Social Background of Peptic Ulcer Patients in Mosul City: A Case-control Study

Dr. Humam G. Al-Zubeer 1, Dr. Rafe H. Al-Kazzaz 2, Dr. Rami M. Adil Khalil 3, Dr. Duha E. Al-Matheedee 4

1 Lecturer - Department of Community Medicine / College of Medicine / University of Mosul.
2 Physician – Ibn Sena Teaching Hospital / Nineveh Directorate of Health / Mosul.
3 Assistance Professor - Head of Department of Medicine / College of Medicine / University of

Abstract

The incidence of gastric and duodenal ulcer is characterized by pronounced geographical and temporal variations which suggest that important environmental factors must play a part in the etiology of both diseases. The aim of the present study is to determine the sociological risk factors in the development peptic ulcer in the study sample in Mosul city.

Study design: Age and sex matched case-control design.

Study period: 15th May 2009 to 15th May 2010.

One hundred eighty new cases of peptic ulcer were collected from endoscope unit in Ibn-Sena General Teaching Hospital in Mosul and 240 controls persons (not having peptic ulcer) community and hospital based age and sex matched control group include healthy peoples; medical students and their families and friends also relatives of patients from other hospitals in the Mosul Medical City were also collected. Interview questionnaire was adopted as a data collection tool.

Outcome measures: Odd's ratio and its 95% confidence interval.

Results: The mean age of the study sample was 41.8 ± 6.45 years for cases and 39.9 ± 7.74 years for controls. Regarding social habits; cigarettes smoking was found to be highly associated with the development of peptic ulcer disease and it was dose-related (OR=2.30, 95% CI: 1.20; 4.39, p= 0.011 for heavy smokers), and also for coffee drink ≥ 3 cups /day (OR= 2.20, 95% CI: 1.11; 4.35, p= 0.021). Whereas, high fiber diet (vegetables, fruits, legumes and cereals fibers) appears to be protective against occurrence of peptic ulcer disease (OR=0.52, 95% CI: 0.35; 0.77, p= 0.001). In respect to personal characteristics; positive family history and personality type A were found to have positive impact in the development of peptic ulcer disease (OR=2.94, 95% CI: 1.65; 5.26, p= 0.000) and (OR=1.72, 95% CI: 1.10; 2.80, p= 0.028) respectively, as well as stressful life events (OR=2.15, 95% CI: 1.45; 3.19, p= 0.000). While high crowding index, occupation, level of education, residence (urban or rural) and excessive tea drinking appear to do nothing in the occurrence of peptic ulcer disease.

Conclusion: The etiology of peptic ulcer disease is multifactorial. Cigarettes smoking, coffee drinking, positive family history of peptic ulcer disease, low fiber diet, stressful life events and personality type A were the main risk factors for incident peptic ulcer disease in Mosul studied sample.

Key words: ***peptic ulcer, risk factors, case-control study.***

Introduction

A peptic ulcer is damage to the lining of the stomach, duodenum, or esophagus due to corrosion caused by digestive juices secreted by stomach cells. The incidence of gastric and duodenal ulcer is characterized by pronounced geographical and temporal variations which suggest that important environmental factors must play a part in the etiology of both diseases. A widespread belief holds that peptic ulcer is related to emotional stress and, therefore, more likely to occur among managers and other professionals (1, 2).

Peptic Ulcer Disease (PUD) is one of the most common disorders of the gastrointestinal tract (3). Currently, the incidence of duodenal ulcer is increasing rapidly in most of the developing world, while in the west it is continuing to decrease. Despite this decline, duodenal ulcer remains an important disease with similar rates in men and women. About 300,000 new cases are diagnosed each year in the United States (4). However, the decline has been more pronounced for uncomplicated duodenal ulcer than for ulcers complicated by hemorrhage or perforation (5), and the death rate from duodenal ulcer has remained relatively constant at about 1/100,000 each year since 1980 (4, 6).

