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Bugs and bleeding varices ►

▲ Hou M-C, Lin H-C, Liu T-T, *et al*. Antibiotic prophylaxis after endoscopic therapy prevents rebleeding in acute variceal hemorrhage: a randomized trial. *Hepatology* 2004;**39**:746–53.

Several lines of evidence indicate that patients with cirrhosis presenting with gastrointestinal bleeding have a higher risk of developing bacterial infection and this in turn may be associated with early rebleeding. A meta-analysis of randomised controlled trials concluded that antibiotic prophylaxis not only reduced the incidence of infections in these patients but also improved survival. Despite this, antibiotic prophylaxis does not seem to have become common practice in patients with variceal bleeding.

Hou *et al* randomised 120 patients with acute gastro-oesophageal variceal bleeding, without any evidence of infection at the time of presentation, to receive prophylactic antibiotic (ofloxacin 200 mg twice daily for seven days) or to be treated with antibiotics when infection became evident (on demand). Clinical and endoscopic findings, timing, and type of endoscopic therapy were similar in the two groups. The incidence of bacterial infection was much lower in patients receiving prophylactic antibiotics than that in the on demand group (3.4% *v* 26%). Antibiotic prophylaxis reduced the actuarial probability of rebleeding, mostly by its effect on early rebleeding (within seven days). There was no difference in survival between the two groups (85% in both groups at six months) although the study does not appear to have been powered to detect this.

Short term antibiotic prophylaxis should be considered standard care in cirrhotic patients with variceal bleeding. Nevertheless, yet again a therapy with confirmed efficacy in reducing rebleeding in patients with bleeding varices has failed to show an effect on survival!

Not just in children ►

▲ Potter JW, Saeian K, Staff D, *et al*. Eosinophilic esophagitis in adults: An emerging problem with unique esophageal features. *Gastrointest Endosc* 2004;**59**:355–61.

Patients with dysphagia but a seemingly normal endoscopy pose diagnostic and management problems and are often labelled as suffering from refractory gastro-oesophageal reflux disease despite lack of convincing evidence. Eosinophilic oesophagitis is well recognised in children but is now being increasingly recognised in adults, yet little is known of the characteristic endoscopic and clinical features in adults. The authors report their analysis of 29 adults with this disorder. A total of 83% were referred with dysphagia for solids, of whom 33% had suffered at least one episode of unexplained food impaction. Only 28% had a history of chronic heartburn while almost half had a history of atopy or asthma. Twenty five (86%) had atypical structural features at endoscopy, the predominant ones being a small calibre oesophagus, corrugations or concentric rings, and proximal stenoses, often of sufficient severity to prevent passage of a standard adult endoscope. Whitish elevated plaques (representing eosinophilic microabscesses), linear mucosal furrows, and an oedematous mucosa with obscured subepithelial detail were also noted commonly. These features occurred in the absence of endoscopic or histological evidence of acid reflux injury. A further notable finding was the extensive mucosal disruption or ''fracturing'' seen after dilatation. Like many aspects of diagnostic practice, the key is a high index of suspicion, along with careful inspection of the mucosa for subtle abnormalities and a low threshold for taking biopsies from a ''normal'' oesophagus in patients presenting in this way.

A *Helicobacter* causing colitis? ►

▲ Kullberg MC, Andersen JF, Gorelick PL, *et al*. Induction of colitis by a $CD4^+$ T-cell clone specific for a bacterial epitope. *Proc Natl Acad Sci USA* 2003;**100:**15830–5.

Although the cause(s) of inflammatory bowel diseases remains to be established, it is recognised that the intestinal flora plays a pivotal role. As these authors (among others) have shown, pathogen free interleukin 10 knockout mice escape colitis until exposed to commensal or pathogenic bacteria, including *Helicobacter hepaticus*. However, it has been unclear whether bacteria are the specific target of the immune response that induces colitis or whether they act indirectly by triggering an anti-self response. In an elegant series of experiments, the authors of this paper have shown that a T cell clone specific for soluble *H hepaticus* antigen transferred colitis to T cell deficient *H hepaticus* infected RAG knockout mice. Uninfected controls did not develop colitis when exposed to the T cell clone, nor did positive controls exposed to a T cell clone specific for another bacterial antigen (*Schistosoma mansoni*). Furthermore, the antigen recognised by the colitis inducing T cell clones was shown to be a 15mer epitope on the flagellar hook protein of *H hepaticus*. The clone did not recognise *H pylori* or other bacterial species. So could colitis be the consequence of infection by a little recognised species of *Helicobacter*? The experiments certainly present convincing evidence that colitis in RAG knockout mice is provoked by a highly specific T cell response to a peptide epitope on the flagellum of *H hepaticus*. This is the first time that a T cell response sufficient to induce colitis has been demonstrated. It is also the first time that the target antigen and specificity of the colitis inducing T cell response has been identified. It is therefore reasonable to conclude that an aberrant T cell response to specific components of the gut flora is likely to cause some forms of colitis in humans, but it seems too much to hope that *H hepaticus* is also the culprit.

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