

Research

Open Access

Association of Peptic Ulcer Disease with Obesity, Serum Insulin Level and Lipid Profile

Foroogh Forghani^{*1} and Mahboube Mirhashemi²

¹Department of Gastroenterology, Shahid Beheshti University of Medical Sciences, Tehran, Iran ²Internal Medicine Department, Shahid Beheshti University of Medical Sciences, Tehran, Iran

*Corresponding author: Forghani F, Department of Gastroenterology, Shahid Beheshti University of Medical Sciences, Tehran, Iran, Tel: +9891218651794; E-mail: foroogh_f200547@yahoo.com

Received date: April 07, 2021; Accepted date: April 21, 2021; Published date: April 28, 2021

Copyright: © 2021 F Forghani, et al. This is an open-access article distributed under the terms of the Creative Commons Attribution License, which permits unrestricted use, distribution, and reproduction in any medium, provided the original author and source are credited.

Abstract

Background: PUD is a common and important type of gastrointestinal disease. Determination of contributing factors for PUD is an important issue for the development of preventive approaches. In this study, the association of Peptic Ulcer Disease with Obesity and Serum Insulin and Lipid Profile was studied.

Methods: In this case control study in Modarres Hospital in Tehran in 2019 among 170 consecutive subjects including 90 patients with PUD and 80 cases without it were enrolled and sampling tools were checklists. Serum Insulin and Lipid Profile and BMI were compared across the groups

Results: The results in this study demonstrated that Serum Insulin and Lipid Profile and BMI had no statistically significant difference between those patients with and without presence of PUD (P>0.05).

Conclusion: This study showed no statistically significant difference between those patients with and without the presence of PUD from points of Serum Insulin and Lipid Profile and BMI.

Keywords PUD; Insulin; Lipid Profile; BMI

Abbreviations PUD (Peptic Ulcer Disease); BMI (Body Mass Index); WHtR (Waist/height ratio); WC (Waist Circumference); H. pylori (Helicobacter Pylori); TG (Triglyceride); LDL (Low Density Lipoprotein Cholesterol); HDL (High Density Lipoprotein Cholesterol); COPD (Chronic Obstructive Pulmonary Disease); CRF (Chronic Renal Failure); NSAIDs (Non Steroid Anti Inflammatory Drugs); FBS (Fasting Blood Sugar)

Introduction

Peptic Ulcer Disease (PUD) is a common severe condition in gastrointestinal tract with 4.1% global prevalence. Despite the controversy, recent studies show a relationship between PUD and environmental factors such as lifestyle, stress, obesity and nutrition in terms of triggering or the chronicity of the disease [1,2]. About 10% of the global population experience PUD at least once in their lives to achieve around 15 thousand death a year and waste billion dollars each year paid for the direct and indirect burden of PUD [3]. Lots of studies tried to assess the relationship between PUD and anthropometric factors as well as individual manners and lifestyle to suggest body mass index (BMI), waist/height ratio (WHER), and waist circumference (WC) as causative factors but some deny this claim [1,4-8].

Despite many studies guess that PUD may associate with obesity, insulin intolerance and lipid profile, there is no global acceptance in this regard directing the current study to the reassessment of the named factors in terms of any causative potential for PUD among our patients.

Materials and Methods

Participants

Through a case control design, this study assessed the profile lipid, insulin serum level and obesity in relationship with PUD. People who referred to the gastrointestinal clinic at Modarres hospital in Tehran enrolled the study regarding inclusion and exclusion criteria. Dyspepsia was the chief complaint of our patients to lead them to experience endoscopy based on the relevant indications and the patients participated in case or control group based on the endoscopy results. Two groups aged between 20-60 containing 90 (42 male/48 female) confirmed PUD by endoscopy in the case group and 80 healthy people (38 male/42 female) in the control group were compared regarding the named risk factor. There was no significant difference among two groups regardind BMI. People with PUD enrolled the case group and the rest made up the control group. Previous history or treatment of PUD, diabetes mellitus, Lipitor tablet intake, using diabetes medications, smoking and addiction were our main exclusion criteria in addition to anti platelets, anticoagulants, and NSAIDs while diseases such as COPD, chronic renal failure (CRF), and cirrhosis were also excluded.

Outcome measures

Fasting blood sugar (FBS), serum insulin, triglyceride, cholesterol, LDL, and HDL were checked after endoscopy results were clear and the participants contributed to their suitable groups. Demographics as well as weight, height, and BMI were also recorded. All the data were gathered for cases and controls.