Recently, several reports (5-7) have provided evidence that infection with Helicobacter pylori increases the risk of duodenal ulcer. Possibly 75 to 100 percent of duodenal ulcer patients have evidence of H. pylori infection (7). Despite the high prevalence of this infection, it is not known why only a small number of those infected eventually develop ulcers. This observation suggests that H. pylori infection is not sufficient to cause ulceration, and that other factors might also be of importance (8). Diet has long been suspected to be associated with duodenal ulceration, and dietary differences have been cited to explain the geographic variation in this disease. A dietary role in the pathogenesis of duodenal ulcer is supported by studies in humans (8-10). In most case-control studies, patients with duodenal ulcer have reported lower fiber intake or that duodenal ulcer heals faster with a high fiber diet. Some investigators (10, 11) have suggested that the decline in incidence of peptic ulcer is explained by the coinciding increase in the consumption of linoleic acid from vegetable oil in the Western diet. However, the findings of case-control studies have mainly been concerned with the diet after the onset of symptoms. In addition, failure to control for potential risk factors, such as alcohol consumption, smoking, coffee drinking, body composition, physical activity, other dietary components, and use of nonsteroidal anti-inflammatory drugs (NSAIDs) and aspirin, limits the interpretation of most case-control studies (5, 11).

Although, stress is no longer considered a cause of ulcers, studies still suggest that stress may predispose a person to ulcers or prevent existing ulcers from healing. Some experts estimate that social and psychological factors play a contributory role in 30 - 60% of peptic ulcer cases, whether they are caused by H. pylori or NSAIDs. Some experts even believe that the anecdotal relationship between stress and ulcers is so strong that treatment of psychological factors is warranted (1).

The aim of the present study is to determine the sociological risk factors in the development peptic ulcer in the study sample in Mosul city and to calculate the magnitude of risk of having peptic ulcer due to unhealthy social and dietary habits as smoking, low fiber diet and sedentary life style.

Materials and Methods

Ethical consideration: Before starting data collection, administrative and ethical agreements were obtained from Nineveh Directorate of Health and a verbal consent was obtained from the potential participants.

Study design: Case – control study was adopted.

Study period: 15th May 2009 to 15th May 2010.

Participants and study setting:
Cases of PU were collected from Endoscopy Unit in Ibn-Sena General Teaching Hospital in Mosul city. Controls: (not having PU) Community and hospital based controls group include healthy peoples; medical students and their families and friends and also relatives of patients (not having PU or gastritis) from other hospitals in the Mosul Medical City were also collected.

Case definition:

- New cases of peptic ulcer diagnosed by upper GIT endoscopy were included in this study.
- Any age group and both sexes.
- Superficial or deep ulcer.
- Site: duodenal, gastric or esophageal ulcers.
- Single or multiple ulcers.
- With or without biopsy sample during the endoscope.

Control definition:

- Healthy persons or patients not complaining from any GIT symptoms.
- Age and sex matched controls.

Method of sample collection and sample size:
Cases of PU were interviewed by endoscope physician after confirming the diagnosis and discussing with them the purpose of the research. This physician works 3 days per week in endoscopy unit. Every day, about 2 – 3 cases of PU were diagnosed (6 – 8 / week). According to this time table, there will be more than 220 potential PU to be enrolled during one year period; started from 15th May 2009. Another convenient (non-random) sample composed of 240 "age and sex matched" control subjects were interviewed by the first and fourth authors and by eight trained 4th year medical students during their practical training sessions on communication skills and research's performance in the Community Medicine Department at that year. Frequency matching method was applied in the collection of those controls. The inter-rater variability was evaluated for the medical students and it was 72%.

Data collection tool: A questionnaire was prepared and submitted to the staffs of Community Medicine Department - College of Medicine - University of Mosul in the preliminary stage and the necessary changes were performed. It compose of demographic and other social and life style variables, which included age, sex, marital status, residence, occupation, smoking, tea drink, coffee drink, alcohol consumption, family history of peptic ulcer, levels of literacy, diet history (including dietary fibers intake, fatty and salty meals), leisure time energy expenditure, housing condition, stressful life events and personality type.

