

CLINICAL FEATURES AND ENDOSCOPIC FINDINGS IN PEPTIC ULCER

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SUMMARY

The occurrence of peptic ulcer is characterized by marked geographical variations. A prospective study regarding the clinical features and endoscopic findings in peptic ulcer was conducted in the department of Gastroenterology at PGMI, LRH, Peshawar from 1.6.95 to 31.5.96. A total number of 70 patients, 53 males and 17 females with age range of 18-85 years having endoscopically proven peptic ulcer were included in the study. The male to female ratio in general was 3:1, for duodenal ulcer (DU) 3.4:1 and for gastric ulcer (GU) 1.3:1. The duodenal to gastric ulcer ratio was 8.8:1. Peptic ulcer (PU) was predominantly seen between 4th and 6th decade of life. Patients presented with more than one symptoms; pain epigastrium being the most common (85%). A small number (7%) of ulcers were 'silent'. Food generally initiated/aggravated pain in GU but relief occurred in DU. In the majority of patients, the symptoms lasted for upto 10 years. The use of NSAIDs and tobacco were two major risk factors seen in this study. Sixty five percent of patients were from lower socioeconomic class. A positive family history for ulcer was seen in 28% patients, the majority of whom had DU. Duodenal ulcer was significantly associated with blood group 'O'. Gastric ulcer can not be differentiated clinically from DU and Oesophagogastro-duodenoscopy is a safe and sensitive diagnostic method.

INTRODUCTION

Peptic ulcer (PU) is a chronic disease characterized by periodic relapses and remissions.^{1,2} Regarding its aetiology and pathogenesis, peptic ulcer is considered a multifactorial disorder^{3,4} the most important and basic factor is the imbalance between the aggressive factors and the defence mechanisms of the gastroduodenal mucosa that resist or protect it from ulceration.^{3,5-8} The isolation of *Helicobacter pylori* (*H. pylori*),^{9,10} has opened new avenues towards the pathogenesis of peptic ulcer disease (PUD).^{11,15} There are significant geographical^{1,6,8,16} variations in the occurrence of PUD; the epidemiology and natural history of DU differ from that of GU.^{1,8,16}

The purpose of this study is to define the pattern of peptic ulcer in the North West Frontier Province (NWFP) of Pakistan, particularly the relative frequency of DU and GU; the association of PUD with any particular age group; sex, socioeconomic status and life style of the patient and clinical presentation of peptic ulcer.

MATERIAL AND METHODS

This study was conducted in the Gastroenterology department of the Postgraduate Medical Institute (PGMI), Lady Reading Hospital (LRH), Peshawar, which is the largest hospital of the province.

A total number of 70 patients, 53 males and 17 females were included in the study.

TABLE - I
PEPTIC ULCER: AGEWISE DISTRIBUTION

TOTAL NO. OF PATIENTS: 70
AGE RANGE: 18-85 YEARS

AGE IN YEARS	DUODENAL ULCER			GASTRIC ULCER			DU+GU			TOTAL G. TOTAL
	MALE	FE-MALE	TOTAL	MALE	FE-MALE	TOTAL	MALE	FE-MALE	TOTAL	
13-20	2	1	3	0	0	0	0	0	0	3(4.29%)
21-30	7	1	8	0	0	0	0	0	0	8(11.43%)
31-40	7	4	11	0	0	0	0	0	0	11(15.75%)
41-50	8	5	13	1	0	1	0	0	0	14(20%)
51-60	14	3	17	2	2	4	0	0	0	21(30%)
61-70	9	0	9	1	0	1	1	0	1	11(15.71%)
71 AND ABOVE	1	0	1	0	1	1	0	0	0	2(2.85%)
GRAND TOTAL	48	14	62	4	3	7	1	0	1	70(100%)

The ages of the patients ranged from 18-85 years. Most of the patients were referred from general Medical and Gastroenterology Out patient Departments and some admitted in the wards with symptoms and signs suggestive of PUD were endoscoped. Every patient, selected for Oesophagogastro-duodenoscopy (OGD) was informed about the procedure, all consented to it. After an overnight fast, they were given topical anaesthesia with lignocaine jelly. Occasionally, very anxious patients were sedated with diazepam 5-10 mg slow intravenous injection. All the OGDs were performed using the Olympus type 2T20 or GIF XQ endoscopes with teaching aid extension.

