Transluminal endoscopic necrosectomy: revisited from the surgeon's perspective

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LETTERS

Novel genetic marker for dilated end stage oesophagus and oesophageal adenocarcinoma risk?

With interest we read the article by Asling et al,1 entitled “Collagen type III (COL3A1) is a gastro-oesophageal reflux disease (GORD) susceptibility gene and a male risk factor for hiatus hernia”, which has been published in a recent issue of Gut. The major findings of the study were that a single nucleotide polymorphism within the gene encoding for collagen type III (COL3A1) was associated with GORD in both sexes and hiatal hernia in males. In keeping with these findings, immunohistochemistry of oesophageal biopsies showed increased connective tissue abnormalities (ie, collagen type III-positive staining) in GORD patients compared with asymptomatic controls.1 GORD has been defined by symptoms, endoscopy and pH monitoring data.2 3 Furthermore, the study is based on the model anatomy that the stomach commences at the level of the rise of the diaphragm, result in gastric type folds covered by the lower oesophageal sphincter, possibly induced by repeated distension, function and shortening of the lower oesophageal sphincter, possibly induced by repeated distension, result in gastric type folds covered by metaplastic columnar epithelium (ie, cardiac, oxyntocardiac mucosa or intestinal metaplasia equals Barrett’s oesophagus). At some point the dilated end stage oesophagus extends proximal to the level of the diaphragm inducing widening of the oesophageal hiatus. During endoscopy this dilated columnar lined distal oesophagus may be mistaken for hiatal hernia. Therefore differentiation between hiatal hernia and dilated end stage oesophagus requires histopathology of biopsies obtained at and above the level of the diaphragmatic impression.2 4 The data obtained by Asling et al1 strongly indicate that mutations within the COL3A1 gene favour the formation of dilated end stage oesophagus. COL3A1 associated dysfunction of the antireflux barrier may contribute to the development of columnar metaplasia, dysplasia and adenocarcinoma of the oesophagus. Thus, identification of COL3A1 gene mutations may act as a marker for increased risk of oesophageal adenocarcinoma and explain why gene mutation induced connective tissue alterations are linked to cancer development.4 The authors are kindly asked to comment on these considerations.

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Authors’ response

We sincerely appreciate the interest in our recent paper in Gut entitled “Collagen type III (COL3A1) is a gastro-oesophageal reflux disease (GORD) susceptibility gene and a male risk factor for hiatus hernia”.1 The diagnosis of hiatal hernia requires endoscopy, a barium meal and/or manometric assessment.2 Histology is not required for confirmation. Barrett’s oesophagus, however, does need histopathological confirmation, to safely separate it from oesophageal squamous epithelium.3 In fact, this has been carefully investigated in the original Kalixanda cohort where the prevalence of Barrett’s was determined to be 1.6% in the general population and 2.5% in patients with reflux symptoms.4 The cohort we have used to assess hiatal hernia association is the Kalixanda cohort with the addition of 100 GORD patients with the same demographics. The Kalixanda cohort contains 16 individuals affected with Barrett’s oesophagus. The 100 GORD patients add an additional two or three Barrett’s cases. In total, we expect 18–19 Barrett’s cases in the cohort we have used. Even if all of these have been misdiagnosed, they amount to less than 10% of the 239 hiatal hernia cases. It is therefore clear that the association we report, where we show that one single nucleotide polymorphism in COL3A1 is associated with hiatus hernia, cannot be attributed to lack of differentiation between hiatal hernia and metaplastic columnar epithelium. Unfortunately, due to the low frequency of Barrett’s and hence adenocarcinoma, the present study does not allow the investigation of biomarkers for these conditions.

