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New diagnostic approach to diagnosis of achalasia after recent Chicago classification

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We read with great interest the recently published article by Müller [1]. The author aimed to explain the importance of high-resolution manometry (HRM) on achalasia diagnosis and management. She explained conventional manometry (CM) and HRM criteria for diagnosis of achalasia. We thank Muller for this valuable study, but we think that there are some controversies needed to be clarified.

First, the author mentioned that additional effort is needed for esophageal body motility evaluation by positioning of pressure sensors in the body, instead of evaluating when positioning on lower esophageal sphincter (LES). CM contains 4-8 pressure sensors. In order to assess the LES and the esophageal body in one investigation, it is better to use a catheter with 4 or more transducers in the esophagus. For example, with 8-sensor CM when the lower sensors (5^{th}-8^{th}) are on LES high-pressure zone, the other 4 sensors (1^{st}-4^{th}) are on the esophageal body 5, 10, 15, and 20 cm above of the LES. Thus, we think that these sensors are adequate to evaluate esophageal motility, especially in the lower two-thirds of esophageal body [2].

Second, the author also mentioned that for diagnosis of achalasia with HRM, mean integrated relaxation pressure (IRP) must be elevated from upper limit of normal (>15 mmHg). But recently, it has been indicated by the International HRM Working Group that it is better to use median IRP rather than mean IRP at the diagnosis of achalasia [3].

Third, the author clearly defined achalasia subgroups and esophagogastric junction (EGJ) outflow obstruction, but the importance of EGJ outflow obstruction should be analyzed in detail in this study [1]. EGJ outflow obstruction is described by an elevated median IRP with some instances of intact or weak peristalsis, which do not meet achalasia criteria. EGJ outflow obstruction may be an achalasia variant, but it also has several potential etiologies including esophageal stiffness as a consequence of an infiltrative disease or cancer, or of vascular obstruction of the distal esophagus [3]. Thus, patients with this diagnosis should be further evaluated by endoscopic ultrasound and CT to clarify the etiology of EGJ obstruction.

In conclusion, although CM still has its place in the diagnosis of achalasia, we think that the exact diagnosis of achalasia has to be done with HRM because of the importance of the diagnosis of EGJ outflow obstruction as described above.

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Author's reply

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Thank you for the opportunity to allow us to address the comments and concerns raised by Sakin *et al* [1]. I appreciate the time they took to read and voice their concern on the present review article.

I understand and share the concerns of Sakin *et al* [1] that with the right conventional manometry catheter (8 pressure sensors) a positioning of pressure sensors in the esophageal body to evaluate the body motility is not necessary. However, in this review article the principle of the procedure as it is used in our clinic was described. Certainly, there are variations depending on the manometry catheter (number of pressure sensors) used which can increase the effort in the measurement of the body motility.

Sakin *et al* [1] pointed out that for the diagnosis of achalasia with high-resolution manometry (HRM) the measurement of the median integrated relaxation pressure (IRP) rather than a mean IRP was recently recommended by the International HRM Working Group [2]. At the time of writing, the mentioned recommendation was not published yet, and, to my knowledge, most of the used HRM systems still calculate the mean IRP, which should be changed in the future. I am grateful for the note because it highlights the importance of integrating such new recommendations on a rapidly changing subject.

Furthermore, Sakin *et al* [1] emphasize the fact that esophagogastric junction outflow obstruction may be an achalasia variant, but also has several other potential etiologies including esophageal stiffness as a result of infiltrative disease

or cancer, as mentioned in the part 'Differential diagnosis of abnormal lower esophageal sphincter relaxation' [3].

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Risk factors and antibiotic prophylaxis of cellulitis in cirrhosis

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We read the article by Hamza *et al* with great interest [1]. The authors have investigated the risk factors and whether antibiotic prophylaxis helps prevent recurrence of cellulitis in cirrhosis . We agree with the authors that bacterial infections are frequent in cirrhotic patients because of their defective defense mechanisms and that these infections precipitate decompensation of cirrhosis. Of note, an association between high MELD for end-stage liver disease score and hepatic encephalopathy with cellulitis are expected as mentioned in phase 1 results of the study.

However, since the most common infection in advanced cirrhosis is spontaneous bacterial peritonitis (SBP), antibiotics for selective intestinal decontamination, such as quinolones, are often prescribed as prophylaxis against SBP recurrence [2]. The effect of widespread norfloxacin use on the epidemiology of severe infections in cirrhotic patients is poorly known. A 5-year retrospective study evaluated the effect of long-term administration of norfloxacin on the epidemiology of severe hospital-acquired infections and showed that long-term norfloxacin administration resulted in a sharp increase in staphylococcal SBP and bacteremia, while the prevalence of *Enterobacteriaceae* and *streptococci* fell and did not change, respectively [3].

Although gram-negative bacteria-induced cellulitis has been reported in cirrhotic patients [4], gram-positive bacteria are still the predominant organism isolated from cellulitis [5]. Recommended use of broad-spectrum antibiotics as prophylactic treatment of such patients may lead to the emergence of gram-positive pathogens rather than preventation of skin infections.

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Authors' reply

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We appreciate the comments of Dr. Basyigit on our recent study showing that antibiotic prophylaxis in cirrhotic patients can reduce recurrence of cellulitis mainly caused by gram-negative bacteria. Dr. Basyigit's concern on the increased gram-negative cellulitis found in our cirrhotic patients is based on a recent study that reported gram-positive bacteria as a common cause of cellulitis in

the general population, and not particularly in cirrhotics. To support our findings, relative literature has already been cited [1,2]. In a position statement, based on EASL special conference 2013, the role of bacterial translocation as cause for infection in cirrhotic patients has clearly been pointed out [3]. Dr. Basyigit posed the possibility that drug-resistant bacteria might emerge using antibiotic prophylaxis. Though it might be true, antibiotic prophylaxis is still being used to prevent conditions such as spontaneous bacterial peritonitis thereby reducing mortality. Finally, Dr. Basyigit claimed that antibiotic prophylaxis will lead to resistant microorganisms rather than prevention from skin infection. We strongly oppose this view because bacterial infection increases 3.75-fold the mortality of patients with decompensated cirrhosis, reaching a rate of 30% at 1 month and 63% at 1 year [3]. If we can prevent recurrence of bacterial cellulitis in cirrhotic patients as we have shown in our study, it might help reduce mortality. However, since we have not examined the benefit from the prevention of cellulitis recurrence on the mortality of cirrhotic patients, further studies are warranted to elucidate this issue.

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