Patients in shock due to massive gastrointestinal haemorrhage or those with ulcer perforation and those with unstable cardiopulmonary disease were excluded. Only those patients with endoscopically proven peptic ulcer were selected for the study. The study extended over a period of one year from June 1, 1995 to May 31, 1996.

Chronic peptic ulcer was, endoscopically, defined as a circumscribed break in the mucosa of stomach or duodenum of 5 mm or more in diameter with an exudate and apparent depth. The size of the ulcer was estimated with open biopsy forceps. Ulcers between 5 mm and 1 cm were grouped as 'Small' ulcers, those more than 1 cm to upto 2 cm as 'large' while ulcers greater than 2 cm in diameter were classed as 'Giant' ulcers. All gastric ulcers were biopsied to exclude malignancy. All OGDs were performed safely and no complication occurred during or after the procedure.

RESULTS

Out of 70 peptic ulcer patients, 62(88.57%) had DU, 7(10%) had GU and one (1.43%) had co-existing DU and GU. The ratio of DU to GU was 8.8:1.

Fifty three out of the 70 patients (75.71%) with peptic ulcer were males and 17(24.29%) patients were females, with a M:F ratio of 3:1. Among 62 DU patients, 48(77.4%) were males and 14(22.6%)

TABLE - II
PEPTIC ULCER: RISK FACTORS
NUMBER OF PATIENTS

RISK FACTOR	DUODENAL ULCER	GASTRIC ULCER	TOTAL	PERCENTAGE
NSAIDS	23	5	28	40%
SMOKING	21	3	24	34.29%
NASWAR	11	1	12	17.14%
NASWAR + SMOKING	2	1	3	4.28%
ALCOHOL	2	0	2	2.86
STEROIDS	1	0	1	1.43%

patients were females with a ratio of 3.4:1. Among 7 GU patients, 4(57.14%) were males and 3(42.86%) were females. The M:F ratio for GU was 1.3:1. The one patient with co-existing ulcers was male. The ages of ulcer patients ranged from 18-85 years. Occurrence of peptic ulcer increased with advancing age. Majority (65%) of the patients were in their 4th to 6th decade of life Table-I. Most of the ulcer patients presented with more than one symptoms; the most common complaint being pain epigastrium (85%) followed by vomiting (45%) and dyspepsia (40%) Fig-I. Five (7%) patients had silent ulcers, presenting for the first time with complications. Twenty four (34.28%) patients had anaemia of varying severity. In GU patients, food generally initiated or aggravated pain (42.86%) while in DU, it generally relieved (36.5%) or did not after (42.86%) the ulcer pain. Majority of the patients had symptoms for 5-10 years, though one patient had symptoms for more than 20 years Fig-II. The intake of non steroidal anti-inflammatory drugs (NSAIDs) and tobacco were the major risk factors in PUD Table-II. A positive family history for peptic ulcer was present in 28% patients, most having DU. A correlation was found between blood group "O" and DU in 44.44% patients, group "B" was found in

26.98%, "A" in 20.64% and blood group "AB" in 7.94% of DU patients. The distribution of blood group in DU patients was "B" (57.14%) "A" (28.57%) and "O" 14.29%. None of the GU patients had blood group "AB". Majority (65) of the patients belonged to the lower socioeconomic class while 35% patients were well off.

