LETTERS TO THE EDITOR

Painful rib syndrome

EDITOR,—The painful rib syndrome recently described by Scott and Scott is, in my opinion, a misnomer. Over the years I have seen numerous cases similar to the ones they report, and have found that the tender spots they allude to are not in the ribs but in the muscles. They are, in fact, myofascial trigger points. Pain develops because of trauma induced activation of nociceptors at these sites in what is now called the myofascial pain syndrome.1 These trigger points may be found in any muscle in the body. In the abdomen they commonly occur in the rectus abdominis and external oblique muscles. They do not only develop, however, at or near to their insertion into the ribs, but also in their bellies and at lower attachment sites such as the iliac crest, inguinal ligament, and pubic bones.

The pain emanating from trigger points in this syndrome may be abolished by injecting a local anaesthetic into them.2 Recently it has been shown that pain is also relieved by stimulating A-delta nerve fibres at these sites with dry needles; treatment that is physiologically more rational, being simpler, safer, and equally effective.3

Gastroenterologists must learn to recognise 'trigger point pain' because it is common and can be treated. The concept of the painful rib syndrome restricts the diagnosis to pain in the lower thorax and upper abdomen, as well as implying that there is no effective treatment other than reassurance. Trigger point pain may occur anywhere in the abdomen with additional sites in the perineum and back. The pain can be recognised easily so unnecessary investigations and operations are avoided. It usually responds quickly to acupuncture; further courses can be given if relapse occurs.

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Colonicoscopy surveillance in ulcerative colitis

EDITOR,—We read with interest the article by Lynch et al (Gut 1993; 34: 1075–80), and agree that the problem of defining those patients at risk of developing colorectal cancer poses great logistical problems. Yearly surveillance colonscopy did not detect most of the cancers in patients with colitis, but this was because nearly all patients in whom cancer eventually occurred fell outside their surveillance programme. Only three of nine patients who developed colon cancer had their disease initially assessed by colonscopy, and a further two patients had total colitis diagnosed by barium enema. We would suggest that ideally all patients with an initial diagnosis of colitis should have the extent of their disease assessed colonscopically, thereby better defining those patients deemed to be at higher risk of developing cancer.

It is obviously true that colonscopy will not prevent cancer from developing in the colitic colon because of the imperfect link between dysplasia and cancer, and because of the low proportion of the surface area of the colon biopsied during surveillance colonscopy. We would therefore propose that surveillance colonscopy should only be the following advice that colitic patients receive. It is artificial to separate colonscopic surveillance from proper clinical care of a patient with a condition that relapses and remits, and during which medical treatment may have to be interrupted. The largest prospective study of follow up of patients with colitis, 13 of 17 cancer patients in the surveillance programme had a Duke's A/B cancer, suggesting that this group of patients will have a better outlook than those presenting symptomatically.4 The finding of dysplasia in 22 patients treated by colectomy would possibly have prevented at least seven operations, thereby presenting a much higher incidence of early tumours compared with a non-prevention group, which translated into a survival advantage of five years that was statistically significant.

We feel that Lynch et al have been too pessimistic in their article on the value of follow up of colitis patients. Colonscopy performed every two years from the time of diagnosis is clearly feasible, requiring 12 colonoscopies per 100,000 population.5 All authors agree that the risk of developing colorectal cancer increases with duration and extent of disease.6 In addition, the onset of colitis is also associated with increased risk,7 although other studies suggest that older age of onset of colitis may be associated with a shorter interval to development of cancer.8 We do agree, however, that the type of follow up of patients with colitis deemed to be at high risk of developing colorectal cancer needs further thought and study. In the meantime, surveillance of high risk patients from the 10th year after onset of colitis seems a sensible approach.


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