Data were tabulated and analyzed using Minitab version 14.1 software program. Simple proportions, percentages and means were used. Chi-square test was applied for categorized variables. Odd's Ratio (OR) were calculated with 95% Confidence Interval (CI) by using Confidence Interval Analysis software program. P-value ≤ 0.05 were considered significant throughout data analysis.

Results

Overall, 180 PUD cases were enrolled in the study with a response rate of 82% for cases. The mean age of the study sample was 41.8 ± 6.45 years for cases and 39.9 ± 7.74 years for controls.

Table (1) shows the distribution of cases and controls according to socio-demographic characteristics and revealed in regarding to marital status that being single had positive association with development of PUD (OR=2.13, 95% CI: 1.36; 3.31, p= 0.001), while urban citizens appeared not to be different in the relation with PUD from rural citizens. Regarding educational factor, the present study revealed that this factor had no impact in the causation process of PUD (OR=1.12, 95% CI: 0.30; 4.26, p= 0.868). However, the effect of occupation was contradictory, except for students who

appear to be protected from the occurrence of PUD (OR=0.48, 95% CI: 0.24; 0.96, p= 0.035). Finally, in regarding to social identity, those with positive family history were around 3 times more prone to develop PUD than those with negative family history, (OR=2.94, 95% CI: 1.65; 5.26, p= 0.000).

Table (2) reveals the effect of unhealthy dietary habits and occurrence of PUD, where unhealthy dietary habits (fatty, caloric and salty foods) appear not to be associated with the development of PUD (OR=1.28, 95% CI: 0.87; 1.88, p= 0.215), whereas high fibers intake (vegetables, fruits, legumes and cereals fibers) was highly protective against PUD etiology, (OR=0.52, 95% CI: 0.35; 0.77, p= 0.001).
Although a small proportion of cases (12.8%) and controls (6.3%) reported consuming large amount of coffee, an OR of 2.20 suggest positive association between coffee drink (≥ 3 cups /day) and PUD, (95% CI: 1.11; 4.35, p= 0.021).
Similarly, a small proportion of alcohol drinkers were insufficient to prove the causal effect (OR=2.72) of this factor; CI: 0.67; 11.04. On the other hand, tea drink ≥ 4 cups /day appear to be do nothing with the occurrence of PUD.
A possible risky effect of sedentary leisure time energy expenditure in the etiology of PUD was seen (OR= 1.38). However, this effect was not significant (CI: 0.86; 2.22, p= 0.185). The habit of cigarettes smoking was moreover associated with the occurrence of PUD. Furthermore, a dose-response relationship was seen between PUD and cumulative number of cigarettes smoke / day and it is evident in moderate (10 – 20 cigarettes) and heavy (≥ 21 cigarettes) smokers with OR of 2.02 and 2.30, respectively.
Regarding psychological makeup, personality type A appeared to be associated with the occurrence of PUD (OR=1.72, 95% CI: 1.10; 2.80, p= 0.028). Also, stressful life events were highly associated with the development of PUD (OR=2.15, 95% CI: 1.45; 3.19, p= 0.000).
Lastly, high crowding index again appear to do nothing with the occurrence of PUD.