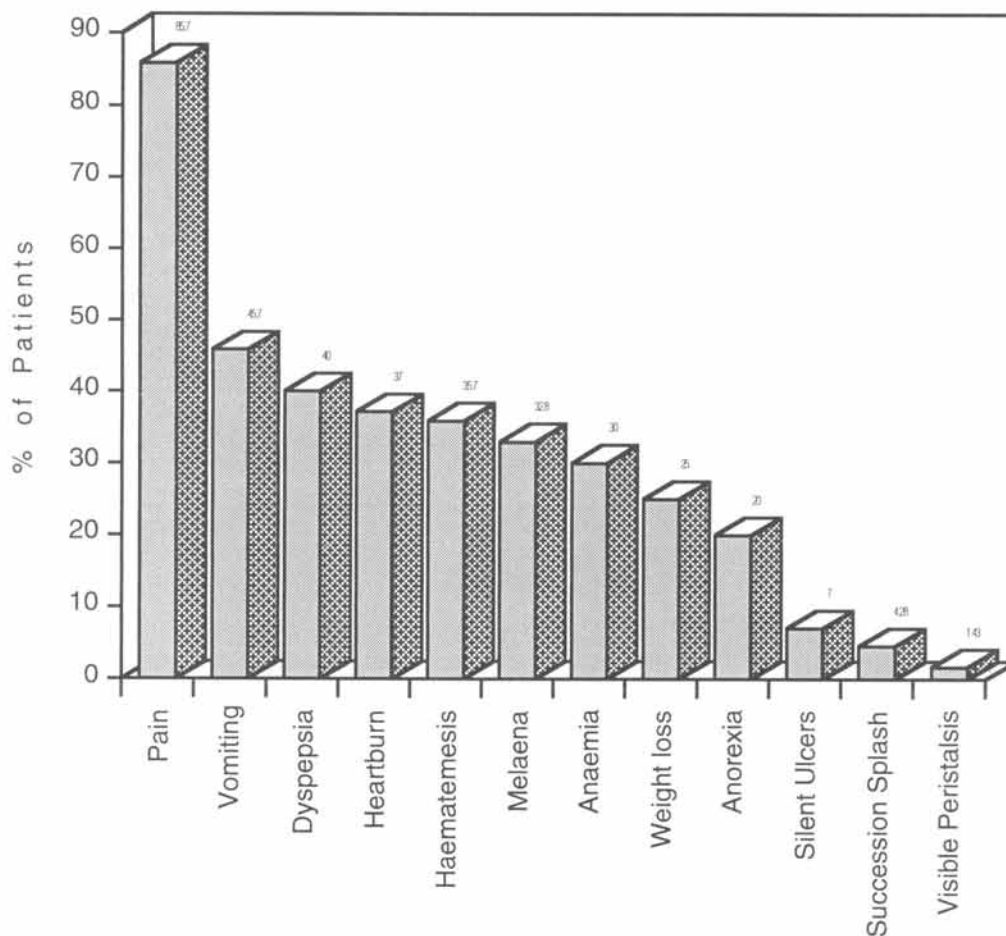
Peptic ulcer complications occurred in 50% patients. The most common complication was haemorrhage (37.14%) followed by gastric outlet obstruction (pyloric stenosis) (10%). One patient (1.43%) has past history of operation for perforated DU. One patient (1.43%) presenting with anaemia was found at OGD to be suffering from DU. At endoscopy, 38(61.29%) out of 62 DU patients had ulcers on anterior duodenal wall and 20(32.26%) patients on posterior duodenal wall of the first part of the duodenum. Four (6.45%) patients had multiple DUs. Of the 7 GU patients, ulcers in 6(85.72%) were found on the lesser curvature near the incisura angularis whereas in one patient (14.28%) the ulcer was located at the greater curvature of the stomach.

DISCUSSION

There are marked geographical variations in the incidence and prevalence of

Figure 1
Peptic Ulcer Presentation Symptoms and Signs

Total No. of Patients =70



peptic ulcer and the DU to GU ratio varies widely in different parts of the world.^{6,8,16} The DU:GU ratio in this study was 8.8:1 which is similar to other studies done in Peshawar (8.7:1),²⁷ DU : GU ratios lower than in this series have also been reported in various studies done in Karachi (5:1),¹⁷ Bangladesh (4.4:1)¹⁸ and in Saudi Arabia (5.4:1).¹⁹ The ratios reported from India varies widely, from 17:1,²⁰ 15:1.²² The ratio reported from United Kingdom²³ (UK) and

