



## RESEARCH ARTICLE

## Role of Clinical Decision and Management of Patients Admitted with Perforated Peptic Ulcer to Damascus Hospital (Al Moujtahed), Damascus, Syria

Muhammad Assem Kubtan\*, Alaa Mousa Alsharif and Mhd Nezar Al Sharif

Department of Surgery, Syrian Private University, Damascus, Syria



\*Corresponding author: Muhammad Assem Kubtan, MD-FRCS, Head of Department of Surgery, Faculty of Medicine, Syrian Private University, Al Mezza Highway, Damascus, Syria

### Abstract

**Background:** As far as we know, there has been no previous published studies concerning the incidence of perforated peptic ulcer (stomach, duodenum) and its related risk factors in Syria, and their managements in association with the current Syrian conflict, that precipitated limited access to admitting such cases to over populated beds. This study addressing the burden shouldered by Health professionals, and aimed to determine the Prevalence of perforated peptic ulcer (PPU) in patients admitted to Damascus Hospital between 24/11/2015-4/11/2017, needless to mention also describe the values of making the diagnosis on clinical bases, and based on the principles that we are under the insult of our current dilemma namely civil conflict in Syria, precipitated shortening of all necessities to provide good and acceptable management with hope of achieving absolute cure.

**Materials and methods:** Retrospectively, we examined the data derived from cases of 67 patients who has been admitted and diagnosed as PPU, they had at least one or more risk factor leading to PPU (smoking, stimulants, non-steroidal anti-inflammatory drugs NSAIDS...). Those patients' symptoms almost initiated about 24 hours prior to admission. Their age ranged between 15-80 years-old. Data were analyzed using the Roc Curve and Kendall's tau-b factor and box plots using SPSS 23.0.

**Results:** 48 cases diagnoses proved as perforated duodenal ulcer (P.D.U) (71.6%), and 19 cases diagnoses proved as perforated gastric ulcer (P.G.U) (28.4%) with one perforated gastric ulcer proved to be malignant confirmed by four quadrants biopsies. 22 cases of all proved to be drug-induced perforated ulcer (D.I.P.U) (32.8%) as duodenal or gastric in 13 and 9 cases, respectively. Significant correlation was found between drug-induced perforated ulcer D.I.U, smoking, other stimulants (coffee and tea) and hypertension. Furthermore, a significant correlation was found between taking NSAID and PGU, P.D.U and (D.I.P.U).

**Conclusion:** Almost always diagnosis was made on clinical experience, as there was extreme shortage even complete absence of X-ray films to confirm the diagnosis by seeing gas under the diaphragm which was seen by observing the X-ray monitor, X-ray films are saved for other more important clinical problems mainly orthopedic trauma. The mean age of participants was 48 years old, and the males were more common 87% with a male to female ratio 6:1. The most common PPU was duodenal 71.6% of all patients. Patients taking NSAIDs have a higher risk for developing gastric ulcer ( $P = 0.045$ ) and D.I.U ( $P = 0.000$ ).

### Keywords

Perforated peptic ulcer, Non-steroidal anti-inflammatory drugs (NSAIDS), Perforated gastric ulcer (P.G.U), Perforated duodenal ulcer (P.D.U), Drug-induced perforated ulcer (D.I.P.U)

### Introduction

Peptic ulcer disease (PUD) is characterized by focal defects in the gastric or duodenal mucosa that extend to the submucosa or deeper. It is caused by an imbalance between gastric acid-pepsin and mucosal defense barriers (48,49). Globally, at least 4 million people are affected by peptic ulcer diseases annually [1]. Its incidence ranged from 1.5%-3% [2].

Usually, 10%-20% of patients with PUD may have complications and only 2%-14% of those will have perforation causing severe illness [3]. On review of the international literature, it was found that PUD had a morbidity rate of up to 50% and mortality rate of up to 30% globally [4], while the mortality rate in our study was (13.4%). It is reported that about 5% of patients with PUD will have a perforation during their life [5].

It is difficult to determine the underlying factor causing peptic ulceration due to a variety of risk factors, including *Helicobacter Pylori* (*H. Pylori*), smoking, alcohol, stress, and (NSAIDs) [2,6-14].

This study aimed to analyze and identify the prevalence of PUD in 67 patients whom they have been reviewed at Damascus Hospital (AL Mujtahid) between 24/7/2015-4/5/2017.