Discussion

H. pylori infection is known to be very common worldwide. However, only small percentages of the infected population develop PUD. Several risk factors such as smoking, alcohol, NSAIDs, sedentary life, stress, and aspirin intake are shown to play a role in the disease outcome. The characterization of these factors according to their strength of effect was found to vary among different populations (1, 12). Schlemper et al. (13) indicated that the proportion of ulcers that can be attributed to H. pylori infection is likely to be higher in countries where H. pylori infection is more common (e.g. Japan). They showed that H. pylori had greater impact on ulcer morbidity in the Japanese than in the Dutch population.
The present study is one of the first analytic case-control studies in Iraq which has examined the impact of several risk factors for incident PUD (apart from H. pylori infection and NSAIDs). The most important findings were that cigarettes smoking, coffee drinking, positive family history of PUD, low fiber diet, stressful life events and personality type A were the main risk factors for incident PUD in Mosul population. The role of age and sex as risk factors were not evaluated; as they were matched from the beginning of the study; to minimize the effect of them as confounding variables and to demonstrate the independent effect of other variables whenever possible.
According to clinical observations, smokers were found more likely to develop ulcers that were difficult to heal and had higher incidence of relapses (14). Thus smoking, although not an independent ulcerogen, may act by augmenting the harmful effects of H. pylori, both by adversely affecting upper gastrointestinal mucosal protection and by increasing the risk of H. pylori infection (1, 13, 14). It was reported that smoking more than 15 cigarettes per day compared with never smoking increased the risk of a perforated ulcer more than threefold (15). Recent studies also showed a significant modification effect between smoking and H. pylori infection, and suggested that smoking only increases the risk of peptic ulcers in those who were infected with H. pylori (1, 15). Similarly Johnson et al. (16) performed a prospective population study in Norway during 1994 and reported that cigarette smoking is an important risk factor for PUD.
In the present study, cigarettes smoking showed significant association with PUD. Furthermore, a dose-response relationship was seen between

PUD and cumulative number of cigarettes smoke / day and it is evident in moderate (10 – 20 cigarettes) and heavy (≥ 21 cigarettes) smokers with OR of 2.02 and 2.30, respectively in comparison with nonsmokers. This finding is consistent with Rosenstock et al. (1) results in Danish population, where the relative risk of 3.7 was found in those smoking ≥ 21 cigarettes /day. In contrast with data from the USA (12), tobacco smoking seems to be a more important risk factor for PUD than H. pylori infection in Denmark (15). Recent studies have suggested that tobacco smoking causes PU only if H. pylori infection is present (17–19). Unfortunately, our findings cannot support this notion because H. pylori prevalence not included in ours, but tobacco smoking remained an independent risk factor for PUD despite control for H. pylori infection status. For that reason, researchers believe that ulcer patients should be advised to cease smoking irrespective of H. pylori infection status (20).

The association between coffee drinking and PUD is controversial (21). In the present work, although a small proportion of cases (12.8%) and controls (6.3%) were reported consuming large amount of coffee (as this social habit is not so popular in our community), an OR of 2.20 suggest a positive association between coffee drink and PUD (p= 0.021). The prospective nature of cases collection in the present study (incident cases) should prevent bias resulting from changes in coffee drinking habits due to medical advice. Still, it is possible that ulcer patients may have reduced their coffee intake prior to ulcer diagnosis because of abdominal discomfort.

Similarly, a very small proportion of alcohol drinkers in this study; most probably because this habit is greatly forbidden in our society, and that the dosage was not considered, invalidated the high OR (OR=2.72) of this factor. Whereas in Western societies, the excessive consumption of alcohol (especially wine) has been shown to increase the risk of PUD (1, 22).

On the other hand, tea drink of ≥ 4 cups /day appear to be do nothing with the occurrence of PUD in the present study, which is concordat with the study of Rosenstock et al. (1).

One of the main environmental exposures contributing to PUD is the nutritional factors. The finding of the present study suggest that a high intake of dietary fiber reduces the risk of PUD (OR=0.52, 95% CI: 0.35; 0.77, p= 0.001) i.e. those who consume high quantities of different dietary fibers are around half time prone to have PUD than the non-consumers. To our knowledge, fibers have been examined prospectively in only two previous studies of duodenal ulcer; first a study among American men of Japanese ancestry in Hawaii (23), which used a rather limited dietary assessment method, and a second study among 47 806 American men (24), aged 40 – 75 years were they found that the total dietary fiber intake was inversely associated with the risk of duodenal ulcer (RR = 0.55, 95% CI: 0.31; 0.96 for men in the highest vs. the lowest quintile of dietary fiber). The soluble component of fiber was strongly associated with a decreased risk of duodenal ulcer (RR = 0.40, 95% CI: 0.22; 0.74 for the highest quintile).