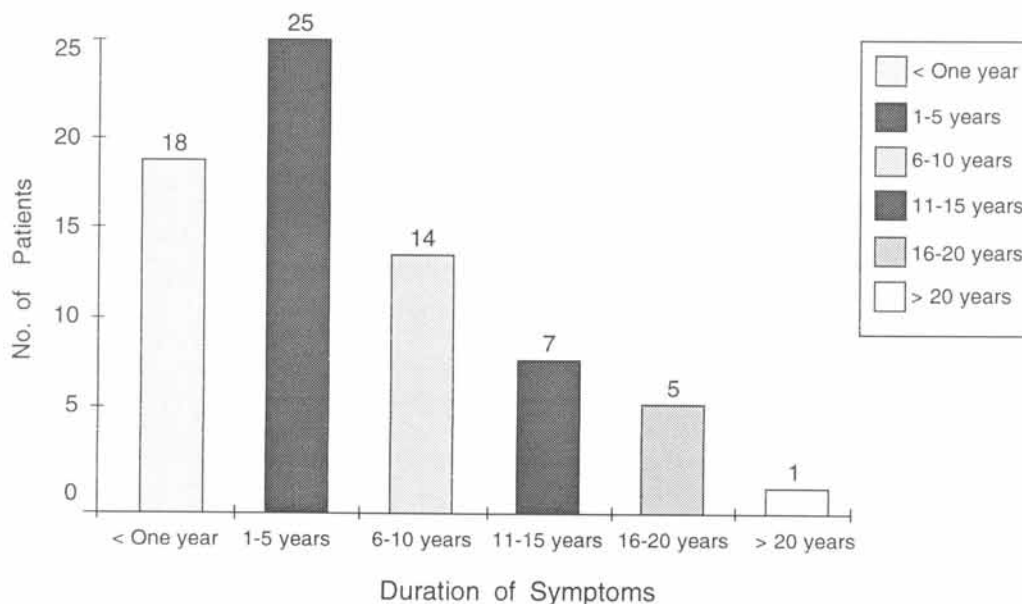
United States of America^{16,24} (USA) is 4:1 which is less than the present study. In Peshawar, many studies have shown predominance of DU over GU, with varying ratios of 15:1,²⁵ 12.8:1,²⁶ 8.7:1,²⁷ 3.3:1²⁸ and 1.8:1.²⁹ The variations in local series are probably due to the differences in the study designs, lack of specification between acute and chronic ulcers and the number of patients. On the contrary, however, GU is 5-10 times more common in Japan.¹⁶ The

male to female ratio for peptic ulcer, in general was 3:1 and for DU 3.4:1 in this series, which is similar to other studies done in Peshawar.²⁸ The sex ratios in some series^{25,27} are slightly less than in present series. The sex ratio reported in DU patients from Karachi¹⁷ is higher (5.7:1) than this report. Studies from India^{20,22} and western world^{8,23} have also shown a greater frequency of peptic ulcer, especially DU, in male population. The difference for GU in the two sexes is slight; many have reported it to be equally common in men and women^{5,29} or slightly more common in females.^{7,25} The exact mechanism for the higher incidence of peptic ulcer in men is not known. Animal studies, however, have shown that male sex hormones increase and female sex hormones decrease the parietal cell mass and basal gastric acid secretion.³⁰ Interestingly, peptic ulcer in women usually disappears during pregnancy and lactation.³¹

In the present study, an increase in the frequency of peptic ulcer was seen with advancing age. Majority of the peptic ulcers occurred in the 4th to 6th decade of life; most of the GU patients were older than DU patients. These findings corroborate other studies from Peshawar,²⁵ Karachi,¹⁷ Northern India²⁰ and literature from UK^{23,32} and USA.^{7,8,16,33} However in Southern India, peptic ulcer occurred in a relatively younger male population.²¹ Reasons for the higher incidence of PUD in old age are, probably the use of ulcerogenic medications particularly NSAIDs for chronic painful musculoskeletal conditions associated with old age and use of tobacco. Other mechanisms include progressive breakdown in the mucosal defence mechanism with decrease in prostaglandin (PG) concentration and bicarbonate secretion,^{34,35} a decrease in the drug metabolism in the elderly, rendering them more vulnerable to ulcerogenic agents³³ and

Figure 2
Peptic Ulcer: Duration of Symptoms

Total No. of Patients =70



higher prevalence of *H. pylori* infection in old age.³⁶

Most of the patients in this study presented with more than one symptoms. The most common symptoms was pain epigastrium (85%). This is in conformity with other series of this area,^{25,27,29} Karachi¹⁷ and from USA⁸. Correlation of food with ulcer pain was noted in patients in this series. Food in general, relieved pain in DU patients and initiated or aggravated it in GU patients. These findings corroborate the observations reported in most of the literature.^{2,6,8,37} Peptic ulcer is a chronic disease characterized by relapses and remissions, lasting for years. Although some patients in this series had symptoms for more than 15 years, however, in majority the duration of symptoms was upto 10 years.

