LEADING ARTICLE

WHAT'S NEW IN PEPTIC ULCER

I Yap, J Y Kang

SING MED J. 1988; 29: 538 – 539

We still do not know what causes peptic ulcer. However, a lot of work is being done on ulcer pathogenesis, while the effect of smoking, anti-inflammatory drugs, diet and psychosomatic factors on ulcer disease has become clearer in recent years. Major advances have also been made in diagnosis and treatment.

Peptic ulcer is thought to arise from an imbalance between attacking forces, acid and pepsin, on the one hand, and mucosal resistance, involving bicarbonate and mucus secretion, on the other. However, abnormalities of acid-pepsin secretion and of mucosal resistance as currently understood do not explain most cases of peptic ulcer. Gastritis ocurrs in most ulcer patients[1] and has recently been found to be associated with Campylobacter pylori infection[2]. It has been suggested that Campylobacter pylori causes both gastritis and peptic ulcer[3]. This is a very exciting postulate because it could radically alter our approach to treatment of ulcer disease.

Peptic ulcer is commoner among smokers compared to non-smokers[4]. Smokers are less likely to heal their ulcers[5], and after healing, there is a higher risk of recurrence[6]. We too have observed an adverse effect of smoking on duodenal ulcer disease[7]. Contrary to popular belief there is no association between alcohol use and peptic ulcer[8].

Aspirin and other non-steriodal anti-inflammatory drugs are associated with peptic ulcer disease[9], especially complicated ulcer amongst elderly patients[10]. These drugs are thought to damage the gastroduodenal mucosa by inhibiting prostaglandin synthesis[11].

Some authors believe that a high fibre diet reduces the recurrence rate of duodenal ulcer[12] but this has not been our own experience[13]. Chillis and spices are thought by many to be harmful to the stomach. Although their ingestion may give rise to symptoms, it does not produce macroscopic mucosal lesions[14] or delay duodenal ulcer healing[15]. The popular belief that milk is beneficial for the ulcer patient is probably a myth. Although milk may relieve ulcer pain it is a potent stimulator of acid secretion and regular intake may in fact retard ulcer healing[16].

Psychosomatic factors probably do not play an important role in most patients with peptic ulcer. Recent studies on personality patterns or life events in ulcer patients have failed to demonstrate any major abnormalities[17-19].

The introduction of fibreoptic gastroduodenoscopy, and to a lesser extent of the double-contrast barium meal, has not only radically improved our ability to diagnose peptic ulcer disease. It has also stimulated the development of a large number of drugs for treating ulcer. They include histamine-2 receptor antagonists, H^+/K^+ ATPase inhibitors, muscarinic inhibitors, prostaglandins, surface agents, and anti-depressants[20, 21]. Most of these drugs heal ulcers by reducing gastric acid secretion, while sucralfate and colloidal bismuth are thought to act locally at the ulcer base to protect the underlying mucosa. While most of these drugs are equally potent, the histamine-2 antagonists are by far the most widely used. Cimetidine, ranitidine, famotidine and nizatidine are all extremely potent and remarkably safe. Omeprazole, a H^+/K^+ ATPase inhibitors yet to be marketed, is even more potent but there is concern about possible long-term effects of achlorhydria.

With one of the above drugs, the ulcer can almost always be healed. Recurrence, however, remains a problem controllable only by continued used of a histamine-2 antagonist. It has been reported that following ulcer healing with colloidal bismuth, recurrence may be less than if the ulcer was healed with a histamine-2 blocker[22]. This may be due to the bactericidal effect of bismuth on Campylobacter pylori, an area under intense investigation at the present time.

Surgery is nowadays indicated mainly for complicated ulcers and when malignancy cannot be excluded. The trend is towards less radical surgery. The latest operation for duodenal ulcer, parietal cell vagotomy, has a low mortality rate, few post-surgical sequelae but a fairly high ulcer recurrence rate[23].

Racial differences in peptic ulcer frequency have been described both in Singapore[24], and in Malaysia[25, 26]. Chinese appear to be the most prone to ulcer disease; Malays the least. These differences have declined over the last three decades suggesting that environmental rather than genetic factors may be involved[27]. We therefore have a unique opportunity to investigate the factors responsible. If identified they may represent a major advance in our understanding of peptic ulcer disease.