In our data, fiber from fruits, vegetables, leguminous, and cereal sources, was associated with a reduced risk of PUD (OR= 0.52). The liquid phase of a meal is emptied more rapidly into the duodenum in patients with duodenal ulcer compared with controls. Dietary fiber might delay this rapid emptying of the liquid phase, which might explain its apparent benefit (24).

Regarding genetic influence, a family history is often present in DU, especially when blood group O is also present, while inheritance appears to be unimportant in gastric ulcers except in regard to Zollinger Ellison syndrome which is a rare gastrin-secreting tumor causing multiple and difficult to heal ulcers (5). In the current study, positive family history was among the significant risk factors (OR= 2.94). This may be attributed to familial dietary habits rather than inherited influence. However, the effect of other factor as blood group may confound this conclusion.

Recent studies have shown a significant reduction in duodenal ulcer risk in American men who exercise regularly (25). Older studies suggest that physical inactivity increases the likelihood of ulcer disease (26, 27). Moderate energy expenditure was shown to reduce the overall likelihood of ulcer disease in the current study (OR= 1.40), however, it was non-significant. Possible mechanisms could include a decrease in gastric acid secretion, lower levels of stress, and differences in dietary factors (27).

Although many physicians and laypersons believe that stress plays a role in the occurrence of PUD, the importance of stress in the pathogenesis of peptic ulcers remains controversial. Stressful life events appeared in this work to be associated with the development of PUD, (OR=2.15, 95% CI: 1.45; 3.19, p= 0.000). This finding agreed with the study of Al-Kayatt et al. (28) during 2008 in Mosul, where stress was found to be associated with the occurrence of gallstones for women in childbearing age (OR=3.00, 95% CI: 1.70; 5.29, p= 0.0001). The probable mechanism of the action of the stress on the body is that it acts as a causal factor in disease or as a contributing one, by reducing the individual's resistance to disease process (28).

Regarding psychological makeup in the present study, personality type A, appeared to be associated with the occurrence of PUD, (OR=1.72, 95% CI: 1.10; 2.80, p= 0.028). In respect to housing condition, high crowding index appear to do nothing in the occurrence of PUD. Again, those results similar to Al-Kayatt et al. (28) and Salih et al. (29) studies.

The present study concludes that the etiology of PUD is multifactorial. Cigarettes smoking, coffee drinking, positive family history of PUD, low fiber diet, stressful life events and personality type A were the main risk factors for incident PUD in Mosul studied sample. Other factors, as level of education, occupation, residence, physical activity, overcrowded houses and excessive tea drinking has no significant role in the occurrence of PUD.

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References

1. Rosenstock S, Jørgensen 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.
2. Pulvertaft CN. Comments on the incidence and natural history of gastric and duodenal ulcers. *Postgrad Med J* 1998;44:597-602.
3. Bonnevie O. Changing demographics of peptic ulcer disease. *Dig Dis Sci* 1985;30(suppl):8S-14S.
4. Bytzer P, Teglbjaerg PS. Helicobacter pylori-negative duodenal ulcers: prevalence, clinical characteristics, and prognosis--results from a randomized trial with 2-year follow-up. *Am J Gastroenterol* 2001; 96: 1409-1416.
5. 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;147:529–36.
6. Bloom BS. Cross-national changes in the effect of peptic ulcer disease. *Ann Intern Med* 1991;114:558-62.
7. Peterson WL. *Helicobacter pylori* and peptic ulcer disease. *N Engl J Med* 1991;324:1043-8.
8. Taylor DN, Blaser MJ. The epidemiology of *Helicobacter pylori* infection. *Epidemiol Rev* 1991;13:42-59.
9. Prichard PJ, Yeomans ND. *Helicobacter pylori* and duodenal ulcer. *J Gastroenterol Hepatol* 1991 ;6:177-8.
10. Hollander D, Tarnawski A. Dietary fatty acids and the decline in peptic ulcer disease—a hypothesis. *Gut* 1986;27:239-42.
11. Rydning A. Dietary fibre and peptic ulcer. *Scand J Gastroenterol Suppl* 1987; 129:232-40.
12. Kurata JH, Nogawa AN. Meta-analysis of risk factors for peptic ulcer. Nonsteroidal antiinfl ammatory drugs, *Helicobacter pylori*, and smoking. *J Clin Gastroenterol* 1997; 24: 2-17.
13. Schlemper RJ, van der Werf SD, Vandenbroucke JP, Biemond I, Lamers CB. Risk factors of peptic ulcer disease: different impact of *Helicobacter pylori* in Dutch and Japanese populations? *J*

