

LETTERS TO THE EDITOR

Utility of surveillance colonoscopy in ulcerative colitis

EDITOR,—We have read with great interest the article by Lynch *et al* (*Gut* 1993; 34: 1075–80) examining the utility of colonoscopic surveillance in ulcerative colitis. We cannot agree, however, that their findings negate the value of regular surveillance in patients with ulcerative colitis.

In their study, only one patient whose cancer was detected as a direct result of cancer surveillance was found to have a favourable Duke's stage A cancer. In contrast, the patient who defaulted and developed carcinoma three years after the last surveillance colonoscopy died of metastatic disease. Similarly, most cancer patients who did not receive surveillance were found to have advanced disease. Instead of showing the futility of surveillance colonoscopy, we believe these results suggest a trend toward its benefit, although the number of patients may have been too small to derive a meaningful conclusion.

Several explanations may be provided for their difficulty in showing the benefit of colonoscopic surveillance. For example, there was only one patient who developed cancer among patients under regular cancer surveillance. It is obviously very difficult to draw a firm conclusion on the utility of a surveillance programme based on such a small number. One explanation for the small number of carcinoma detected in their programme is that the study period may not have been sufficiently long. This is reminiscent of a similar difficulty we and others have encountered previously that could be overcome only after an extended study duration.¹ In addition, the comparatively short mean disease duration of 14.1 years suggests that these patients were at comparatively low risk for developing cancer.² This aspect of the study may have been further compounded by the fact that 26% of patients in the surveillance programme had defaulted and 10% had had colectomy for failed medical treatment, leaving a smaller number of patients available for analysis. Taken together, their difficulty in detecting the benefit of surveillance may have been the result of the comparatively small sample size and short duration of study, rather than a true 'failure' of colonoscopic surveillance.

We agree that the current surveillance technique may benefit from reassessment. Their recommendation, however, of screening colonoscopy at eight years followed by an expectant management may not be the optimal solution. The clinical course of the patient who developed metastatic disease after initial negative surveillance colonoscopies emphasises some of the problems with this approach. Others^{1,3} have also shown a similar problem with an expectant management after a negative 'screening' colonoscopy. Instead of abandoning it altogether, a method of improving the diagnostic yield of colonoscopic surveillance may be what is necessary.

This may include adjustments of sampling interval and timing of colonoscopy to better utilise the rectosigmoid predominance of ulcerative colitis-related neoplasia⁴ and the duration dependent exponential increase in cancer risk.² Other proposals have included analysis of various molecular markers of neoplasia in patients at increased risk. With the better understanding of the natural biology of colorectal cancer complicating ulcerative colitis, it is hoped that these modifications would further enhance our ability to predict patients at increased risk and thereby effectively reduce the mortality related to colorectal cancer in ulcerative colitis.

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- 1 Choi PM, Nugent FW, Schoetz DJ Jr, Silverman ML, Haggitt RC. Colonoscopic surveillance reduces mortality from colorectal cancer in ulcerative colitis. *Gastroenterology* 1993; 105: 418–24.
- 2 Lashner BA. Recommendations for colorectal cancer screening in ulcerative colitis: a review of research from a single university-based surveillance program. *Am J Gastroenterol* 1992; 87: 168–75.
- 3 Camach SM, Rutter KRP, Brown RL. Colorectal carcinoma in patients with ulcerative colitis and recent colonoscopy. *Gut* 1993; 34: 1148–9.
- 4 Choi PM. Predominance of rectosigmoid neoplasia in ulcerative colitis and its implication on cancer surveillance. *Gastroenterology* 1993; 104: 666–7.

Reply

EDITOR,—We are grateful to Drs Kim and Choi for drawing attention to our paper.