Division Of Gastroenterology,
Department Of Medicine,
National University Of Singapore,
National University Hospital, Singapore

I Yap, MBBS, M Med (Int. Med) AM
Senior Lecturer
J Y Kang, MRCP, FRACP, FRCP (Ed) AM
Assoc. Professor

Correspondence to: Dr Yap

REFERENCES

1. Schrager J, Spink R, Mitra S: The antrum in patients with duodenal and gastric ulcers. Gut 1967: 8: 497-508.
2. Marshall BJ, Warren JB, Unidentified curved bacilli in the stomach of patients with gastritis and peptic ulceration. Lancet 1984; 1: 1311-5.
3. Axon ATR: Campylobacter pyloridis: What role in gastritis and peptic ulcer? Brit Med J 1986; 293: 772-3.
4. Piper DW, Nasiry Mcintosh J, Shy CM, Pierce J, Byth K: Smoking, alcohol, analgeisc and chronic duodenal ulcer. Scand J Gastroenterol 1984; 19: 1015-21.
5. Korman MG, Hansky J, Merrett AC, Schmidt GT. Ranitidine in duodenal ulcer: incidence of healing and effect of smoking. Dig Dig Sci 1982; 27: 712-5.
6. Sontag S, Graham DY, Belsito A et al. Cimetidine, cigarette smoking, and recurrence of duodenal ulcer. N Eng J Med 1984; 311: 689-93.
7. Yap I, LaBrooy SJ, Guan R, Kang JY. Cigarette smoking and duodenal ulcer disease in Singapore. Sing Med J 1987; 28: 308-10.
8. Cooke AR. Drug damage to the gastroduodenum in 'Gastrointestinal Disease' ed MH Sleisenger, HS Fordtran 2nd ed, WB Saunders, Philadelphia P 807-26.
9. Sommerville KW, Hawkey CJ, Non-steroidal anti-inflammatory agents and the gastrointestinal tract. Postgrad Med J 1986; 62: 23-8.
10. Sommerville KW, Faulkner G, Langman M. Non-steroidal anti-flammatory drugs and bleeding peptic ulcer. Lancet 1986; 1: 462-4.
11. Vane JR. Inhibition of prostaglandin synthesis as a mechanism of action of aspirin-like drugs. Nature (London, New Biology) 1971; 238, 232-5.
12. Berstad A. Dietary treatment of peptic ulcer. Scand J Gastroenterol 1987, 22 (Supp 129): 228-31.
13. Kang JY, Tay HH, Guan R, Math MW, Yap I, LaBrooy SJ. Dietary supplementation with pectin in the maintenance treatment of duodenal ulcer. A controlled study. Scand J Gastroenterol, 1988; 23: 95-9.
14. Kang JY, Yap I, Guan R, Lim TC. Chilli ingestion does not lead to macroscopic gastrointestinal mucosal damage in healthy subjects. J Gastroentero Hepatol 1988; in press.
15. Kumar N, Viji JC, Sarin SK, Anand BS. Do chillies influence healing of duodenal ulcer? Brit Med J 1984, 288: 1803-4.
16. Kumar N, Kumar A, Broor SL, Viji JC, Anand BS. Effect of milk on patients with duodenal ulcer. Brit Med J 1986; 293: 666.
17. Fung LH, Gilligan IJ, Talley NJ, Piper DW. Chronic duodenal ulcer, marital status and personality pattern. A case-control study. J Gastroenterol Hepatol 1986; 1: 251-6.
18. Thomas J, Greig M, Piper DW. Chronic gastric ulcer and life events. Gastroenterol 1980; 78: 905-11.
19. Tsoi WF, Kok LP, LaBrooy SJ, Kang JY, Lim KP. Psychological characteristics of duodenal ulcer patients. Ann Acad Med Sing 1983; 12: 532-4.
20. Weir DG. Peptic ulceration. Brit Med J 1988; 296: 195-200.
21. Annonymous. New drugs for peptic ulcer. Brit Med J 1980; 2:95-6.
22. Lane MR, Lee SP. Recurrence of duodenal ulcer after medical treatment. Lancet 1988; 1: 1147-9.
23. Annonymous. Peptic ulceration: the eye of the cyclone. Lancet 1986; 11: 1015-6.
24. Kang JY, LaBrooy SJ, Yap I, Guan R, Lim KP, Math MV, Tay HH. Racial differences in peptic ulcer frequency in Singapore. J Gastroenterol Hepatol 1987; 2: 239-44.
25. Alhady SA. The incidence of peptic ulceration in the three main races of Malaysia. Proceedings, Second Malaysian Congress of Medicine 1965; 2: 146-8.
26. Insert Malaysian article in present issue.
27. Kang JY. Peptic ulcer surgery in Singapore 1951-80, with particular reference to racial differences in incidence. Aust N Z J Med 1985; 15: 604-8.