In the present study, the intake of NSAIDs appeared as a major risk factor for PUD (in 40 patients). It was associated more with GU (71%) than with DU (37%). This is in conformity with the results of other series.^{5,6,8,33,38-41} The second major risk factor for PUD was tobacco whether in the form of cigarette smoking or naswar intake or both. Most of the tobacco addicts had DUs. The data augment the findings in a series of DU patients¹⁷ and also support the views that cigarette smoking is associated with PUD,^{3,42,43} increases the incidence of PUD,⁴⁴ delays ulcer healing^{5,45} and increases the rate of ulcer relapse.⁴⁶ Smoking has also been found to be associated with high prevalence of *H. pylori* infection.^{36,43} Only a small number (2.8%) of ulcer patients, included in this series occasionally took alcohol, perhaps for a variety of reasons including religious, legal and social.

Peptic ulcer was previously believed to be a disease of the highly stressed, professional, upper class, middle aged men but in the present study 65% of the ulcer patients were poor, belonging to the lower socioeconomic class and 35% patients were well off. This corroborates the results from

most of the western countries.⁸ In contrast, however, no association was found between PUD and socioeconomic status of the patients in the Northern India.²⁰

PUD is considered to have a genetic predisposition and various genetic traits have been associated with it, particularly with the DU. In the present series 44.44% of DU patients had blood group 'O', although the common blood group in the Pakistani population is 'B'. In GU patients the common blood group was 'B' (57%). This study shows a correlation between blood group 'O' and DU which is similar to the findings in other studies from Peshawar.²⁷ The data is also comparable to those of other workers from different parts of the world.^{6,8,24,32,47} Moreover, 28% of patients in this series had a positive family history of PDU which is similar to the data available in the literature.^{8,32} However in contrast to this, only 8% of DU patients had a positive family history of PUD in one series.¹⁷ The differences in these figures are probably due to the fact that family history of PUD was not proven by documentary evidence.

In the present study, 37% of ulcer patients had history of ulcer bleeding at some stage of the disease. However 5% presented with acute episodes of haemorrhage. The second common complication was gastric outlet obstruction (pyloric stenosis) in 10% patients. The data is similar to that in other series from Karachi,¹⁷ India^{20,22} and in the literature from USA.⁴⁸ In contrast studies from Germany⁴⁹ showed a higher incidence of bleeding peptic ulcer of 51.4%. The figures in my series, for bleeding peptic ulcer are higher than those of a classic series (14%).²³ The reasons are probably the injudicious use of ulcerogenic agents such as NSAIDs, smoking and naswar intake by the patients included in this series. The figures for pyloric stenosis in this study are comparable to those reported from India (6.2%)²² and (8.7%).²⁰ However these are in contrast to the figures reported from Karachi

(1%)¹⁷ and in the literature from USA (2%).⁵⁰ The frequency of peptic ulcer perforation was less (1.43%) in the present study than reported by others^{23,50} because patients with perforated ulcers were not included in this study and the only patient in this study had a past history of perforated DU. In the present series 61% of DUs were located on the anterior duodenal wall, 33% on the posterior duodenal wall and 6% patients had multiple DUs. These findings are in accordance with those reported in the literature.^{6,37} Most (86%) of the GUs were found on the lesser curvature near the incisura angularis and 14% were on the greater curvature of the stomach. These findings are also similar to those reported in the literature.^{6,7,16,20} In the present study only one patient had combined DU and GU which is less than the frequency reported by others (10%),¹⁶ (7%)²² and 28% in South India.²¹ The possible explanation for this is probably, the small number of patients included in this study.

It is concluded from this study that in NWFP, peptic ulcer is a relatively common disease, predominantly, occurring in men in the 4th to 6th decade of life. DU is approximately 9 times more common than GU. The intake of NSAIDs and tobacco are the major risk factors for peptic ulceration. GU can not be differentiated clinically from DU and endoscopy is a safe and sensitive diagnostic method for PUD.

