Gastric epithelial dysplasia

SIR,—We read with great interest the two papers on gastric epithelial dysplasia in Gut.1,2 These are only the latest of a series, which testifies to the increasing interest in gastric precancerous conditions and lesions in the early diagnosis of cancer, but only partly shows a better understanding of problems relating to the diagnosis and interpretation of dysplastic changes in gastric mucosa. Most reports agree that severe, or high grade, dysplasia is the most important precursor of gastric cancer and should be treated as neoplasia, particularly in light of the high percentage of early gastric cancers which this approach enables us to diagnose.3,4 None the less, others have suggested, in a further paper published in an authoritative journal, that 'gastrocnemius is not always the treatment of choice for severe dysplasia and patients must receive a conservative clinical treatment and have frequent endoscopies until the appearance of early carcinoma.' Moreover, various papers report that mild, or low grade, dysplasia progresses to moderate dysplasia in only 9% of cases,5 is associated with or progresses to cancer in a small but significant percentage of cases, is6 not distinguished from high grade dysplasia in terms of evaluation of results,7 and is not even included among cancer precursor lesions.8 This is probably confusing for those who are not directly concerned in the problem and discouraging for those who would like to find in published papers a rational approach to pre-malignant gastric lesions. We think that the reasons for these contrasting results are as follows: (1) Gastric epithelial dysplasia is a rare diagnosis and in all the reports quoted (all of which appeared in authoritative journals) there were no more than 250 cases; only multicentre studies, such as those carried out by the British Society of Gastroenterology and in which we also collaborated, are therefore likely to provide us with sufficient information. (2) As Lansdown and coworkers correctly emphasized,9 distinction of the two forms, particularly in its mild form, from atypical hyperplasia is not done easily or always reliably; we think that the concept of mild dysplasia is changing and that only five years ago we were confident in saying that mild dysplasia was not an indication for follow-up, we now consider follow-up of these lesions, when correctly classified, to be mandatory.10 (3) The stomach is a relatively large organ and in the absence of a persistent focal lesion it is difficult to target biopsies and ensure that samples are obtained from the same site (which is why 'regression of severe dysplastic lesions is reported so often). (4) Few papers have been published with results from a truly prospective study, and retrospective investigations, particularly in this field, are burdened by the risk of bias. Nevertheless, we think that a few clinical aspects are fairly well established. Firstly, severe, or high grade, dysplasia, whether associated with gastric ulcer, polyps, erosions, or any endoscopic change, is the most reliable indicator that cancer is present or will develop in a short time and that patient must therefore undergo surgery when feasible. We think that such a policy will save the patient and the doctor medical and legal problems. Secondly, new prospective and multicentre studies focusing more on mild and moderate or low grade dysplasias are needed because we still do not know the relative risk of cancer for each type of lesion (though we have made an attempt in this direction), whether it is justified to consider moderate dysplasia as a separate entity, or how to follow up such patients. Finally, we agree that when expert advice is not available locally specimens suspected of dysplastic changes should be examined by expert pathologists, who should be entrusted with educating, with suitable tools, their colleagues in the field.

This work was done with the support of the R Farini Foundation for Gastroenterological Research.


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