Gastroenterol Hepatol 1996; 11: 825-831.

14. Parasher G, Eastwood GL. Smoking and peptic ulcer in the *Helicobacter pylori* era. *Eur J Gastroenterol Hepatol* 2000; 12: 843-853.
15. Andersen IB, Jørgensen T, Bonnevie O, *et al.* Smoking and alcohol intake as risk factors for bleeding and perforated peptic ulcers: a population-based cohort study. *Epidemiology* 2000;11:434–9.
16. Johnsen R, Forde OH, Straume B, Burhol PG. Aetiology of peptic ulcer: a prospective population study in Norway. *J Epidemiol Community Health* 1994; 48: 156-160.
17. Chan FKL, Sung JJY, Lee YT, et al. Does smoking predispose to peptic ulcer relapse after eradication of Helicobacter pylori? *Am J Gastroenterol* 1997;92:442–5.
18. Borody T, George L, Brandl S, *et al.* Smoking does not contribute to duodenal ulcer relapse after Helicobacter pylori eradication. *Am J Gastroenterol* 1992;87:1390–3.
19. Stack WA, Atherton JC, Hawkey GM, *et al.* Interactions between Helicobacter pylori and other risk factors for peptic ulcer bleeding. *Aliment Pharmacol Ther* 2002;16:497–506. 192 Rosenstock, Jørgensen, Bonnevie, et al www.gutjnl.com Downloaded from gut.bmj.com on March 3, 2012 - Published by group.bmj.com
20. Parasher G, Eastwood GL. Smoking and peptic ulcer in the Helicobacter pylori era. *Eur J Gastroenterol Hepatol* 2000;12:843–53.
21. Suadicani P, Hein HO, Gyntelberg F. Genetic and life-style determinants of peptic ulcer. A study of 3387 men aged 54 to 74 years: The Copenhagen Male Study. *Scand J Gastroenterol* 1999;34:12–17.
22. Chou SP. An examination of the alcohol consumption and peptic ulcer association—Results of a national survey. *Alcohol Clin Exp Res* 1994;18:149–53.
23. Kato I, Nomura AMY, Stemmermann GN, *et al.* A prospective study of gastric and duodenal ulcer and its relation to smoking, alcohol, and diet. *Am J Epidemiol* 1992; 135:521-30.
24. Aldoori WH, Giovannucci EL, Stampfer MJ, *et al.* Prospective study of diet and the risk of duodenal ulcer in men. *Am J Epidemiol* 1997;145:42–50.
25. Cheng Y, Macera CA, Blair SN. Physical acitivity and peptic ulcers. Does physical activity reduce the risk of developing peptic ulcers? *West J Med* 2000;173:101–7.
26. Paffenberger RS, Wing AL, Hyde RT. Chronic disease in former college students XIII. Early precursors of peptic ulcer. *Am J Epidemiol* 1974;100:397–15.
27. Chuong JJH, Fisher RL, Chuong RLB, *et al.* Incidence, risk factors, and predictive value of plasma pepsinogen. *Dig Dis Sci* 1986;31:1178–84.
28. Al-Kayatt MK, Al-Youzbaki DB, Al-Zubeer AA. **Sociological Risk Factors in the Development of Gallstones for Women in the Childbearing Age; A Case-control study. *Iraqi J. Comm. Med., Apr.*2008;21(2):102-108.**
29. 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* 2007; 13(23): 3245-3248.

