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

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(defined as fever alone, C-reactive protein levels raised to eight times normal, bacteremia or positive FNA culture) probably would not fit the well-established international definition of sepsis. Only 20% of the patients analysed had been treated in the ICU after admission, only 4.3% showed signs of septic shock and all endoscopic interventions had been possible under sedation. Importantly, infected necrosis was only present in 54% of patients. Indeed, what the GEPARD study investigated is endoscopic therapy of postnecrotic collections. Typically, this cohort of patients is reasonably healthy and only exhibits infrequently severe complications.6 From this perspective, the mortality rate of 7.5% does not appear particularly low and the two patients who died during surgery are to be taken seriously. In addition, the authors noted that 22 patients were withdrawn from the current report due to a previous publication, but that study included only 11 patients with acute pancreatitis.5

From our perspective, there are two main lessons that can be learnt from the GEPARD study. First, the technique of transluminal endoscopic necrosectomy can be performed relatively safely in a subset of patients with (peri)pancreatic necrosis or collections. However, in some patients severe complications may be induced by endotherapy with a potential lethal result owing to bleeding or perforations. It therefore seems imperative to gain more experience with this technique through an interdisciplinary approach including gastroenterologists and surgeons. In this context, agreement should be reached regarding the necessity and timing of an invasive intervention, both of which seem debatable in the GEPARD study. Second, from the scientific point of view, the GEPARD study demonstrates the need for a precise description of patient characteristics including (peri)pancreatic findings. A clear nomenclature is necessary to allow a precise comparison of studies investigating therapeutic interventions in severe pancreatitis. The revision of the Atlanta criteria seems essential.

We surgeons have learnt our lessons with this disease in recent years and have stepped back from early and radical interventions. It seems that the use of endoscopic necrosectomy also needs to be applied with caution, ideally in an interdisciplinary approach and within clinical trials.

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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. Through 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.7 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.8 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.9 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 practitioner, 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-naïve 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 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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Metabolic syndrome and chronic hepatitis B: is the evidence enough?

We appreciated the interest and comment of Li and colleagues on our recent publication.1 We would like to acknowledge the results from the Taiwanese longitudinal studies concerning the effect of fatty liver, obesity and type 2 diabetes on the risk of liver cirrhosis and hepatocellular carcinoma (HCC) in chronic hepatitis B (CHB) patients. This evidence echoed the conclusion of the present study that metabolic syndrome increased the risk of liver cirrhosis in CHB.

In the present cross-sectional study, the causal relationship between metabolic syndrome and liver cirrhosis could not be established as patients were assessed for once at a snapshot. We believed our prospective recruitment and large sample size could partly compensate the potential bias of disease fluctuation in CHB patients. Nonetheless, longitudinal studies on the natural history of CHB could hardly be conducted nowadays, as patients with advanced liver fibrosis would be treated with antiviral therapy.

Although patients were referred from all primary care and hospital clinics in Hong Kong in our patient cohort, a significant proportion of these patients were followed up in our Hepatology clinics. Our clinic received referral from both community-based screening programmes of asymptomatic subjects and family doctors.7 Furthermore, we had no experience of elastography to assess liver fibrosis can avoid the limitation of deep-seated focus of infection, most likely related to the pacemaker lead, was strongly suspected. He underwent surgical removal of the pacemaker. A new pacemaker was implanted (on the contralateral side) while he remained on antibiotics. Culture of the pacemaker wire tip grew MRSA. He was treated with a further 2 weeks of teicoplanin, made an uneventful recovery and was discharged home.

Mucosal trauma during endoscopic procedures is known to cause bacterial translocation and transient bacteraemia.1 In most patients, the bacteraemia does not pose any risk, since the organisms are rapidly removed from the bloodstream. However, as in a very few cases, endoscopy has been shown to cause endocarditis, previously published guidelines advocated prophylaxis in high-risk patients such as with prosthetic valves or those with previous history of endocarditis.2 3 These recommendations were based on individual experiences or anecdotal case reports, rather than any well-designed prospective randomised trials primarily due to the rarity of the condition.1 4 5 Recently published updated guidelines, however, have advocated a much more limited use of antibiotics.1 6 8

Pacemaker-lead-associated endocarditis is a rare but serious complication of permanent prophylaxis even for high-risk patients having an endoscopy.1 We would like to present a case which suggests that these guidelines may be underplaying the possible risk in certain patients.

We experienced a 67-year-old patient with recurrent Methicillin resistant Staphylococcus aureus (MRSA) septicaemia due to pacemaker-lead endocarditis, as a complication of a gastroscopy. He had a past medical history of coronary artery bypass grafting and permanent pacemaker implant, and was admitted with a 2-day history of melena. A day after endoscopy, he developed features of sepsis, complicated by ventricular tachyarrhythmia necessitating treatment in the intensive care unit. Blood cultures were positive for MRSA, and throat swabs revealed MRSA with sensitivities similar to those of the organisms identified on blood culture, thus localising the infection to the throat. An echocardiogram did not reveal any obvious vegetation on the valves. He was treated with vancomycin, rifampicin and gentamicin. Seven days after stopping treatment, he developed recurrence of MRSA septicaemia. He was treated with a prolonged course of teicoplanin along with fusidic acid, tazocin and oral linezolid. Further thoracic and transoesophageal echocardiograms and a white cell isotope scan failed to identify the source of infection. After being symptom-free for 10 days, he was discharged, to be admitted again with recurrence of septicaemia. He was transferred to the nearest tertiary-care unit, where in view of the recurrence of septicaemia despite such prolonged courses of antibiotics, an unidentified deep-seated focus of infection, most likely related to the pacemaker lead, was strongly suspected. He underwent surgical removal of the pacemaker. A new pacemaker was implanted (on the contralateral side) while he remained on antibiotics. Culture of the pacemaker wire tip grew MRSA. He was treated with a further 2 weeks of teicoplanin, made an uneventful recovery and was discharged home.

Recurrent methicillin-resistant Staphylococcus aureus septicaemia and pacemaker-lead-associated endocarditis following diagnostic gastroscopy

We read with interest the recent guidelines for antibiotic prophylaxis produced by the British Society of Gastroenterology which do not support the routine use of antibiotic