early diagnosis of the acute abdomen. New York: Oxford University Press, 1996.

- Søreide K, Thorsen K, Harrision EM, et al. Perforated peptic ulcer. Lancet, 2015; 386(10000): 1288–98.

- Søreide K, Thorsen K, Søreide JA. Strategies to improve the outcome of emergency surgery for perforated peptic ulcer. Br J Surg 2014; 101: 51-64.

- Sostres C, Carrera-Lasfuentes P, Benito R, et al. Peptic ulcer bleeding risk. The role of Helicobacter pylori infection in NSAID/low-dose aspirin users. Am J Gastroenterol, 2015; 110: 684-89.

- Song EM, Jung HK, Jung JM. The association between reflux esophagitis and psychosocial stress. Dig Dis Sci, 2013; 58: 471–7.

- Stanghellini V. Relationship between Upper gastrointestinal symptoms and Lifestyle, Scand. J. Gastroenterol, 1999; (34) 231.

- Stephen J and Maxine A. Current Medical Diagnosis and Treatment, 47th, Mc Graw Hill, USA, 2008: 518.

- Strand DS, Kim D, Peura DA. 25 Years of Proton Pump Inhibitors: A Comprehensive Review. Gut Liver, 2017; 11: 27-37.

- Sugano K, Tack J, Kuipers EJ, et al. faculty members of Kyoto Global Consensus Conference. Kyoto global consensus report on Helicobacter pylori gastritis. Gut, 2015; 64: 1353-67.

- Sugimoto M, Yamaoka Y. The association of vacA genotype andÂ Helicobacter pylori-related disease in Latin American and African populations. Clin Microbiol Infect, 2009; 15: 835-42.

- Sung JJY, Kuipers EJ, Serag HB. Systematic review: The global incidence and prevalence of peptic ulcerdisease. Aliment Pharmacol Ther, 2009; 29: 938-946.

- Sverdén E, Mattsson F, Lindström D, Sondén A, Lu Y, Lagergren J. Transcatheter arterial embolization compared with surgery for uncontrolled peptic ulcer bleeding: a population-based cohort study. Ann Surg, 2019; 269: 3049.

- Talley NJ, Vakil NB, Moayyedi P. American Gastroenterological Association technical review on the evaluation of dyspepsia. Gastroenterology, 2005; 129(5): 1756–1780.

- Tanikawa C, Urabe Y, Matsuo K, et al. A genome-wide association study identifies two susceptibility loci for duodenal ulcer in the Japanese population. Nat Genet, 2012; 44: 430-34.

- Torpy JM, Lynm C & Golub RM. Peptic ulcer disease. JAMA, 2012; 307: 1329–1329.

- Tsuda M, Asaka M, Kato M, Matsushima R, Fujimoro K, Akino K, Kikuchi S, Lin Y, Sakamoto N. Effect on Helicobacter pylori eradication therapy against gastric cancer in Japan. Helicobacter, 2017; 22: e12415.

- Tulassay Z, Stolte M, Sjölund M, et al. Effect of esomeprazole triple therapy on eradication rates of Helicobacter pylori, gastric ulcer healing and prevention of relapse in gastric ulcer patients.Eur J Gastroenterol Hepatol, 2008; 20: 526-36.

- Turunen JHO, Mäntyselkä PT, Kumpusalo EA, Ahonen RS. Frequent analgesic use at population level: Prevalence and patterns of use. Pain, 2005; 115: 374-81.

- Vaira D, Menegatti M, Miglioli M. What is the role of Helicobacter pylori in complicated ulcer disease? Gastroenterology, 1997; 113: S78-S84.

- Wang FW, et al. Prevalence and risk factors of asymptomatic peptic ulcer disease in Taiwan. World J. Gastroenterol, 2011; 17: 1199–1203.

- Warren JR and Marshall B. Unidentified curved bacilli on gastric epithelium in active chronic gastritis. Lancet, 1983; 1: 1273–1275.

- Wen Q, Liu T, Yu Y, Zhang Y, Yang Y, Zheng R, Li L, Chen R, Wang S. Self-Reported Primary Cooking Fuels Use and Risk of Chronic Digestive Diseases: A Prospective Cohort Study of 0.5 Million Chinese Adults. Environ Health Perspect, 2023 Apr; 131(4): 47002.

- Wong GL, Wong VW, Chan Y, et al. High incidence of mortality and recurrent bleeding in patients with Helicobacter pylori-negative idiopathic bleeding ulcers. Gastroenterology, 2009; 137: 525-31.