## Materials and Methods

This was a retrospective study of the patients who admitted as an emergency to Damascus hospital between 24/11/2015 to 4/11/2017. Our study included all the patients who were diagnosed with gastric or duodenal perforated ulcer (67 patients). All The data were collected by authors to ensure the privacy and all the names were blinded. The data collected included the age of the participants, the time onset of symptoms and history of PUD risk factors. A careful clinical history, physical examination were the core factors in making up the diagnosis, diagnostic procedures were done according to availability, including checking erect chest and abdomen X-rays on X-ray monitor, abdominal ultrasound,) as well as routine laboratory tests (blood count and formula, bleeding and coagulating time, prothrombin time, creatinine, urea & electrolytes, blood sugar, and urinalysis). Surgical interventions was based on clinical impression whether it was compatible with investigational finding or not, since the seriousness of the clinical impression was the most important factor in deciding the steps of the management, Furthermore, days of hospitalization and mortality. Statistical Analysis was done using SPSS 23.0 (SPSS Inc.,).

## Results

67 patients were admitted to the surgical department at Damascus Hospital (Al-Mujtahid) with PPU. They were between 15-80 years old; the mean age was 48-years-old. 58 patients were males (87%) compared to only 9 females (13%) with a prevalence ratio male: female (6:1).

On review we found 48 patients (71.6% of all patients) had perforated duodenal ulcer (P.D.U), 19 patients (28.4%) with perforated gastric ulcer (P.G.U) and 22 patients (32.8%) either duodenal or gastric (13 and 9 cases, respectively) had a drug-induced ulcer (D.I.U), and one patient with P.G.U proved to be malignant and excluded from the current study (Table 1).

All the patients had abrupt onset of acute abdominal pain on presentation. In addition, half of them complained of vomiting, 38.8% had constipation, 17.9% had a fever, 3% had hematemesis and 4.5% had melena. Table 2 summarizes the symptoms of patients at presentation.

Patients taking NSAIDs had an increased chance of having D.I.U ( $p < 0.05$ ) 79.1% of all patients were smok-

ers, 10.4% were alcoholic, 32.8% had stimulants intake (tea, coffee) and 38.8% were taking NSAIDs. Table 3 shows the patients' relation to PUD risk factor's. Only 26 patients (38.8%) have been taking NSAIDs and 9 of them (out of the 26, 34.6%) were between 60-69 years old.

Erect abdomen X-ray including the dome of diaphragm proved that 39 patients (58.2%) with free air under the diaphragm the X-ray report based on the Radiologist experience in reviewing the radiological finding from the monitor due to the lack of X-ray films. Patients were treated either by laparotomy (95.5%) of all patients and by laparoscopy (4.5%), the surgical intervention consisted of the closure of perforation with an omental patch on also four quadrant biopsies taken in PGU.

## Discussion

It is a serious complication of PUD characterized by (PPU) which occurs in about 5% of PUD. PPU is considered a serious insult due to its high morbidity rates and its (1.3% to 20%) mortality rates [1].

16 out of 67 patients in our study were within the age group (30-39) years (23.8% of all) compared to 21 patients in the age group (50-59) years (31.3%). Thus, making the latter age group the most vulnerable and more common to PPU. With varying rates among other ages. 58 male patients (86.6%) and 9 females (13.4%), with a ratio of males to females 6:1 which is inconsistent with global data stating that female are more than half of PPU cases [2].

39 patients (58.2%) were admitted within less than 24 hours, and the remaining 28 patients (41.8%) attend-

**Table 1:** Distribution of types of PPU among patients in this study.

Ulcer type	Patients	Percentage
P.D.U	48	71.60%
P.G.U	19	28.40%
D.I.U (duodenal or gastric)	22	32.80%

**Table 2:** Occurrence of different symptoms in patients of this study.

Symptoms	Number of patients	Percentage
Abdominal pain	67	100%
vomiting	34	50.70%
constipation	26	38.80%
fever	12	17.90%
hematemesis	2	3.00%
melena	3	4.50%

**Table 3:** Relation between patients of this study and PUD risk factors.

Risk Factor	Number of patients	Percentage
Smoking	53	79.10%
Alcohol	7	10.40%
Stimulants	22	32.80%
NSAIDs	26	38.80%

ed after more than 24 hours from the onset of symptoms. In our study, we found that 100% of patients complained of abdominal pain.

PPU has many risk factors such as Helicobacter pylori (H. pylori), smoking, NSAIDs, alcohol, stress, and stimulants (tea, coffee) [1].

