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 mucus (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 mucus, 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,2 the lowest values are detectable in subjects with H pylori infection.3

Sidebotham et al observed a breakdown of gastric mucin 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 mucus 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 induced erosion of mucus and 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 by them. Reference to a study7 to justify the use of viscosimetry 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 mucus 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 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.


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 present. Given the fact that the diagnostic specificity of ultrasonography cannot be high as 96%–2% with corresponding likelihood ratio = 18 (depending on subtype of ultrasonographic stigmata),1 one can utilise the principles of Bayes’ theorem2 to predict the post-test probability of malignancy for this 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 haemophiliac patients undergoing biopsy.1

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

There are few data on which to base decisions on the use of anticoagulation in patients with coagulopathy before 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.2

The level of prothrombin of the 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.3 If fresh frozen plasma is given then the dose should follow published guidelines — that is, 12–15 ml/kg body weight,4 given immediately before the biopsy.
Increased CA 125 in tuberculous peritonitis

EDITOR,—O’Riordan et al reported the case of a patient with tuberculous peritonitis, ascites, and pleural effusion who also had a remarkably increased concentration of serum CA 125 (Gut 1995; 36: 303–5). Because of the increase in this tumour marker an ovarian cancer was suspected and a laparotomy was performed with negative results for cancer. The authors consider the increased values of CA 125 the most interesting aspect of the case, and review other published reports on increased CA 125 values in patients with tuberculous ascites.

I believe, however, that the only interesting and noticeable aspect of the case would have been if the patient had had a normal value of CA 125. This tumour marker, commonly used in the diagnosis of ovarian cancer, increases in a variety of processes involving pleura, pericardium, and peritoneum including endometriosis, peritonitis, pelvic inflammatory disease, and surgical trauma. The increase in CA 125 is not unexpected because this antigen has been detected on mesothelial cells in pleura, pericardium, and peritoneum, particularly in areas of inflammation.

Benign peritoneal effusions, particularly ascites, are associated with increased serum concentrations of CA 125 with values up to 100 times the upper normal limit in some cases. We have proved to be an excellent marker of tuberculous peritonitis, and increased serum CA 125 is very low in the presence of ascites of whatever origin.

In addition, the return of CA 125 to normal after anti-tuberculous treatment in the patient of O’Riordan et al is not unexpected. CA 125 decreases to normal values when the ascites is removed in cirrhotic patients and increases again when ascites recur.

The authors conclude that 'tuberculous needs to be considered in the differential diagnosis of ascites with increased tumour markers'. It is well known, however, that CA 125 is an unspecific marker of ascites of whatever aetiology. Tuberculous peritonitis, a disease that only uncommonly produces peritoneal effusions, represents only one of the many aetiologies of ascites. Finally, I believe that a laparotomy should not have been performed in this case because both computed tomography and cytological study of ascitic fluid were negative for ovarian cancer and the very high concentration of CA 125 could have been easily explained by the existence of both ascites and pleural fluid.