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

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pacemaker implantation. It can rarely result from a haematogenous seeding during transient bacteraemia. The condition, which is difficult to diagnose due to its rarity, should be suspected in patients with a pacemaker who present with features of sepsis in the absence of any other obvious source.

This is the first reported case of pacemaker-lead endocarditis due to MRSA occurring after a diagnostic gastroscopy. It is important because it identifies two areas where we should be vigilant in our endoscopic practice. First, with the current guidelines suggesting a conservative use of antibiotics, it is possible we might encounter more cases of endocarditis. It is imperative that adequate surveillance and reporting of cases be maintained to build a good database to help produce evidence-based guidelines in future as advocated by the recent British guidelines. Second, this case highlights the importance of eradicating any obvious source of infection in the body. It might be important to screen and eradicate high-risk patients for MRSA carriage in the throat prior to upper GI endoscopy, particularly if further cases are reported.

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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 MRSA 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.

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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

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HCC between the groups would not be so obvious.

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Authors’ response

Professor Gin has questioned the validity of our findings that visceral fat accumulation (VFA) is an independent risk factor for recurrence of non-B, non-C, non-alcoholic hepatocellular carcinoma (HCC) after radiofrequency ablation (RFA), suggesting that the results might have been confounded by the difference in tumour size and other factors. First, we would like to clear up a couple of misunderstandings. The two cases of local tumour progression in the high VFA group were not counted as events but censored, and did not favour the results. Vascular invasion, as detected by ultrasonography or CT, was a contraindication for RFA and was found in none of the studied patients.

Secondly, regarding the tumour size, we agree that small HCC, say <20 mm in diameter, has a substantially low risk of recurrence. However, we do not think the risk of recurrence is much different between lesions of 32 mm and 27 mm. We previously reported the multivariate adjusted hazard ratio (HR) for recurrence to be 1.030 (95% CI 1.006 to 1.054, p = 0.014) per 1 cm increase in size—that is, HR = 1.015 for a 5 mm increase.1 The number of events was small in the present study, and admittedly we could not have enumerated every risk factor of recurrence. Lastly, while VFA was found to be associated with the risk of metachronous hepatocarcinogenesis, it is not known whether the relationship is causal. We will need a prospective interventional study to elucidate this issue, which we think is worth carrying out.

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