Letters to the Editor

Effect of Helicobacter pylori on the gastric mucous gel

EDITOR,—In their paper on the basis of viscometrical studies performed on human gastric mucus Markesich et al challenge the concept that Helicobacter pylori exerts unexpected effects on the mucous layer coating the gastric mucosa (Gut 1995; 36: 327-9).

Their results are clearly at variance with the bulk of the experiments carried out in this area1 and Markesich and colleagues try to show the reasons for this discrepancy by pointing out the potential drawbacks of the previous studies.

Even if we accept Markesich’s concept, however, that the results of some experiments on H pylori have to be discarded because they are not performed on human mucosa, other data are consistent with a mucus damaging effect of the microorganism in humans. Assessing the quality of gastric soluble mucus in humans we found that, although on the whole mucus is normal both in chronic gastritis and in duodenal ulcer, the lowest values are detectable in subjects with H pylori infection.2

Sidebotham et al also observed a breakdown of gastric mucus in patients infected with H pylori, although they ascribed this phenomenon to a carbonate-bicarbonate buffer at the mucosal surface due to the urease activity of the germ, rather than to a direct mucolytic activity.4 Measurements of the gastric mucous gel thickness in vivo showed that the mucous coating is significantly thinner in H pylori infected patients,5 possibly resulting from increased erosion.

In keeping with the above results H pylori infection seems to reduce mucus hydrophobicity of human gastric mucous, which returns to normal after successful eradication.6

The reason for the discrepancy of Markesich’s results is probably the method used to assess the mucus. Reference should be made to a study7 to justify the use of viscometry analysis for assessing the characterisation of mucus, but it is well recognised that viscometry is an unreliable technique for evaluating mucous properties.8 The finding that gastric mucous viscosity is increased in duodenal ulcer patients9 is only a further demonstration of how misleading the method can be.

As only luminal mucus was examined in Markesich’s study, it must be also considered that proteolytic enzymes produced by H pylori could promote a greater peptic erosion of the adherent mucous gel with consequent increase in the mucoprotein content of gastric juice influencing viscosity measurements. Eradication of H pylori would clearly reduce mucus shedding and thus apparently decrease the viscosity of intraluminal mucus.


Reply

EDITOR,—Gulisandi takes issue with our finding that gastric mucus gel in H pylori patients has a higher viscosity than in the unaffected. We simply gathered and tested the clear mucous gel that is normally present in the stomach. We used standard methodology for evaluating the viscosity of a gel. Our data were both consistent and reproducible.

There are many possible reasons why the gastric mucus gel in patients with H pylori infection may be more viscous than those without. As the mucus cells seem to be under pressure to actively secrete, the gel may be younger than normal. It may also contain many more cells and cellular debris including DNA, which will in itself enhance viscosity. We make no apologies for the fact that Dr Gulisandi studied soluble mucous and obtained different results.1 This should not be a surprise as soluble mucus is ‘used’ mucus that has completed its function and is probably better considered a waste product. The fact that the mucous gel coat might be thinner in H pylori infected patients, tells nothing about the rate of synthesis, erosion, effectiveness or viscosity. We have previously shown that changes in mucosal hydrophobicity can be separated from the H pylori infection.2 This mucosal hydrophobicity seems to be more likely to be due to the inflammatory infiltrate (as has been previously shown in the colon) than for any factor made by the bacteria.3 More than 40 years ago, Hollander described increased viscosity in response to damage and other workers in gastric physiology who previously published on breakdown of gastric mucous have had no difficulty incorporating our findings.4

The histological picture of peptic ulcer disease has been one of exploring what turned out to be blind alleys. Researchers have seen their cherished beliefs destroyed and discovered that their lifelong work has not been in the main stream of research. We did not investigate any merely a triviality. The truth will be discovered over time and only by additional experimentation.


Audit of percutaneous liver biopsy

EDITOR,—The 50% liver biopsy rate cited by Gilmore et al (Gut 1995; 36: 437-41) for patients aged >65 with suspected malignancy would only be justified if concurrent clinical, biochemical, and ultrasonographic stigmata were equivocal. Given the fact that the diagnostic specificity of ultrasonography can be as high as 96-2%, with corresponding likelihood ratio=18 (depending on subtype of ultrasonographic stigmata),1 we can utilise the principles of Bayes’ theorem2 to predict the probability of malignancy in a subgroup of patients who already have clinical and biochemical stigmata of this diagnosis. Under such circumstances the minimal increment in post-test probability, generated by the adjunctive use of needle biopsy, might well be largely offset by the risk of procedure related morbidity and mortality.

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