in our series was surprisingly high. Conversely, their experience appears unusually good, even if compared with the prospective trial reported by McDougall et al.1 who used a similar sclerosing technique. The major question is which techniques provide the best protection against bleeding. We are willing to pay fairly heavily in terms of non-lethal, treatable complications such as dysphagia and stricture for a lesser risk of rebleeding. This risk is greatly variable among patients with oesophageal varices. It is very important to obtain balance in the pretreatment risk between groups of patients compared with the efficacy of various techniques of sclerotherapy. We believe that this balance cannot be established except by using the closed randomisation procedure.

We would like also to comment on the possibly misleading manner of reporting the risk of rebleeding presented by McCormack and Johnson and by others.1 By dividing the total number of rebleeds in a series of patients by the total number of patient-months of follow up, an undue weight is given to long term survivors with few, or no episodes. To avoid this dilution of the denominator, we suggest the calculation of the incidence for each patient (number of rebleeds per months) and having this as the basis of reporting. The average, or median – for specified intervals of time after the first bleeding – of these individual risk estimates with appropriate measures of between patient variation would in our view give a more adequate picture of the rebleeding.

T I A SØRENSEN, F BURCHARTH,
M L PEDERSEN, AND F LINDAHL
Dept of Surgical Gastroenterology,
Herlev University Hospital,
DK-2730 Copenhagen,
Denmark.

Reference


More about refractory duodenal ulcers

SIR,—I was interested to read, in the July 1984 issue of Gut, the papers by Pounder,1 Lam et al.2 and Bardhan3 on the problem of refractory duodenal ulcers. I would like to discuss some personal experiences in this field, further to a previous report4 that Dr Bardhan was kind enough to quote in his paper.3

Since submission of our abstract to the BSG in December 1982, the number of refractory subjects has increased. To date 32 duodenal ulcers resistant to an eight week treatment with cimetidine 1 g daily (19 patients) or ranitidine 300 mg daily (13 patients) have been shifted to sucralfate therapy for six weeks.

The healing rate was 81.2%, which is approximately the same observed with the first 20 cases.4 The remaining six patients showed neither healing nor reduction in ulcer size even after treatment was changed.

The absence of a control group of subjects continuing treatment with H2-blockers makes it impossible to definitely conclude that ulcer healing was because of the change of therapy rather than to the mere prolongation of treatment. It seems unlikely, however, that more than 80% of our subjects were just 'slow responders'. Indeed ulcers the size of which remained unchanged after eight weeks of therapy with H2-blockers can hardly be expected to completely heal during the following six weeks of treatment to such an extent. By and large our study, although unfortunately uncontrolled, compares well with the results by Dr Lam et al2 with tripotassium dicitrato bismuthate (the mode of action of which partially resembles that of sucralfate). This seems to confirm the view that in refractory duodenal ulcers mucosal protection may be more effective than control of gastric acidity.

As for the profile of patients who failed to respond to H2-receptor antagonists, we were unable to detect any particular features regarding sex, age, disease history, and gastric secretory capacity. Contrary to Lam’s suggestion2 and in agreement with Bardhan,3 smoking habits did not appear to influence the lack of response to H2-blockers. On the other hand, refractory ulcers appear to represent a subgroup of duodenal ulcers with a different pathophysiology.2

Our incapacity to recognise the factors involved emphasised once again how little we know of the disease we are treating. At the present time we are unable to predict which subjects may benefit from therapy with antisecretory drugs or with agents strengthening mucosal defences. As long as we cannot discriminate between different types of ulcers, we should expect the usual 20% of error (treatment failure) in the choice of the drug. Hopefully the dilemma might be solved in the future by using prostaglandins with both antisecretory and cytoprotective activities.

M GUSLANDI
3rd Medical Clinic,
University of Milan,
Milan,
Italy.
Correspondence

I realise that reading the text reveals that the figure is 18. Because summaries are used as abstracts by many journals, however, and consequently read by a larger number of people than would read the complete article, then a false impression will be given. Thirty five is twice the figure found in the experiment or 100% instead of 50%!

As Dr McMillan and his co-authors are using information and figures directly related to my own research, I feel I must ask that they are correctly reported.