REFERENCES

- Misiewicz JJ, Pounder RE. Peptic ulceration. In: Weatherall DJ, Ledingham JGG, Warrell DA (eds). Oxford textbook of Medicine 3rd ed. New York: Oxford 1996; 1877.
- Katz J. The course of peptic ulcer disease. Med Clin North Am 1991; 75:831.
- Mertz HR, Walsh JH. Peptic ulcer Pathophysiology. Med Clin North Am 1991; 75:799.
- Szabo S, Vattay P. Experimental gastric and duodenal ulcers: advances in pathogenesis. Gastroenterol Clin North Am 1990;19:67.
- Pounder R. Peptic ulceration. Medicine international 1994;7: 225.
- Shearman DJC. Diseases of the alimentary tract and pancreas In: Edwards CRW, Bouchier IAD, Haslett C, Chilvers E (eds). Davidson's Principles and Practice of Medicine Edinburgh: 1995; 405.
- McGulgan JE. Peptic ulcer and Gastritis. In: Isselbacher KJ, Braunwald E, Wilson JD Martin JB, Fauci AS, Kasper DL (eds). Harrison's Principles of Internal Medicine. New York McGraw-Hill, 1994; 1363.
- Soll AH. Duodenal ulcer and drug therapy. In: Sleisenger MH, Fordtran JS (eds) Gastrointestinal disease Philadelphia: WB Saunders 1989; 814.
- Marshall BJ. Unidentified curved bacilli on gastric epithelium in active chronic gastritis. Lancet 1983; 1:1273.
- Warren JR. Unidentified curved bacilli on gastric epithelium in active chronic gastritis. Lancet 1983; 1:1273.
- Marshall BJ, McGeachie DB, Rogers PA, Glacy RJ. Pyloric campylobacter infection and gastroduodenal disease. Med J Aust 1985; 142: 439.
- Graham DY, Go MF. Helicobacter pylori: Current status. Gastroenterology 1993; 105: 279.
- Prieto G, Polanco I, Larrauri J, Rota L, Lama R, Carrasco S. Helicobacter pylori infection in children: clinical endoscopic and histologic correlations (abstract). J Pediatr Gastroenterol Nutr 1992; 14: 420.
- Clearfied HR. Helicobacter pylori: aggressor or innocent by-stander? Med Clin North Am 1991; 75: 815.
- Tytgat GN, Rauws EAJ. Campylobacter pylori and its role in peptic ulcer disease. Gastroenterol Clin North Am 1990; 19:183.
- Richardson CT. Gastric ulcer. In: Sleisenger MH, Fordtran JS (eds). Gastrointestinal