Table (1): Descriptive characteristics of the study sample.

Variables	PUD cases (n=180)		Control (n=240)		OR	95% C.I	P-value
	No.	%	No.	%			
Age mean ± SD (years)	41.8 ± 6.45		39.9 ± 7.74				
< 25	13	7.2	35	14.6			
25-44	68	37.8	71	29.6			
45-64	94	52.2	125	52.1			
65+	5	2.8	9	3.8			
Sex							
Male	112	62.2	152	63.3			
Female	68	37.8	88	36.7			
Marital status							
Married	142	78.9	153	63.8	2.13	1.36; 3.31	0.001
Single	38	21.1	87	36.2			
Residence							
Urban	125	69.4	183	76.3	0.71	0.46; 1.09	0.119
Rural	55	31.6	57	23.7			
Education							
Illiterate [a]	4	2.2	5	2.1	1.00	---	---
Primary & 2nd schools	56	31.1	67	27.9	0.96	0.27; 3.74	0.950
University +	120	66.7	168	70.0	1.12	0.30; 4.26	0.868
Occupation							
Civil servant [a]	75	41.7	82	34.2	1.00	---	---
Laborer	35	19.4	42	17.5	0.91	0.53; 1.58	0.739
House keeper	32	17.8	52	21.7	0.67	0.39; 1.16	0.150
Students	15	8.3	34	14.2	0.48	0.24; 0.96	0.035
Others	23	12.8	30	12.5	0.84	0.45; 1.57	0.581
Positive family history	38	21.1	20	8.3	2.94	1.65; 5.26	0.000

[a] Reference group.

Table (2): Distribution of the study population according to diet, drinks and personal characteristics.

Variables	PUD cases (n=180)		Control (n=240)		OR	95% C.I	P-value
	No.	%	No.	%			
High fatty, salty and calories intake	95	52.8	112	46.7	1.28	0.87; 1.88	0.215
High fibers intake	65	36.1	125	52.1	0.52	0.35; 0.77	0.001
Coffee drink ≥ 3 cups /day	23	12.8	15	6.30	2.20	1.11; 4.35	0.021
Tea drink ≥ 4 cups /day	64	35.6	94	39.17	0.86	0.57; 1.28	0.450
Alcohol drink	6	3.3	3	1.3	2.72	0.67;11.04	0.145
Cumulative number of cigarettes daily							
(0) *	70	38.9	135	56.3	1.00	---	---
1-9 cigarettes	20	11.1	22	9.2	1.75	0.89; 3.43	0.098
10-20 cigarettes	65	36.1	62	25.8	2.02	1.29; 3.19	0.002
≥ 21 cigarettes	25	13.9	21	8.8	2.30	1.20; 4.39	0.011
Leisure time energy expenditure							
Active *	45	25.0	76	31.7	1.00	---	---
Ambulatory	59	32.8	71	29.6	1.40	0.85; 2.33	0.188
Sedentary	76	42.2	93	38.8	1.38	0.86; 2.22	0.185
Stressful life	103	57.2	92	38.3	2.15	1.45; 3.19	0.000
Personality type A	44	24.4	38	15.8	1.72	1.10; 2.80	0.028
Crowding index > 8 persons / household	34	18.9	45	18.8	1.01	0.62; 1.65	0.971

* Reference group

الخلفية الاجتماعية لمرضى قرحة المهضم في مدينة الموصل: دراسة الحالة والشاهد

د. همام غانم إبراهيم الحاج زبير - دكتوراه طب مجتمع- مدرس - كلية الطب - جامعة الموصل
د. رافع محمد حازم القزاز - بورد باطنية - طبيب اختصاص- دائرة صحة نينوى - م.ابن سينا التعليمي
د. رامي محمد عادل- بورد باطنية - أستاذ مساعد - رئيس فرع الطب - كلية الطب - جامعة الموصل
د. ضحى عصام أحمد المعاضيدي- دبلوم طب مجتمع - طبيبة اختصاص- دائرة صحة نينوى- م. ص. الحدباء التدريبي