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In a retrospective analysis of 93 patients, Seiffert et al2 investigated the role of transluminal endoscopic necrosectomy after acute pancreatitis. From the authors’ point of view, the mortality rate of only 7.5% and a good long-term outcome appear to make this relatively new approach look favourable when compared with the results of published studies on surgical debridement in necrotising pancreatitis. However, a detailed look at the characteristics of the patients in the GEPARD study shows that this is like comparing apples and oranges. Surgical series on acute pancreatitis predominantly investigate the subset of patients with infected necrosis and severe organ dysfunction. Data on the severity of the disease are regularly presented, usually as severity scores (eg, Ranson, APACHE II, SOFA), the incidence of single or multiple organ failure or the extent of pancreatic necrosis.3 4 However, severity data are missing in the GEPARD study. Importantly, the patients in the GEPARD study appear to be much healthier. Their predominant symptoms were abdominal pain and gastric obstruction/vomiting. The majority of patients with so-called sepsis

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Metabolic syndrome on top of chronic hepatitis B: the more, the worse?

We read with interest the article entitled ‘Metabolic syndrome increases the risk of liver cirrhosis in chronic hepatitis B’ by Wong et al in the January 2009 of Gut.1 Chronic hepatitis B virus (HBV) infection remains a global health problem, affecting more than 350 million people worldwide. It is estimated that approximately one million chronic hepatitis B (CHB) patients die of liver-related diseases per annum. Although the launch of an effective hepatitis B vaccination programme for more than two decades, a dramatic decline of HBV infection rate as well as hepatocellular carcinoma (HCC) incidence rate has been observed in children of many HBV endemic countries.2 Nevertheless, CHB is still a health threat to adult HBV carriers. Therefore, it is important for practising gastroenterologists to understand more about the aggravating factors of chronic HBV infection. In the past decade, both host and viral factors have been shown to affect the development of adverse clinical outcomes in HBV carriers. In addition, recent data showed that environmental and metabolic factors may also contribute to the progression of disease. Among these risk factors, metabolic syndrome (MS) is an emerging comorbidity that should be paid attention and actively controlled in the future.

MS is a worldwide health problem accompanying the westernisation of lifestyle. For example, one nationwide cross-sectional population-based survey in Taiwan found that the overall prevalence of obesity was 19.2% in men and 13.4% in women, indicating a high prevalence of obesity and MS in Taiwan. Accumulating evidence suggests that MS may adversely influence the progression and treatment outcomes of chronic hepatitis C patients. In contrast, whether MS has a similar impact on the progression of HBV-related liver diseases is in its infancy. One cohort study enrolling 2903 HBsAg-positive males in Taiwan with a mean follow-up of 14.7 years demonstrated the positive correlation between the risk for fatty liver, cirrhosis, incident HCC and liver-related mortality as well as the predictive value of body mass index.3 Another 14-year follow-up study in Taiwan showed that obesity and type 2 diabetes synergistically contributed to the development of HBV-related cirrhosis and HCC.4 In this article, although Wong et al provided additional data to this area of active investigation, several issues need to be clarified.

First, this was a cross-sectional study, and the causal relationship between MS and HBV-related cirrhosis could not be confirmed. Second, enrolled patients were referred from specialists rather than general practitioners, and so the results may not represent patients with whole disease spectrum but represent those with more severe liver disease. Accordingly, whether the association of MS with more severe liver disease holds true for general HBV carriers remains unclear. Third, the gold standard for the diagnosis of metabolic syndrome is controversial. Different ethnic groups may require different criteria to evaluate the prevalence and severity of MS before assessing the association of MS with HBV-related liver fibrosis. Finally, patients with MS frequently suffer from central obesity, and so the value of transient elastography (TE) to evaluate liver fibrosis in this particular study might be doubtful. TE was initially recommended for the evaluation of hepatic fibrosis stage in treatment-naive chronic hepatitis C patients. Clinical usefulness of TE in patients with chronic hepatitis B needs further confirmation, particularly in the presence of MS.

In summary, even if MS is an important determinant of the progression of HBV-related cirrhosis, further longitudinal and population-based cohort studies from different parts of the world are still needed to clarify the genuine impact of MS on the clinical outcomes of chronic HBV infection. Moreover, the influence of MS on the treatment outcomes of CHB patients is also interesting and deserves further examinations.

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Competing interests None.

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