Effect of Helicobacter pylori on the gastric mucus gel

Editor,—In their paper on the basis of viscosimetric studies performed on human gastric mucus Markesich et al challenge the concept that Helicobacter pylori exerts unfavourable 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 present studies.

Even if we accept Markesich’s concept, however, the results of some experiments on H pylori have to be discarded because they are not performed on human mucus, other data are consistent with a mucus damaging effect of the micro-organism 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 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, possibly resulting from increased erosion.

In keeping with the above results H pylori infection caused a marked mucosal hydrophobicity of human gastric mucus, which returns to normal after successful eradication.6

The reason for the discrepancy of Markesich’s results is probably the method used to test the hypothesis. Reference is made to a study7 to justify the use of viscosity analysis for assessing the characteristics of mucus, but it is well recognised that viscosimetry is an unreliable technique for evaluating mucus properties.8 The finding that gastric mucous viscosity is increased in duodenal ulcer patients7 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 mucus 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.

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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 is 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 post-test probability of liver biopsy in the 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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Editor,—One of the benefits of this audit is that it has supported the widely held belief that coagulopathy predisposes to haemorrhagic complications following liver biopsy. It is surprising that previous large reviews have, in contrast, failed to support this belief, with the exception of haemophilic patients undergoing biopsy.1,4

It would have been interesting to know in the audit what measures, if any, were taken to correct the coagulation abnormalities before biopsy; practice by biopsy varies between different units. Therein may lie the reason for the different incidence of haemorrhagic complications between this and other audits.

There are few data on which to base decisions about when coagulopathy is a contra-indication to liver biopsy. Parenteral vitamin K should be given at least six hours beforehand, but if coagulopathy persists then fresh frozen plasma is the agent of choice for correction.5 The level of prothrombin time at which fresh frozen plasma should be given has not been defined but a prolongation of three seconds or more is generally accepted to require correction. If fresh frozen plasma is given then the dose should follow published guidelines — that is, 12–15 ml/kg body weight,7 given immediately before the biopsy.