They point out that the patient whose cancer was detected as a result of direct cancer surveillance had a favourable Duke's stage A. We accept that some patients undergoing surveillance do benefit, our concern is that the discovery of one early cancer for 476 colonoscopies is not the best use of resources. Furthermore, the fact that more cancers occurred in patients who defaulted or were not recruited for surveillance is mirrored in other studies and draws attention to the fact that surveillance is also ineffective because in practice protocols fail to survey those subjects who are at risk. These findings are confirmed by Dr Choi's own recent paper in *Gastroenterology*.¹ Had all 2050 of their patients been colonoscoped on a two yearly basis as they recommend the pick up rate of early cancer per colonoscopy over 12 years' surveillance would have been 1:512 (assuming that those not surveyed would have behaved in the same way as those who were under surveillance).

The problem that arises from more avid surveillance and closer follow up is that even more colonoscopies have to be performed for a diminishing return of cancers. The fact is that the incidence of cancer in ulcerative colitis is such that regular annual and biannual colonoscopic surveillance will never produce better results than those already published and it is therefore difficult to justify this form of surveillance on a cost/benefit basis.

Drs Kim and Choi criticise the size of our study. It is in fact the third largest in the world medical literature.

We accept that the length of history in the patients we surveyed was only 14.1 years, however, any surveillance programme that recruits at eight years from onset is bound to

have figures skewed towards the lower end, particularly as some patients default or are operated on over the years. The mean disease duration to diagnosis of carcinoma in the nine patients who did develop cancer was 18.2 years (7–26) so the period of surveillance is not greatly different taking into account those who dropped out of the study.

We agree with the comments on potential new methods of screening.

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- 1 Choi PM, Nugent FW, Schoetz DJ Jr, Silverman ML, Haggitt RC. Colonoscopic surveillance reduces mortality from colorectal cancer in ulcerative colitis. *Gastroenterology* 1993; 105: 418–24.

Duodenal ulcer pain – the role of acid and inflammation

EDITOR,—We read with interest the leading article by McColl and Fullarton (*Gut* 1993; 34: 1300–2). The authors argued that duodenal ulcer pain cannot be entirely due to the effect of duodenal acid on the ulcer crater and cited evidence for a weak correlation between the presence of active duodenal ulceration and epigastric pain. They suggested that duodenal ulcer pain is not directly related to the ulcer but perhaps to a combination of acid hypersecretion and mucosal inflammation, in the stomach as well as in the duodenum.

We agree that duodenal ulcer pain cannot be totally explained by the effect of acid on the ulcer crater. In our own studies on direct acidification of the duodenal ulcer crater in symptomatic duodenal ulcer subjects, ulcer pain was reproduced in only one third of cases.¹ We feel, however, that available data are still compatible with duodenal ulcer pain arising only from the duodenum. It is true that among patients with painful duodenal ulcer, correlation between ulcer pain and active ulceration is imperfect. In some,^{2–4} although not all⁵ of these studies, however, it is not stated whether residual erosive duodenitis was present in patients who were said to have healed ulcer. Erosive duodenitis may have the same significance as active duodenal ulcer in this respect in that there is a mucosal breach. In our own studies, we did not encounter subjects who remained actively symptomatic after total re-epithelialisation of their ulcer crater. Among treated asymptomatic subjects, however, whose last episode of pain occurred more than 24 hours previously, four of 14 with active ulcers developed pain compared with none of 20 whose ulcers had totally re-epithelialised ($p < 0.05$).⁶ This would seem to suggest that the presence of a mucosal breach in the duodenum may be important in the development of duodenal ulcer pain. The common finding of painless duodenal ulcer does not support the hypothesis that duodenal ulcer pain arises from inflamed antral mucosa as subjects with painless ulcer would be expected to have gastric mucosal inflammation also.

McColl and Fullarton did not consider the possibility that spasm or dysmotility may contribute to duodenal ulcer pain. Texter found that anti-cholinergics lead to cessation of gastroduodenal motility and abolishment of acid induced duodenal ulcer pain in 25 of 26 cases.⁷ Although other workers did not