- Yamaoka Y, Graham DY. Helicobacter pylori virulence and cancer Pathogenesis. Future Oncol, 2014; 10: 1487-500.

- Yim MH, Kim KH, Lee BJ. The number of household members as a risk factor for peptic ulcer disease. Sci Rep. 2021 Mar 5; 11(1):5274.

- Zaman, WK, Loh SP and Esa NM. Coffee and gastrointestinal health: A review. Malaysian J. of Medicare and Health Sci, 2019; 15 (1): 96-103.

- Zhang BB, Li Y, Liu XQ, Wang PJ, Yang B, Bian DL. Association between vacA genotypes and the risk of duodenal ulcer: a meta-analysis. Mol Biol Rep, 2014; 41: 7241-54.

- Zhang BB, Liu XZ, Sun J, Yin YW, Sun QQ. Association between TNF Î± gene polymorphisms and the risk of duodenal ulcer: a meta-analysis. PLoS One, 2013; 8: e57167.

- Zibima SB, Oniso JI, Wasini KB and Ogu JC. Prevalence trends and associated modifiable risk factors of peptic ulcer disease among students in a university community South-South Nigeria. Int J Health Sci Res, 2020; 10(6): 97-105.

Appendices

Case definitions:

Peptic ulcers: Peptic ulcer disease (PUD) is a deep destruction of the stomach lining or mucosa and/or duodenum, reaching beyond the muscularis mucosa, specifically to the muscle layer owing to the environmental gastric acid synthesis (Guerra *et al*, 2022).

Perforated peptic ulcer: is a hole in the wall of the gastrointestinal tract following a gastric ulcer regularly prompts disastrous outcomes whenever left untreated

Asymptomatic: Refers to infection or disease without symptoms and signs of illness. Many patients with peptic ulcer disease do not have any symptoms during a new or chronic ulcer; they are considered to be asymptomatic. (CDC, 2009)

Emesis (vomiting): clinical defined as the oral eviction of gastrointestinal contents, due to contraction of the gut and muscles of thoracoabdominal wall (Morra *et al*, 2017).

Gastric outlet obstructions: Are a narrowing of the pyloric canal by scarring and swelling of the gastric antrum and duodenum because of PUs. The person often presents with serious vomiting (Lanas *et al*, 2017).

Helicobacter pylori: This microorganism is a Gram negative helical, microaerophilic bacteria which colonizes the antrum and body of the stomach, surviving in its harsh environment through mechanisms of acid resistance and colonization factors (Warren *et al, 1983*).

Endoscopy: Another name is esophagogastroduodenoscopy is a diagnostic and therapeutic device used to visualize the oropharynx, esophagus, stomach, and proximal duodenum. It is one of the most procedures for gastroenterologists (Ahlawat *et al*, 2022).

Consent form:

I am agreeing to participate in the research project; the purpose of this study is to learn more about prevalence and risk factors of PUD in Yemeni student. I know that he /she will be one of the participants who will be studied. I understand that I will be asked to answer health questions. Information's that obtained in this study will help health sector and physicians to determine the most effective public measures for prevention, being a volunteer or refusal to participate in this study will have no effect upon me regarding access to routine medical care.

I understand that my name will not be used in any scientific publications and that my family privacy will be strictly maintained I may withdraw from this study at any time without any loss of health care privileges.

Questionnaire

Prevalence of peptic ulcer disease among Yemeni universities and the most affected group	
1. Name:	**4. Marital status:**
……	Married ( ) Single ( )
2. Age:	**5. Monthly income: (YER)**
Less than 20 ( )	Less than 100,000 ( )
20 to 25 ( )	Between 100 to 200 thousand ( )
25 to 30 ( ) More than 30 ( )	More than 200,000 ( )
3. Sex:	**6. House/living:**
Male ( )	With family ( )
Female ( )	With colleagues ( ) Own house ( )
7. University:	**9. Grade:**
Emirates international university ( )	Frist level ( )
21 September university ( )	Second level ( )
8. Specialty:	Third level ( )
Medicine ( )	Four level ( )
Business administration ( )	Five level ( )
10. Have you been diagnosed with peptic ulcer:	
Yes ( )	No ( )
11. How you have diagnosed:	
Endoscopy ( )	Barium meal ( )
Test for H. pylori in stool or blood ( )	Other tests ( )
Never been diagnosed ( )	
12. Do you have any of these symptoms? Selected all the symptoms you are suffering of:	
Change in appetite ( )	Heartburn ( )
Change in weight ( )	Change in color of stool and/or blood on it ( )
Abdominal bloating ( )	Nausea and Vomiting ( )
Abdominal pain ( )	No symptoms appeared ( )
Early satiety ( )	
13. When dose the symptoms started	
Before starting the university ( )	When started the university ( ) No symptoms appeared ( )