Statistics

The variables were checked by Kolmogorov Smirnov test regarding normal distribution before being analyzed through an independent two sample t test and Mann Whitney test as a parametric test for normally distributed and non-normal distributed variables, respectively. The association of the variables was checked by Pearson's and Spearman's correlation parameters. The current study used a 95% confidence interval beside type one error of α =0.05 and a significance of 0.05 to have 0.8 power.

Ethics

All the individual private data were safely kept by the principal investigator and there was no extra test or charge for the patients. There was no obligation to participate or maintain the study and the patients were free to quit the study without penalty whenever they wished.

Results

The total number of the participants was 170 including 90 PUD patients as case and 80 patients without PUD as control. There was no statistical difference between the groups regarding age and sex distribution. The mean age was 44.74 ± 16 and 48.18 ± 15.6 years in the case and control group, respectively (P>0.05). Males made up 46.7% and 47.5% of cases and controls while 53.3% of cases and 52.5% of controls were females (P>0030.05). The Chi-square test showed no difference between the groups in terms of past medical history or recently administered medications. Although some points in this regard would be discussed later as can be concluded from figure, BMI did not statistically differ comparing the studied groups. Underweight participants as diagram Fasting blood sugar, total cholesterol, LDL, HDL, TG, and serum insulin were measured and compared but no one was different between the groups (P>0.05) summarize the findings (Figure 1) (Tables 1 and 2). Although the chisquare test showed the same findings in terms of PUD and past medical history, the rate of PUD was a bit more in the group of people who had no comorbidities as can be seen in (Figure 2). Drug history showed no difference between the groups.

	PUD	Patients (n)	Significance
Male	Positive	42	0.913
	Negative	38	
Female	Positive	48	
	Negative	42	
РМН	Positive	20	0.065
	Negative	28	
DH	Positive	20	0.965
	Negative	18	

Table 1: Comparison of sex, past medical history and drug history among the participants regarding the existence of peptic ulcer disease.

	PUD	Participants (n)	Mean±SD	Significance
Age	Positive	90	44.74 ± 16.0	0.16
	Negative	80	48.18 ± 15.6	
ВМІ	Positive	90	27.35 ± 7.08	0.577
	Negative	80	27.03 ± 7.28	
LDL	Positive	90	101.92 ± 31.63	0.164
	Negative	80	109.89 ± 130.1	
HDL	Positive	90	40.32 ± 12.0	0.355
	Negative	80	44.08 ± 20.7	
TG	Positive	90	156.69 ± 83.7	0.359
	Negative	80	148.83 ± 95.7	
Insulin	Positive	90	12.89 ± 5.74	0.235
	Negative	80	11.91 ± 4.92	

Table 2: Comparison of quantitative factors among the participants regarding the existence of peptic ulcer disease.



Figure 1: Distribution of laboratory parameters showing the mean values of FBS, total cholesterol, LDL, HDL, TG and insulin regarding the existence of PUD in participants

Page 2 of 4



Figure 2: The frequency of PUD in participants with and without past relevant medical history

Discussion

The current study tried to find any correlation between obesity, lipid profile, serum insulin, and PUD. There was no correlation in this matter although many studies declare and some regret such a relationship. Studies believe that increased serum lipids or atherogenic lipid profiles may be resulted by infections and consequent inflammatory reactions in the body but there is no performance to investigate this relationship directly between PUD and lipid profile [9]. Published an article following a study focused on obesity and PUD among more than 32,000 through a big cohort between 2005 and 2017. They found that obesity did not correlate with a gastric ulcer when adjusted for smoking habits, physical activity, alcohol intake, fasting blood sugar, TG, HDL, LDL, H. pylori status, and some medications. This was while duodenal ulcer had a lower rate in obese patients than in non-obese individuals [10]. studied serum insulin and C peptide in healthy people and patients with duodenal ulcer in 1990 in Russia to explain that the named hormones rise in the named condition but our work included all types of gastric ulcers resulting in nothing to report in this regard [11]. A decade later, indicated that insulin level had increased in PUD patients before surgery experiencing a dramatic reduction after repair surgery among 74 cases in Russia [12].

It seems there is a force need to a cohort to follow up PUD patients while monitoring life style, BMI, lipid profile and insulin like what [13]. Reported in Ukraine in 1996 studying 120 duodenal ulcer patients to find age related changes in some hormones such as gastrin, insulin, and glucagon beside gastric mucosal layer when the patients were treated. Despite similar findings to our results in terms of gastric and gastro duodenal ulcer in males [14]. Reported in 2000 that men with duodenal ulcers got rid of their ulcers after increased physical exercise and reduced BMI in Texas, US. Participants' BMIs showed a few unexpected hints in through which diagram A illustrates almost all the underweighted individuals were in the group without PUD (Figure 3). In this regard, diagram B of the same figure indicates an increased frequency of PUD in participants with normal weight; although it may be due to a higher number of participants in that weight category.