- Disease. Philadelphia: WB Saunders 1989; 879.
17. Ahmed WU, Qureshi H, Alam E, Zuberi SJ. Pattern of duodenal ulcer in Karachi. *J.P.M.A.* 1990; 40: 212.
 18. Haque MM, Islam MM. Fibrogastro-duodenoscopy: a reliable diagnostic procedure in upper gastrointestinal disorders. *Specialist* 1994; 10: 203.
 19. Al-Amri SM. Frequency of peptic ulcers in patients with portal hypertension. *Ann Saudi Med* 1995; 15(5): 451.
 20. Khuroo MS, Mahajan R, Zargar SA, Javid G, Munshi S. Prevalence of peptic ulcer in India; an endoscopic and epidemiological study in urban Kashmir. *Gut* 1989; 30: 930.
 21. Rao SS, Murthy KV. Postbulbar and co-existing ulceration: unique features of peptic ulcer in Hyderabad. *Gut* 1993;34:1327.
 22. Goenka MK, Kochhar R, Mehta SK. Changing pattern of peptic ulcer in India. An endoscopic study of 1188 ulcer patients. *J Clin Gastroenterol* 1991; 13:575.
 23. Fry J. peptic ulcer: a profile. *Br Med J* 1964; 2:809.
 24. Kumar V, Cotran RS, Robbins SL. *Basic Pathology. The gastrointestinal tract.* 5th ed. Philadelphia: WB Saunders 1992; 473.
 25. Orakzai RU. Correlation between symptomatology and upper GI endoscopy findings. *J Med Sci* 1994; 4: 21.
 26. Rehman SU, Orakzai RU, Zarif M. Endoscopic evaluation of dyspepsia. *J Med Sci* 1992; 3: 3.
 27. Shah MS. Upper gastrointestinal endoscopy. *J Postgrad Med Institute* 1986; 1:71.
 28. Khan C. Endoscopic evaluation of disease of the upper gastrointestinal tract. *J Med Sci* 1990; 1:14.
 29. Khan PM, Shakeel, Hayat Z. Endoscopic interpretation of symptomatology of patients with upper gastrointestinal problems: a review of 1390 patients. *J Postgrad Med Institute* 1993; 6:59.
 30. Adeniyi KO. Gastric acid secretion and parietal cell mass: effect of sex hormones. *Gastroenterology* 1991;101:66.
 31. Robert A, Kauffman GL Jr. Stress ulcers, erosions and gastric mucosal injury. In: Sleisenger MH, Fordtran JS (eds). *Gastrointestinal disease.* Philadelphia: WB Saunders 1989; 772.
 32. Rees WD, Dowd AB. Pathophysiology of duodenal ulcer. In: Jewel DP, Lowes JR (eds). *Topics in gastroenterology* 16. Oxford: Black Well Scientific Publications 1989;49.
 33. Graham DY. The relationship between non steroidal anti-inflammatory drug use and peptic ulcer disease. *gastroenterol Clin North Am* 1990; 19:171.
 34. Cryer B, Redfern JS, Goldschmiedt M, Lee E, Feldman M. Effect of aging on gastric and duodenal prostaglandin concentrations in humans. *Gastroenterology* 1992; 102:1118.
 35. Kim SW, Parekh D, Townsend CM Jr, Thompson JC. Effects of aging on duodenal bicarbonate secretion. *Ann Surg* 1990; 212:332.
 36. Kazmi SU, Amjad M, Shahid M, Manzoor H, Quraishy S. A five year study of prevalence of *Helicobacter pylori* infection in Karachi Pakistan. *JCPSP* 1996; 6(1): 39.
 37. Moynihan BGA. *Duodenal ulcer.* 2nd ed. Philadelphia: WB Saunders 1912.
 38. Lanas AL, Remacha B, Esteva F, Sainz R. Risk factors associated with refractory peptic ulcers. *Gastroenterology* 1995; 109: 1124.
 39. Langman MJS, Weil J, Wainwright P, et al; Risk of bleeding peptic ulcer associated with individual non steroidal anti-inflammatory drugs. *Lancet* 1994; 343: 1075.
 40. Henry D, Robertson J. Non Steroidal anti-inflammatory drugs and peptic ulcer hospitalization rates in New South Wales. *Gastroenterology* 1993; 104: 1083.
 41. Laporte JR, Carne X, Vidal X, Morena M, Juan J. Upper gastrointestinal bleeding in

- relation to previous use of analgesic and NSAIDs. *Lancet* 1991; 337:85.
42. Moxham J. Smoking. *Medicine international* 1995; 9: 83.
 43. Endoh K, Leung FW. Effects of smoking and nicotine on the gastric mucosa: A review of clinical and experimental evidence. *Gastroenterology* 1994; 107: 864.
 44. Friedman GD, Siegelau AB, Seltzer CC. Cigarette, alcohol, coffee and peptic ulcer. *N Engl J Med* 1974; 290:496.
 45. Doll R, Jones FA, Pygott F. Effect of smoking on the production and maintenance of gastric and duodenal ulcers. *Lancet* 1958; 1: 657.
 46. Korman MG, Hansky MG, Hansky J, Eaves ER, Schmidt GT. Influence of cigarette smoking on healing and relapse in duodenal ulcer disease. *Gastroenterology* 1983; 85: 871.
 47. Graham DY, Lidsky MD, Cox AM, et al. Long term nonsteroidal anti-inflammatory drug use and *Helicobacter pylori* infection. *Gastro-enterology* 1991; 100: 1651.
 48. Rubin W. Medical treatment of peptic ulcer disease. *Med Clin North Am* 1991; 75: 981.
 49. Ohmann C, Thoni K, Hengels KJ, Imhof M. Incidence and pattern of peptic ulcer bleeding in a defined geographical area. DUSUK Study Group. *Scand J Gastroenterol* 1992; 27: 571.
 50. Graham DY. Complications of peptic ulcer disease and indications for surgery. In: Sleisenger MH, Fordtran JS (eds). *Gastrointestinal disease*. Philadelphia: WB Saunders 1989; 925.