14. Lifestyle Select only one box in each row:					
	No	**Rarely**	**Sometime**	**Often**	**Usually**
• **Smoking (cigarettes, hookah, etc…)**					
• **Get stressed**					
• **Having depression**					
• **Taking sedative (Iboprofen, profenid, etc...)**					
• **Drinking coffee, tea**					
• **Chewing khat**					
• **Drinking soft drink**					

الملخص العربي

تعتبر القرحة الهضمية واحدة من اكثر امراض الجهاز الهضمي انتشاراً حيث تاثر على حوالي ٤ مليون من سكان العالم سنوياً مع حدوث مضاعفات في حوالي ١٠-٢٠%. تم اكتشاف عوامل خطر مؤاخراً للقرحة الهضمية مثل الإجهاد، التدخين، شرب القهوة و مضادات الإلتهاب غير الستيروئيدية. وقد لُحض في بعض الدراسات ان معضم عوامل الخطر التي ذُكرت من قبل ترتبط مع طلاب الجامعات، مما يمكن ان تؤدي الى ارتفاع في معدل الإنتشار وقد يؤدي الى الإعياء المتكرر عند الطلاب والتغيب من الفصول الدراسية و المحاضرات و قد يترتب على ذالك ضعف في الأداء الأكاديمي وقد يؤثر سلبياً على صحة الطلاب لذلك، تحديد اتجاهات انتشار القرحة الهضمية ومعرفة عوامل الخطر المرتبطة بها بين السكان المعرضين للخطر ضرورية لاتخاذ القرارات السريرية و الوبائية اللازمة.

هدفت دراستنا على تقيم معدل انتشار (القرحة) وعوامل الخطر للفئة المتضررة من طلاب الجامعات اليمنية و تحديد اهم الاعراض التي توحي بوجود قرحة و المقارنة مابين الجامعات الخاصة و الحكومية، التخصصات الطبية و الغير طبية، وما بين المستويات الدراسية المختلفة من حيث معدل الإنتشار.

هذه الدراسة مقطعية بين تخصصين من جامعتين تم اختيارهم عشوائياً في مدينة صنعاء; من إبريل الى مايو ٢٠٢٣، تضمنت ٥٦٥ طالباً. تم تصميم الاستبيان لجمع البيانات حول المتغيرات الاجتماعية، الاقتصادية و الديموغرافية، وعوامل الخطر.

كان معدل انتشار القرحة (٢٢.٧ %) بين الطلاب، وكان هناك علاقة ذات دلالة احصائية مابين القرحة و التخصصات في الكليات، مستوى التعليم، التدخين، الإجهاد، الإكتئاب، الدخل الشهري وبين قطاع الجامعات; و بنسبة للفئات العمرية، الجنس، الحالة الاجتماعية، السكن/المعيشة، مضغ القات، شرب الشاي او القهوة او مشروبات الطاقة، واستخدام مضادات الإلتهاب غير الستيروئيدية لم نلاحظ أن هناك علاقة ذات دلالة إحصائية مع القرحة الهضمية; وكان تغير في لون البرازاو وجود دم علية هو اكثر الاعراض التي توحي للقرحة الهضمية بنسب(٦٢.٥%)، يليها القيئ و الغثيان (٥٦%)، الحرقة في البطن (٤٨.٢%)، الم في البطن (٤٧%)، الشبع المبكر(٤٢.٤%)، انتفاخ البطن (٤١.٦%)، تغير في الشهية (٤٠.٥%)، و تغير في الوزن (٣٦.٤%).

في الختام، يوفر هذا البحث رؤى عن معدل إنتشار و اعراض القرحلة الهضمية بين طلاب الجامعات في اليمن، و يقدم توصيات للمسؤلي عن الرعاية الصحية و للمنظمات و الجامعات لتحسين منع و ادارة القرحة الهضمية لدى الطلاب من خلال تنفيذ هذه التوصيات، حيث يمكننا العمل من اجل تقليل عوامل خطر القرحة الهضمية بين طلاب الجامعات و تحسين نتائجهم الصحية الشاملة