MEDICAL PRACTICE

General Practice Observed

Endoscopic studies of dyspepsia in a general practice

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Summary and conclusions

In an urban general practice serving 7800 patients, all patients presenting over five and a half years with dyspepsia lasting more than two weeks were investigated by fibreoptic endoscopy and cholecystography, and many by barium meal. Of the 393 patients with dyspepsia, 346 completed the investigation: 180 had specific disease of the oesophagus, stomach, duodenum, or gall bladder, including six with carcinoma. A further 67 had mucosal disease, and only 99 patients had no abnormality.

After the first year the number of patients presenting annually and the percentage of patients with specific lesions remained constant. The annual incidence for patients with dyspepsia was about 1% and for patients with specific lesions 0·4%, suggesting that each year those who became symptom free (either spontaneously or because of treatment) were balanced by a similar number who developed symptoms.

In contrast to the conclusions of other workers that an "open-access" endoscopy service could not be justified because the number of patients with specific lesions fell during their survey,[1] we suggest that such endoscopy services are indeed worth while for providing an accurate diagnosis of dyspepsia.

Introduction

We report on a general practice study of the epidemiology of conditions causing dyspepsia and the value of an "open-access" endoscopy service. In our study, which began in August 1973 and continued for five and a half years, we used the same symptom criteria for including patients and the same investigations throughout. Routine barium-meal examination was stopped after the first 300 patients had presented. We have published a preliminary report on the first 50 patients presenting in the first six months.[2]

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Patients and methods

Patients with dyspepsia presented to a single general practice run by three principals and one trainee practitioner serving a mainly urban community. The average number of patients in the practice was 7800. One endoscopist (RJB) examined all the patients within about four weeks of presentation. Brush cytology and multiple biopsy were[3 4] done on all specific lesions in the oesophagus and stomach. In addition, gastric biopsy specimens were taken from four standard sites from every patient to assess mucosal abnormality.[5] A standard symptom questionnaire was completed for each patient before endoscopy for follow-up studies.[6]

Results

During five and a half years 393 patients with dyspepsia were referred for endoscopy. Of these, 47 patients were either excluded on medical grounds or failed to continue. Of the 346 who completed the investigations, 180 had organic disease of the upper digestive tract. Specific lesions were found in 141 (table I), including six patients with histologically confirmed carcinoma, two of the oesophagus and four of the stomach. Seven patients had a benign stricture of the oesophagus and 22 hiatus hernia seen at endoscopy but no other lesions. Twenty-two patients had benign gastric ulceration, 42 duodenal ulceration, and 42 showed scarring and deformity of the pyloric canal or of the first part of the duodenum ("pyloroduodenal disease"). Cholecystography showed gall stones or an ill-functioning gall bladder in 26 patients.

A further 67 patients had evidence of mucosal disease of the stomach or duodenum but no specific lesions as defined in table I. Thirteen had evidence of a hiatus hernia on barium-meal examination, although this was not seen at endoscopy. The remaining 99 patients had no abnormality (table II).

TABLE I—*Specific lesions found on endoscopic examination of 346 patients between August 1973 and January 1979*

	No of patients	%
Carcinoma of oesophagus	2	0·6
Carcinoma of stomach	4	1·2
Benign oesophageal stricture with or without ulceration	7	2·0
Hiatus hernia	22	6·4
Gastric ulcer	22	6·4
Duodenal ulcer	42	12·1
Pyloroduodenal disease*	42	12·1
Total with specific lesions	141	40·8

*Evidence of previous ulceration with scarring and deformity of the pyloric canal or first part of the duodenum with or without inflammation.

