Original Research Article

Rise in incidence of perforated peptic ulcer in young-change in the trends in etiology

Mohammed Muzamil Pasha, Divya R. I.*

Department of General Surgery, Bangalore Medical College and Research Institute, Bangalore, Karnataka, India

Received: 08 April 2022
Revised: 22 May 2022
Accepted: 24 May 2022

*Correspondence:
Dr. Divya R. I.,
E-mail: Divya_swamy@hotmail.com

ABSTRACT

Background: A total of 50 patients who were admitted and treated for perforated peptic ulcer, from February 2021 to December 2021, were included in the study to study the recent changes in the trends of etiology of perforated peptic ulcer in the young.

Methods: Patients who were diagnosed and treated with perforated peptic ulcer (prepyloric perforation and duodenal perforation) from February 2021 to December 2021 were included in the study. Data regarding dietary habits, use of addictive recreational substances, smoking, tobacco use, and alcohol consumption were collected and tabulated to study the recent trend in the etiology of perforated peptic ulcer in the young.

Results: Higher incidence of peptic ulcer perforation seen in young males with history of use of recreational drugs

Conclusions: There is a rise in cases of perforated peptic ulcers in young males with a history of use of habit-forming substances.

Keywords: Perforated peptic ulcer, Recreational drug use, Duodenal perforation

INTRODUCTION

Peptic ulcers occur when there is an imbalance between the gastric acid production and the gastro-duodenal defense mechanisms. Perforated peptic ulcer is a life-threatening condition, with an incidence of 7/1,00,000 population per year.1 It usually affects middle and old age groups. There are many incriminating agents in the formation of peptic ulcer, such as use of steroids, NSAIDs, stress, Helicobacter pylori infection, alcohol consumption, and smoking.2 It is one of the most common surgical emergencies. Surgery is still the mainstay treatment for perforated peptic ulcers.

Objectives

The purpose of our study was to correlate the rise in cases of perforated peptic ulcer in young patients with the use of habit-forming recreational drugs and substances.

METHODS

It is an observational study which was conducted in a tertiary care center in Bangalore, from February 2021 to December 2021. A recent rise in perforated peptic ulcer was seen in young patients. A total of 50 patients who were diagnosed and treated for perforated peptic ulcer during this period were selected by random sampling and were included in the study. The patients presented to the surgical emergency with history of pain abdomen, with tenderness, guarding and rigidity present on examination. The chest and abdomen X-ray showed presence of gas under the domes of diaphragm (Figure 1). All patients underwent emergency exploratory laparotomy and modified Graham’s omental patch repair. The data regarding dietary habits, use of addictive recreational substances, smoking, use of tobacco, and alcohol consumption, were collected and tabulated.
RESULTS

Out of the 50 patients included in the study, 32 patients (64%) were young patients, aged 18-30 years, 10 patients (20%) were middle aged between 31-50 years, and 8 patients (16%) were old patients above 50 years of age. (Figure 2).

DISCUSSION

In our study, the majority of patients who presented with perforated peptic ulcers were young patients, aged between 18-30 years of age, as compared to middle and old aged patients. Prepyloric perforation (Figure 4) was more common than duodenal perforation (Figure 5).
A number of studies proved that smoking is a major cause of peptic ulcer. Nicotine from smoking is known to have several effects on the GI tract. A) High concentration of nicotine increases gastric acid secretion in stomach and decrease bicarbonate production in duodenum. This process interferes with the defense mechanism in the stomach and duodenum leading to ulcer development, B) Platelet activating factor [PAF] is a vasoconstrictive phospholipid, it increases due to nicotine effect contributing more incidence of peptic ulcers in tobacco consumers and C) Nicotine causes immediate vasoconstriction and ischemia which reduces mucosal resistance resulting in perforation.10–13

**CONCLUSION**

There is an increase in the use of recreational, habit-forming drugs amongst young people, which is likely to be a new etiological factor for perforated peptic ulcers in the young. Further study is needed in the area of its pathophysiology.

**Funding:** No funding sources  
**Conflict of interest:** None declared  
**Ethical approval:** The study was approved by the Institutional Ethics Committee

**REFERENCES**

1. Watkins RM, Dennison AR, Collin J. What has happened to perforated peptic ulcer? Bri J Surg. 1984;71(10):774-6.

2. Ahmed N. 23 years of the discovery of *Helicobacter pylori*: is the debate over? Ann Clin Microbiol Antimicrob. 2005;4:17.

3. WHO. WHO urges more countries to require large, graphic health warnings on tobacco packaging: the WHO report on the global tobacco epidemic, 2011 examines antitobacco mass-media campaigns. Cent Eur J Public Health. 2011;19:133-51.

4. Shin YY, Cho CH. Nicotine and gastric cancer. Alcohol. 2005;35:259-64.

5. Chu KM, Cho CH, Shin VY. Nicotine and gastrointestinal disorders: its role in ulceration and cancer development. Curr Pharm Des. 2013;19:5-10.

6. Peluso ME, Munnia A, Srivatanakul P, Jedpiyawongse A, Sangrajrang S, Ceppi M et al. DNA adducts and combinations of multiple lung cancer at-risk alleles in environmentally exposed and smoking subjects. Environ Mol Mutagen. 2013;54:375-83.

7. Jain G, Jaimes EA. Nicotine signaling and progression of chronic kidney disease in smokers. Biochem Pharmacol. 2013;86:1215-23.

8. Li W, Zhou J, Chen L, Luo Z and Zhao Y. Lysyl oxidase, a critical intra- and extra-cellular target in the lung for cigarette smoke pathogenesis. Int J Environ Res Public Health. 2011;8:161-84.

9. Zhang L, Ren JW, Wong CC, Wu WK, Ren SX, Shen J et al. Effects of cigarette smoke and its active components on ulcer formation and healing in the gastrointestinal mucosa. Curr Med Chem. 2012;19:63-9.

10. Maity P, Biswas K, Roy S, Banerjee RK, Bandyopadhyay U. Smoking and the pathogenesis of gastroduodenal ulcer - recent mechanistic update. Mol Cell Biochem. 2003;253:329-38.

11. Miyaura S, Eguchi H, Johnston JM. Effect of a cigarette smoke extract on the metabolism of the proinflammatory autacoid, platelet-activating factor. Circ Res. 1992;70:341-7.

12. Iwao T, Toyonaga A, Ikegami M, Oho K, Sumino M, SakakiM et al. Gastric mucosal blood flow after smoking inhealthy human beings assessed by laser Doppler flowmetry. Gastrointest Endosc. 1993;39:400-3.

13. Sørbye H, Svanes K. The role of blood flow in gastricmucosal defence, damage and healing. Dig Dis. 1994;12:305-17.

**Cite this article as:** Pasha MM, Divya RI. Rise in incidence of perforated peptic ulcer in young-change in the trends in etiology. Int Surg J 2022;9:1156-58.