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Response to Riegler *et al.*

Ausilia Grigolon, MD¹ and Roberto Penagini¹

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To the Editor: We thank Dr Riegler *et al.* (1) for their interest in our study and for their stimulating comments. They suggest that in our gastroesophageal reflux disease (GERD) patients, pH measurements were not in the fundus of the stomach within the hiatal hernia but were in the “dilated end-stage esophagus.”

Controversy still exists on how to measure the esophagogastric junction accurately and also on the prevalence and meaning of the cardiac mucosa that is interposed between the squamous epithelium and the gastric oxintic mucosa.

The theory of defining the location of the esophagogastric junction based solely on histopathology is fascinating, but it still needs further validation and study of a control population (2).

For the time being, and until a better method is developed and accepted, there is consensus in using simple endoscopic landmarks for the location of the esophagogastric junction (3). According to these landmarks, our pH capsule was in the gastric fundus both in healthy subjects and in GERD patients with and without hiatal hernia.

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The Serological Profile of the Autoimmune Hepatitis/Primary Biliary Cirrhosis Overlap Syndrome

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To the Editor: We read with great interest the study report of Muratori *et al.* (1), regarding the serological profile of autoimmune hepatitis and primary biliary cirrhosis overlap syndrome (AIH/PBS OS). They screened anti-double-stranded DNA (dsDNA) antibodies in serum samples from 15 patients with AIH/PBS OS, and 9 of them were found to be positive (60%).

We performed a retrospective analysis of autoimmune liver disease patient files during the period 1998–2009. A total of 12 patients had been diagnosed as AIH/PBC OS according to the criteria of Chazouilleres *et al.* (2). During or before the diagnosis of AIH/PBS OS, antimitochondrial antibodies (AMAs) were positive in all patients, but serum anti-dsDNA antibodies were investigated in only four patients, two of whom were positive. After Muratori *et al.*'s report, we collected serum samples from 12 patients, and 7 of them were positive (58.3%). Furthermore, the anti-dsDNA antibody status of the first four patients did not change after appropriate therapy (three of them received combination therapy with immunosuppressive and ursodeoxycholic acid (UDCA) and one received only UDCA).

Anti-dsDNA antibodies are highly specific for systemic lupus erythematosus (SLE) and present in nearly 70% of patients. In some but not all SLE patients, the presence of anti-dsDNA antibodies can reflect disease activity (3). The importance of anti-dsDNA antibodies is well established in SLE patients, but their relevance in AIH/PBS OS patients is not clear yet.

In conclusion, the results of the study performed by Muratori *et al.* are similar to our findings. Anti-dsDNA antibodies were positive in approximately 60% of AIH/PBC OS patients in both studies, suggesting that their presence may have both diagnostic and clinical significance. The concomitant presence of anti-dsDNA and AMA suggests that these tests might be candidates for specific serological tests for the diagnosis of AIH/PBS OS.

CONFLICT OF INTEREST

The authors declare no conflict of interest.

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Response to Efe *et al.*

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To the Editor: The data from Efe *et al.* (1) about the presence of anti-double-stranded DNA (dsDNA) in their patients with primary biliary cirrhosis/autoimmune hepatitis overlap syndrome (PBC/AIH OS) represent an important confirmation of our previous results.