الخلاصة

مقدمة: إن حدوث قرحة المعدة والإثني عشري تمتاز بتغيرات جغرافية وزمنية واضحة مما يدل على أن للعوامل البيئية دورا مهما في مسببات حدوث هذين المرضين.

هدف الدراسة: تهدف الدراسة الحالية إلى تحديد عوامل الخطورة الاجتماعية المتعلقة بحدوث قرحة المهضم في عينة البحث في مدينة الموصل.

تصميم الدراسة: دراسة الحالة والشاهد.

المشاركون: تم جمع 180 حالة حديثة من قرحة المهضم شخصت بواسطة منظار الجهاز الهضمي العلوي في وحدة التنظير في مستشفى ابن سينا التعليمي. كذلك تم اخذ 240 شخص كعينة ضابطة تتألف من طلاب كلية الطب وعوائلهم وأقاربهم وكذلك مرافقين للمرضى في مستشفيات أخرى. كما تم اختيارهم بحيث أن أعمارهم وجنسهم مشابه لعينة حالات قرحة المهضم. استغرق جمع العينات سنة واحدة اعتبارا من 15 أيار 2009 إلى 15 أيار 2010.

النتائج: العُمر المتوسط لعينةِ الدراسة كانَ 41.8 ± 6.45 سنَة للحالاتِ و39.9 ± 7.74 سنَة لعينة الشاهد. بخصوص العادات الاجتماعية: تدخين السجائر وُجِدَ انه يُرتَبط بحدوثِ مرضِ قرحة المهضم إلى حدٍ كبير ومتعلّق بكمية السكائر في اليوم الواحد (OR=2.30, 95% CI: 1.20; 4.39, p= 0.011) للمدخنين بإسرافٍ، أيضاً لشربِ القهوةِ أكثر من 3 كؤوسٍ / يوم (OR= 2.20, 95% CI: 1.11; 4.35, p= 0.021). بينما الغذاء الذي يحتوي على نسبة عالية من الألياف يكون وقائيا ضدَ حدوثِ مرضِ قرحة المهضم (OR=0.52, 95% CI: 0.35; 0.77, p= 0.001). فيما يتعلق بالخصائص الشخصية: التأريخ العائلي الإيجابي و الشخصية نوع ا تبيّن بأن لهم تأثيرٌ إيجابيٌ في تطويرِ مرضِ قرحة المهضم (OR=2.94, 95% CI: 1.65; 5.26, p= 0.000) و(OR=1.72, 95% CI: 1.10; 2.80, p= 0.028) على التوالي، بالإضافة إلى أحداثِ الحياةِ المجهدةِ (OR=2.15, 95% CI: 1.45; 3.19, p= 0.000). بينما العوامل الأخرى مثل مكان العيش (مدينة أو ريف)، المهنة، مستوى التعليم، كثرة الأشخاص في المنزل الواحد، وشرب الشاي بكثرة يَبْدوا أن ليس لها علاقة في حدوثِ مرضِ قرحة المهضم.

الاستنتاجات: عوامل الخطورة لمرضِ قرحة المهضم متعدّدة. تدخين السجائر، شرب القهوة، تأريخ عائلي إيجابي مِنْ مرضِ قرحة المهضم، نسبة منخفضة من الألياف الغذائية، أحداث الحياةِ المجهدة، و الشخصية نوع ا تبيّن بأن لهم تأثير إيجابي في حدوثِ مرضِ قرحة المهضم للعينة في مدينة الموصل.

الكلمات الدليلية: قرحة المهضم، عوامل خطورة، دراسة الحالة والشاهد .