Figure 3: Comparison of the frequencies of PUD and BMI among the participants. Diagram A illustrates the distribution of different bodyweight categories regarding the existence of PUD. Diagram B shows the frequency of PUD in different weight categories based on BMI.

In a prominent work in 2014, more than 47 thousand of patients were studied by [1]. In the United States to find a total number of 600 PUD cases to be assessed for central obesity and global obesity to suggest that any shape of obesity correlated with increased risk of PUD, especially gastric ulcer as well as H. pylori negative ones. Several studies indicated that genetic characteristics of the microorganisms existing in the human gastrointestinal tract are in a relationship with nutrition and obesity while affecting on hormonal paths, insulin resistance, and fatty tissue aggregation in the body which was also raised in mice [4,15-18].

Conclusion

The current study, despite many supportive types of research, found no correlation between PUD and obesity, serum insulin, and lipid profile. This means the fact that there is no need to monitor or control the mentioned factors as the main risk factors of PUD. However, this is worth thinking about a multi central cohort study for decades in order to put an end to the controversy in this regard.

Study limitations

The current study was done in a short time at a single hospital resulting in a limited sample size. Also, many of the patients declined to participate due to blood sampling and time wasting such as elongated hospital stay or later serial medical visits.

References

1. BoylanMR, Huang ES, Chan AT (2014) Measures of adiposity is associated with an increased risk of peptic ulcer. Clin Gastroenterol Hepatol.12:1688-1694.

Page 3 of 4

- 2. Kim J, Kim KH, Lee BJ (2017) Association of peptic ulcer disease with obesity, nutritional components, and blood parameters in the Korean population. PloS one. 12.
- Babaee G, Keshavarz M, Shaigan M (2007) Effect of health education program on quality of life in patients undergoing coronary artery bypass surgery.45:69-75.
- 4. Raybould HE (2012) Gut microbiota, epithelial function and derangements in obesity. J Physiol.590:441-446.
- 5. Weatherall R, Shaper AG (1988) Overweight and obesity in middle-aged British men. Eur J Clin Nutr. 42:221-231.
- 6. Patel NM, Khan B, Gerkin R, Kiafar C, Ramirez FC (2011) Obesity is associated with high risk stigmata of peptic ulcer disease. Gastroenterology.140:724-731.
- Rosmond R,Lapidus L, Marin P, Bjorntorp P (1996) mental distress, obesity and body fat distribution in middle-aged men.Obes Res. 4:245-252.
- Tsai WL, Yang CY, Lin SF, Fang FM (2004) Impact of obesity on medical problems and quality of life in Taiwan. Am J Epidemiol. 160:557-565.
- Chimienti G, Russo F, Lamanuzzi B, Nardulli M, Messa C, et al (2003) Helicobacter pylori is associated with modified lipid profile: impact on Lipoprotein. Clinical biochemistry.36:359-365.

- 10. Pyo JH, Lee H, Kim JE (2019) Obesity and risk of peptic ulcer disease: A large-scale health check-up cohort study. Nutrients.11:1288.
- 11. Valenkevich LN, Zaichik A, Eremina E (1990) pancreatic hormonal function and proteolytic activity in peptic ulcer. Vrach Delo.12:63-65.
- 12. Babalich AK (2001) Blood insulin levels in surgical treatment of peptic ulcer. Lik Sprava.6:87-89.
- Haidychuk VS, Kolomoiets M (1996) The level of gastrin, insulin and glucagon in the blood and the morphometric characteristics of the gastric mucosa in peptic ulcer patients of different ages. Lik Sprava. 12:80-84.
- 14. Cheng Y, Macera CA, Davis DR, Blair SN (2000) Physical activity and peptic ulcers. Does physical activity reduce the risk of developing peptic ulcers? West J Med.173:101-107.
- 15. Tilg H, Moschen AR, Kaser A (2009) Obesity and the microbiota. Gastroenterology.136:1476-1483.
- 16. Turnbaugh PJ, Gordon JI (2009) The core gut microbiome, energy balance and obesity. J Physiol. 587:4153-4158.
- 17. Ley RE (2010) Obesity and the human microbiome. Curr Opin Gastroenterol.26:5-11.
- Ding S, Chi MM, Scull BP, Rigby R, Schwerbrock NM, et al (2010) High-fat diet: bacteria interactions promote intestinal inflammation which precedes and correlates with obesity and insulin resistance in mouse. PLoS One.5:12191.

Page 4 of 4