TABLE II—*Summary of main findings at endoscopy*

	No of patients	%
Carcinoma of stomach or oesophagus	6	
Specific lesion of oesophagus, stomach, or duodenum (excluding carcinoma)	135	
Hiatus hernia on barium meal not seen at endoscopy	13	
Abnormal cholecystogram with no other finding	26	
Total	180	52·0
Mucosal disease with no specific lesion	67	19·4
No abnormality	99	28·6

TABLE III—*Number of patients presenting annually between 1973 and 1979 with dyspepsia, and findings at endoscopy*

	Year											
	1st		2nd		3rd		4th		5th		6th (half-year figures)	
	No	%	No	%	No	%	No	%	No	%	No	%
Specific lesion	46	48·4	21	38·2	21	41·2	19	38·0	25	37·8	9	31·0
Mucosal disease	16	16·8	13	23·6	7	13·7	14	28·0	13	19·7	4	13·7
No abnormality	23	24·2	10	18·2	15	29·4	14	28·0	23	34·8	14	48·3
Total No of patients examined	95	100·0	55	100·0	51	100·0	50	100·0	66	100·0	29	100·0
Total No of patients with dyspepsia	97		69		61		57		73		34	

TABLE IV—*Annual incidence of dyspepsia in our practice compared with two other practice populations*

	Practice population	No of patients with dyspepsia per 1000 patients per annum
Study practice	7800	12·2 (1st year) 6·9 (average over 5½ years)
Another urban practice	5770	10·4
Rural practice	5500	27·3

Analysis of the findings for each 12-month period (table III) shows that when the first year is excluded, between 60 and 70 patients presented each year, and about 38% had a specific lesion. Thus the annual incidence for patients with dyspepsia was about 1%, and for specific lesions about 0·4%.

To check on the incidence of dyspepsia in other general practice communities, one urban practice in a different part of the city and a rural practice 10 miles from Gloucester were asked to record all patients presenting with dyspepsia over several months (table IV). The criteria were the same as for this study. The incidence of dyspepsia in the urban practice was 10·4 per 1000 patients, similar to that of the study practice during the first year (12·2 per 1000 patients). The incidence in the rural practice was 27·3 per 1000 patients. This higher incidence may reflect a different assessment of the referral criteria.

Discussion

We expected initially that this survey would show a low incidence of organic disease and a large number of patients with no abnormality. Thirty of the first 50 patients examined, however, had a specific lesion of the upper digestive tract, a further four had gall-bladder disease, and nine had mucosal disease detected at endoscopy. Only seven patients had no abnormality.[2]

When so many patients were found to have organic disease during the first six months, we expected that the number of patients presenting for investigation would fall, as would the prevalence of disease. But the results over five and a half years showed that this was not the case. Although there was a higher referral rate and a higher percentage of patients with specific lesions during the first year, the numbers presenting for examination during subsequent years remained similar (average 54 per year). Furthermore, the percentage of patients with specific lesions remained constant from the second to the fifth year, with a slight trend downwards in the last six months. This supports the suggestion of Weir and Backett[7] that the number of patients with dyspepsia who become symptom free or die is balanced by a similar number who develop dyspepsia for the first time.

References

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What alterations from the normal can be expected in the blood films of patients who either smoke or drink to excess?

The commonest abnormality found in the peripheral blood film of heavy drinkers is an increase in red cell size (macrocytosis)[1]; in addition the reticulocyte count may be low, and there may be thrombocytopenia. Hypersegmented polymorphs are seen in those with co-existing folate deficiency. Macrocytosis is readily detected (as an increase in mean corpuscular volume) by electronic cell counters and is a useful screening test for alcoholism. Smoking also results in an increase in mean corpuscular volume, but only of about 2-3 fl.[2] It may also raise carboxyhaemoglobin (COHb) concentrations. Neither of these changes, however, would be apparent on examination of the peripheral blood film.

[1] Wu A, Chanarin I, Levi AJ. Macrocytosis of chronic alcoholism. *Lancet* 1974; i:829-31.

[2] Eschwege E, Papoz L, Lellouch J, *et al.* Blood cells and alcohol consumption with special reference to smoking habits. *J Clin Pathol* 1978;**31**:654-9.