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

Debabrata Majumdar, Jyothi G Rao, Kapil Kapur, et al.

*Gut* 2010 59: 277-278
doi: 10.1136/gut.2009.188268

Updated information and services can be found at:
http://gut.bmj.com/content/59/2/277.2.full.html

These include:

**References**
This article cites 9 articles, 4 of which can be accessed free at:
http://gut.bmj.com/content/59/2/277.2.full.html#ref-list-1

**Email alerting service**
Receive free email alerts when new articles cite this article. Sign up in the box at the top right corner of the online article.

Notes

To order reprints of this article go to:
http://gut.bmj.com/cgi/reprintform

To subscribe to *Gut* go to:
http://gut.bmj.com/subscriptions
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.2 Furthermore, transient elastography to assess liver fibrosis can avoid the limitation of invasiveness by liver biopsy. Therefore, we managed to recruit a reasonable number of patients with inactive liver disease. In our study, 59% of patients had hepatitis B virus DNA lower than 10,000 copies/ml, and 66% had normal alanine aminotransferase levels.

There are many definitions of metabolic syndrome with slight differences in their criteria. We used the International Diabetes Federation criteria, as ethnic-specific definitions of central obesity were adopted.3 IDF criteria were also the most commonly used definition of metabolic syndrome in the Asian-Pacific region.4

We agreed that central obesity might increase the difficulty for the use of transient elastography, particularly in the White population. In our study, 91% of patients with central obesity have reliable liver stiffness measurement.5 Although central obesity reduced the success rate of transient elastography, it was still acceptable in the Chinese population. The development of a XL probe may be a solution to the very obese patients. Based on our previous validation studies, it seemed that central obesity did not affect the accuracy of liver fibrosis assessment by transient elastography.6 7

Henry Lik-Yuen Chan, Grace Lai-Hung Wong, Vincent Wai-Sun Wong
Institute of Digestive Disease and Department of Medicine and Therapeutics, The Chinese University of Hong Kong; Hong Kong SAR, China
Correspondence to Dr Henry Lik-Yuen Chan, Department of Medicine and Therapeutics, 9/F Prince of Wales Hospital, 30-32 Ngan Shing Street, Shatin, Hong Kong, China; hlychan@cuhk.edu.hk
Competing interests None.

Provenance and peer review Not commissioned; externally peer reviewed.

Gut 2010;59:277. doi:10.1136/gut.2009.196352

REFERENCES

Recurrent methicillin-resistant Staphylococcus aureus septicemia 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 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) septicemia 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 septicemia. He was treated with a prolonged course of teicoplanin along with fusidic acid, tazocin and oral linezolid. Further trans-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 septicemia. He was transferred to the nearest tertiary-care unit, where in view of the recurrence of sepsis 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 pacing system. 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.2 However, as in a very few cases, endoscopy has been shown to cause endocarditis, previously published guidelines advocated prophylaxis in low-risk patients such as with prosthetic valves or those with previous history of endocarditis.3 4 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.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
Recurrent methicillin-resistant Staphylococcus aureus (MRSA) septicemia and pacemaker-lead-associated endocarditis following diagnostic gastroscopy

We thank Dr Majumdar and colleagues for their comments, and for their case description which at first sight appears to be unique in two respects. We are unaware of any previous case reports linking pacemaker infection to endoscopy, and our literature review for the guidelines failed to identify any previous reports of meticillin resistant Staphylococcus aureus (MRSA) bacteraemia following endoscopy. Nonetheless, we consider that the extreme rarity (or indeed uniqueness) of the proposed potential source of their patient’s pacemaker infection argues against widespread policy changes in antibiotic prophylaxis for endoscopy. Indeed, the traditional prophylactic regime of amoxicillin and gentamicin would almost certainly have failed to prevent MRSA bacteraemia and its consequences in this case.

There are also several aspects of Majumdar’s case description that are far from clear. First, it would be of interest to know whether any previous screening had been carried out prior to endoscopy. Second, the MRSA infection could have originated from microbial contamination (1) during the initial pacemaker insertion procedure (ie, the patient may have had a pacemaker infection at presentation); (2) following any intravenous cannulation or urinary catheterisation prior to endoscopy; or (3) arising from mucosal damage at the site of gastrointestinal bleeding at the time of this presentation (we are not given the findings at gastroscopy). The commonest factor predisposing to MRSA bacteraemia is the presence of intravascular catheters, and it should also therefore be confirmed that none of the blood cultures from their patient had been drawn from intravenous catheters. All of these are potential sources of the MRSA infection, and they need to be considered in any such case.

Miles C Allison, Jonathan A T Sandoe, Thomas S J Elliott

1Royal Gwent Hospital, Newport, UK; 2Leeds General Infirmary, Leeds, UK; 3University Hospital, Birmingham, UK

Is visceral fat accumulation really an independent risk factor for hepatocellular carcinoma recurrence after curative treatment in patients with suspected NASH?

I read with interest the article demonstrating that visceral fat accumulation (VFA) is an independent risk factor for hepatocellular carcinoma (HCC) recurrence after curative treatment in patients with suspected non-alcoholic steatohepatitis (NASH) by Okhi et al published in Gut.1 The authors assessed the recurrence of HCC ascribed to NASH in patients as an aetiological factor treated with percutaneous radiofrequency ablation (RFA). The diagnosis of HCC and recurrent HCC was based mostly on typical findings on CT. The accuracy was questionable. Multivariate analysis revealed that high VFA is an independent risk factor for recurrence of HCC. However, if we examine the baseline characteristics of both groups, patients belonging to the group with high VFA had a larger tumour size than controls—that is 3.2 cm vs. 2.7 cm. Also, a higher frequency of multinodular and cirrhosis was noted in patients with high VFA as compared with controls. Though these factors were not shown by the current study to be independent factors for HCC recurrence by multivariate analysis, previous studies have already disclosed that tumour size >2.3 cm, tumour stage, presence of vascular invasion and a multinodular tumour were associated with recurrence of HCC after RFA.2,3 The proportion of patients with tumour size >2.3 cm, the presence of vascular invasion and tumour stage in both groups were not shown in the current study. Local tumour progression was usually related to the size of the tumour and was noted only in two patients with high VFA but not in controls. If these two cases were excluded, the difference in recurrence of