CIGARETTE SMOKING AND DUODENAL ULCER DISEASE IN SINGAPORE

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SYNOPSIS

The effect of cigarette smoking on healing and relapse rates of duodenal ulcers seem to vary in different countries though the majority of evidence suggests that it retards healing of ulcers and increases relapse rates. We have studied the effect of cigarette smoking on both healing and recurrence of duodenal ulcer in 112 patients with endoscopically proven duodenal ulceration. They were treated with cimetidine (N = 65), Ranitidine (N = 25) or trimipramine (N = 22) for four or six weeks and ulcer healing was assessed endoscopically. 52 (46.4%) of the patients smoked. The majority of them were males (86.5%). There were no differences in age and duration of symptoms between smokers and non-smokers. Healing rates were similar for smokers (73.1%) and non-smokers (78.3%). 69 patients were followed up after their ulcers healed. They received no treatment and were re-endoscoped only if symptoms recurred. At the end of 6 months, significantly more smokers (81.2%) than non-smokers (40.5%) had recurrent ulceration ($P < 0.001$) and this was related to the number of cigarettes smoked.

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INTRODUCTION

It has long been accepted that cigarette smoking affects the healing of gastric ulcers. Recently, however, attention has been focused on the effect of smoking on duodenal ulcer healing. Results of studies to date are equivocal and seem to vary in different countries. A British study (1) found similar healing rates in smokers and non-smokers treated with cimetidine while others (2,3), have indicated that smoking adversely affects ulcer healing. Smoking has also been shown to increase the likelihood of relapse in duodenal ulcer patients (2,4).

This paper reports a study on the effect of cigarette smoking on both the healing and recurrence of duodenal ulcers in Singapore.

PATIENTS AND METHODS

112 patients (82 males, 30 females) with symptomatic, endoscopically proven duodenal ulcer were studied. There were 52 (46.4%) smokers and 60 (53.6%) non-smokers. 86.5% of the smokers were males. The mean age and duration of symptoms were similar between smokers and non-smokers (Table I).

65 (58%) patients were treated with cimetidine 0.8 or 1 gm per day, 25 (22.3%) patients with Ranitidine 300 mg per day and 22 (19.7%) with trimipramine 50 mg per day. Treatment was for 4 or 6 weeks and ulcer healing was confirmed endoscopically.

TABLE 1: PATIENT DETAILS IN CIGARETTE SMOKERS AND NON-SMOKERS

	No. of patients	Sex		Mean age (yr)	Mean duration of symptoms (yr)
		M	F		
Smokers	52	45	7	41.9	5.79
Non-Smokers	60	37	23	40.6	5.68
Total	112	82	30		

69 patients with (endoscopically proven) healed duodenal ulcers were followed up without therapy for 6 months. They were seen on at least 3 occasions during this period and were asked to come back to the clinic if their symptoms recurred. Only those who had symptomatic relapses were re-endoscoped. Smoking was not discouraged throughout the study and the cigarette consumption for each patient remained unchanged throughout this period.

Statistical analysis was by the X^2 test.

RESULTS

The overall healing rate of duodenal ulcers was similar for smokers (73.1%) and non-smokers (78.3%), (Table II). Healing rates were not influenced by the amount of cigarettes smoked daily (Table III).

TABLE II: ULCER HEALING RATES FOR SMOKERS AND NON-SMOKERS

	Non-Smoker	Smoker
Healed	47 (78.3%)*	38 (73.1%)
Not healed	13	14
total	60	52

*P > 0.5

The overall clinical recurrence rate was 59% (41/69). Recurrence rate for smokers was 81.2% (26/32) and for non-smokers 40.5% (15/37). This difference was significant (P < 0.001), (Table IV). Though the number of cigarette smoked per day had no influence on ulcer healing, recurrent duodenal ulcers occurred with significantly greater frequency in patients who smoked more than 10 cigarettes per day (p < 0.01) (Table V). Those who smoked less than 10 cigarettes per day had similar relapse rates to non-smokers.

TABLE IV: ULCER RELAPSE RATES FOR CIGARETTE SMOKER AND NON-SMOKERS

	Non-Smokers	Smokers
Total No	37	32
Recurrent ulcer	15 (40.5%)	26 (81.2%)*
No Symptoms	22	6

*P < 0.001

TABLE V: INFLUENCE OF CIGARETTE SMOKING IN ULCER RELAPSE

	<10 cig/day	10—20 cig/day	> 20 cig/day
Recurrent ulcer	4 (50%)	15 (93.8%)	7 (87.5%)*
No symptoms	4	1	1

*p < 0.01

DISCUSSION

Smoking has been implicated as an etiological factor in duodenal ulceration (2,4-9). In the United States, duodenal ulcers tend to heal less well amongst smokers whether on a high dose antacid regime (8) or on no treatment at all. Similar findings were noted by Korman et al in Australia (4), Sonnenberg and coworkers in West Germany (2) and by Blanchi's group in Italy (3). Bardhan, however, found no difference in the healing rates of duodenal ulcers amongst smokers compared to non-smokers in the north of England (1). This was true whether patients were on treatment or not. The above study also showed no difference in the ulcer healing rate among smokers and non-smokers. The differences in healing rates of duodenal ulcers in different regions of the world cannot be readily explained. The reasons for smoking are complex and varied and can be associated in most instances with a stressful personality or environment. These are themselves factors in the pathogenesis of duodenal ulceration. It could be that a heavy smoker and a stressful environment tend to heal his duodenal ulcer less well than one in a less stressful situation. Cigarette smoking has not however been shown to be of any beneficial effect on duodenal ulcer healing.

TABLE III: RELATIONSHIP OF NUMBER OF CIGARETTES SMOKED TO ULCER HEALING

	Non-Smokers	<10 cig/day	10—20 cig/day	>20 cig/day
Healed	47 (78.3%)	8 (72.7%)	23 (71.9%)	7 (77.8%)
Not healed	13	3	9	2
Total	60	11	32	9

After successful healing of duodenal ulcers, both Korman's and Sonnenberg's groups found significantly higher rates of relapse amongst smokers than non-smokers (2,4). Our local rsults were similar to the above. Patients smoking more than 10 cigarettes daily were noticed to have a significantly higher relapse of their duodenal ulcers after successful treatment compared with those non-smokers and those smoking less than 10 cigarettes daily. Most relapse occurred within 3 months after cessation of therapy which is in keeping with the natural history of the disease. Smokers do not relapse sooner than non-smokers in this respect.

Smoking is known to increase gastric acid secretion and to alter the motility of the duodenum. This together with decreased pancreatic bicarbonate output would all lead to an increase in duodenal acid load thereby creating ideal condition in the genesis and relapse of an ulcer-prone individual (10-14). The above could also explain the delay in healing of duodenal ulcers amongst smokers.

No studies have been conducted to compare the relapse rates of ex-smokers with that of smokers, although our data on the amount of cigarette consumption and ulcer recurrence rate would suggest a favourable drop in the rate of relapse amongst ex-smokers.

Patients with duodenal ulcers should therefore be advised against continued heavy smoking. Cessation of smoking is probably the cheapest adjunct to the treatment and prophylaxis of duodenal ulcer disease.

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