NSAIDs are worldwide known to be a risk factor in precipitating PPU (28.29). 1 in 4 patients taking NSAIDs for a long period will get a PUD and 2-4% will have complications (bleeding or perforation) [15-18]. In our study, 26 patients (38.8%) were taking NSAIDs and mostly within the age group (60-69) (13.4%).

23% of PUD is related to smoking [19]. Most likely, due to its effect on pancreatic bicarbonate secretion, which increases gastric acidity [19,20]. In our study, 79.1% (53 patients) were smokers. Alcohol and stimulants also increase PUD risk and, in our study, 10.4% and 32.8% of all patients were ingesting alcohol, respectively.

All patients referred to Radiology Dept for chest and abdomen screening in the erect position urgently and reviewed by an expert Radiologist on the monitor only due to a lack of X-ray films, in the case of acute abdomen presentation with serum amylase/lipase [21]. 75% of PPU have free air under the diaphragm [22]. In our study, 58.2% of patients had free air under the diaphragm.

Closure of perforation is the treatment of choice for PPU [2]. Sixty-four patients (95.5%) were treated with classical surgery while only 3 patients (4.5%) were treated with laparoscopic surgery.

The size of PPU played a major factor in switching to conventional from laparoscopic surgery treatment when the ulcer size is larger than 9 mm [23]. In our study, most of the patients' ulcers were 2-10 mm while only two had an ulcer wider than 15 mm.

## Conclusion

In our study, the mean age of participants was 48-years-old with a predominance of males > females 87% with a male to female ratio 6:1. The most common PPU was duodenal 71.6% of all patients. In our study it was proved that making the diagnosis must be made on the clinical suspicion and experiences of the examining physician when X-ray finding may be unreliable due to shortage of accessory elements (Having only to review radiological Monitor). It is clear that making a prompt clinical diagnosis represent the land stone in the management of the clinical problem in saving the patient and averting the risk of morbidity and mortality associated with PPU.

Patients taking NSAIDs have a higher risk of developing gastric ulcer (P = 0.045) and D.I.U (P = 0.000). Our study demonstrates that abdominal pain and vomiting are the two major and early clinical predictors of the diagnosis and risk.

## Declaration Section

1. Ethics approval and consent to participate: The Ethics approval and consent to participate has been given by the Ethics and Scientific comity of the Syrian Private University.
2. Consent for publication: The Syrian Private University has given the approval of publication of the related project.
3. Data and material: All raw data on which our study is based are available, presenting tables and figures are absolutely true and correct.
4. Competing interests: There is no competing interest.
5. Funding: There is no funding.

## Author's Contributions

M.A.Kubtan, MD-FRCS Main & corresponding author, Alaa Mousa Alsharif, MD, 1<sup>st</sup> co-author, Mhd Nezar Al Sharif, 2<sup>nd</sup> co-author.

Under the supervision and guidance of the Main corresponding author MAK.

- a. AMA and MNA together worked on translating all information's collected from Arabic to English, and they were all the time in contact with Main & corresponding author for close revision and adjustments.
- b. AMA made the collection of information's from the Patients Cases, classified them and wrote the 1st attempt of the manuscript.
- c. MNA reviewed all the statistics and formed the tables.
- d. MAK own the Idea of this study conduct and planned the work, giving the appropriate instruction and modifications for each paragraph of the manuscript.
- e. Main & corresponding author linguistically reviewed the manuscript frequently to bring to the suitable English Language properly.
- f. Only the authors listed on the manuscript contributed towards this article.

## Acknowledgments

Not applicable.

## References

1. Zelickson MS, Bronder CM, Johnson BL, Camunas JA, Smith DE, et al. (2011) Helicobacter pylori is not the predominant etiology for peptic ulcers requiring operation. *Am Surg* 77: 1054-1060.
2. Chung KT, Shelat VG (2017) Perforated peptic ulcer - an update. *World J Gastrointest Surg* 9: 1-12.
3. Lau JY, Sung J, Hill C, Henderson C, Howden CW, et al. (2011) Systematic review of the epidemiology of complicated peptic ulcer disease: Incidence, recurrence, risk factors and mortality. *Digestion* 84: 102-113.
4. Kao LT, Tsai MC, Lin HC, Pai F, Lee CZ (2015) *Weekly*