P G SARGEAUN T

Amoebiasis in homosexual men

Sir,—I wish to bring to your notice errors that appear in the article by Dr A McMillan et al (Gut 1984; 25: 356–60). These authors say: 'Based on their association with dysentery or hepatic abscess formation these workers consider that only amoebae of zymodeme types II, VI, VII, and XI are pathogenic'. This statement is wrong, the authors to whom Dr McMillan is referring quite clearly state: 'The accompanying figure shows the isoenzyme patterns for all the zymodemes so far demonstrated. Many of these zymodemes have been demonstrated in E histolytica from various areas of the world. The marker for pathogenicity is the presence of a β band and absence of an α band in PGM. This feature is confirmed, with the exception of zymodeme XIII by the advanced bands in HK'.

The accompanying figure referred to above is shown quite plainly to show that not only are zymodemes II, VI, VII, and XI pathogenic but also XII, XIII, and XIV. This mistake is inexcusable because it may lead the reader to a false impression of pathogenic amoebic infections. It is difficult to understand how such a gross error has occurred, particularly when it is known that Dr McMillan and his co-authors are aware of another published article, Entamoeba histolytica in male homosexuals,1 in which all 18 zymodemes were again clearly figured.

The authors write that 'Isolates from 18 men were available for isoenzyme characterisation. Each was of zymodeme type I'. They do not relate, however, the zymodeme to any clinical, histopathological, or serological feature. In that case, what value is it then to know the zymodeme?

Leaving the above points apart I must protest in the strongest fashion possible about the summary which contains a thoroughly ambiguous statement. This quite clearly gives the impression that 35 homosexual men were infected with Entamoeba histolytica expressing zymodeme I. Surely this is not true!


References


Reply

Sir,—We thank Mr Sargeaunt for his comments. Regardless of what zymodeme types he considers ‘pathogenic’, Mr Sargeaunt considers type I amoebae to be non-pathogenic. The statement that ‘the vast majority of homosexuals are passing only non-pathogenic amoebae’ is gravely misleading; neither clinical, histological, nor serological data were presented to substantiate this assertion. This view has been challenged in our paper: 83% of the 18 individuals excreting type I amoebae showed histological evidence of proctitis (grades B and C) which resolved after anti-amoebic treatment. Since the completion of the reported study, a further 38 homosexual men with amoebiasis have been identified. Entamoeba histolytica was the sole pathogen or potential pathogen detected in 31 of these men; 24 (77.4%) had proctitis (19 grade B; five grade C). Treatment with diloxanide furoate only was associated with resolution of the proctitis; when therapy was delayed, the proctitis persisted.

When the pathogenicity of an organism is considered, host factors cannot be ignored. As a result of careful contact tracing, we found that eight asymptomatic cyst excreters (three of whom had been infected with amoebae of zymodeme type I) who subsequently developed symptomatic amoebiasis.

A MCMILLAN, G J C MCNEILLAGE, N M GILMOUR, AND G R SCOTT

Dept of Genito-Urinary Medicine, Royal Infirmary, Edinburgh.

Amoebiasis in homosexual men

Sir,—I wish to bring to your notice errors that appear in the article by Dr A McMillan et al (Gut 1984; 25: 356–60). These authors say: 'Based on their association with dysentery or hepatic abscess formation these workers consider that only amoebae of zymodeme types II, VI, VII, and XI are pathogenic'. This statement is wrong, the authors to whom Dr McMillan is referring quite clearly state: 'The accompanying figure shows the isoenzyme patterns for all the zymodemes so far demonstrated. Many of these zymodemes have been demonstrated in E histolytica from various areas of the world. The marker for pathogenicity is the presence of a β band and absence of an α band in PGM. This feature is confirmed, with the exception of zymodeme XIII by the advanced bands in HK'.

The accompanying figure referred to above is shown quite plainly to show that not only are zymodemes II, VI, VII, and XI pathogenic but also XII, XIII, and XIV. This mistake is inexcusable because it may lead the reader to a false impression of pathogenic amoebic infections. It is difficult to understand how such a gross error has occurred, particularly when it is known that Dr McMillan and his co-authors are aware of another published article, Entamoeba histolytica in male homosexuals,1 in which all 18 zymodemes were again clearly figured.

The authors write that 'Isolates from 18 men were available for isoenzyme characterisation. Each was of zymodeme type I'. They do not relate, however, the zymodeme to any clinical, histopathological, or serological feature. In that case, what value is it then to know the zymodeme?

Leaving the above points apart I must protest in the strongest fashion possible about the summary which contains a thoroughly ambiguous statement. This quite clearly gives the impression that 35 homosexual men were infected with Entamoeba histolytica expressing zymodeme I. Surely this is not true!