- pattern of emergency room admissions for peptic ulcers: A population-based study. *World J Gastroenterol* 21: 3344-3350.
5. Vaira D, Menegatti M, Miglioli M (1997) What is the role of *Helicobacter pylori* in complicated ulcer disease? *Gastroenterology* 113: 78-84.
  6. Chey WD, Wong BC, Practice Parameters Committee of the American College of Gastroenterology (2007) American College of Gastroenterology guideline on the management of *Helicobacter pylori* infection. *Am J Gastroenterol* 102: 1808-1825.
  7. Fuccio L, Minardi ME, Zagari RM, Grilli D, Magrini N, et al. (2007) Meta-analysis: Duration of first-line proton-pump inhibitor based triple therapy for *Helicobacter pylori* eradication. *Ann Intern Med* 147: 553-562.
  8. García Rodríguez LA, BarrealesTolosa L (2007) Risk of upper gastrointestinal complications among users of traditional NSAIDs and COXIBs in the general population. *Gastroenterology* 132: 498-506.
  9. Gisbert JP, Pajares JM (2003) *Helicobacter pylori* infection and perforated peptic ulcer prevalence of the infection and role of antimicrobial treatment. *Helicobacter* 8: 159-167.
  10. Lewis JD, Strom BL, Localio AR, Metz DC, Farrar JT, et al. (2008) Moderate and high affinity serotonin reuptake inhibitors increase the risk of upper gastrointestinal toxicity. *Pharmacoepidemiol Drug Saf* 17: 328-335.
  11. Malfertheiner P, Dent J, Zeijlon L, Sipponen P, Veldhuyzen Van Zanten SJ, et al. (2002) Impact of *Helicobacter pylori* eradication on heartburn in patients with gastric or duodenal ulcer disease -- results from a randomized trial programme. *Aliment Pharmacol Ther* 16: 1431-1442.
  12. Schubert ML, Peura DA (2008) Control of gastric acid secretion in health and disease. *Gastroenterology* 134: 1842-1860.
  13. Sonnenberg A, Müller-Lissner SA, Vogel E, Schmid P, Gonvers JJ, et al. (1981) Predictors of duodenal ulcer healing and relapse. *Gastroenterology* 81: 1061-1067.
  14. Vergara M, Catalán M, Gisbert JP, Calvet X (2005) Meta-analysis: Role of *Helicobacter pylori* eradication in the prevention of peptic ulcer in NSAID users. *Aliment Pharmacol Ther* 21: 1411-1418.
  15. Laine L (1996) Nonsteroidal anti-inflammatory drug gastropathy. *Gastrointest Endosc Clin N Am* 6: 489-504.
  16. Larkai EN, Smith JL, Lidsky MD, Graham DY (1987) Gastrointestinal mucosa and dyspeptic symptoms in arthritic patients during chronic nonsteroidal anti-inflammatory drug use. *Am J Gastroenterol* 82: 1153-1158.
  17. Singh G (2000) Gastrointestinal complications of prescription and over-the-counter nonsteroidal anti-inflammatory drugs: A view from the ARAMIS database. *Arthritis, Rheumatism, and Aging Medical Information System*. *Am J Ther* 7: 115-121.
  18. Bombardier C, Laine L, Reicin A, Shapiro D, Burgos-Vargas R, et al. (2000) Comparison of upper gastrointestinal toxicity of rofecoxib and naproxen in patients with rheumatoid arthritis. VIGOR Study Group. *N Engl J Med* 343: 1520-1528.
  19. Kurata JH, Nogawa AN (1997) Meta-analysis of risk factors for peptic ulcer. Nonsteroidal antiinflammatory drugs, *Helicobacter pylori*, and smoking. *J Clin Gastroenterol* 24: 2-17.
  20. Stabile BE, Passaro E (1984) Duodenal ulcer: A disease in evolution. *Curr Probl Surg* 21: 1-79.
  21. Nuhu A, Madziga AG, Gali BM (2009) Acute perforated duodenal ulcer in Maiduguri: Experience with simple closure and *Helicobacter pylori* eradication. *West Afr J Med* 28: 384-387.
  22. Anbalakan K, Chua D, Pandya GJ, Shelat VG (2015) Five year experience in management of perforated peptic ulcer and validation of common mortality risk prediction models - are existing models sufficient? A retrospective cohort study. *Int J Surg* 14: 38-44.
  23. Kim JH, Chin HM, Bae YJ, Jun KH (2015) Risk factors associated with conversion of laparoscopic simple closure in perforated duodenal ulcer. *Int J Surg* 15: 40-44.