pylori infected compared to the 39 uninfected patients (61.3% vs. 12.8%, P<0.001; OR 10.767, 95% CI 3.293-35.205). Since the pathogenesis of PGH is not completely disclosed - and the role of *H. pylori* in such a field is still controversial - any new information is welcome. Unfortunately, some potential drawbacks occur in this study.

Firstly, at least two specific studies demonstrated that both sensitivity (78.6-85.4%) and specificity (38.4-52%) of serology for *H. pylori* diagnosis are disappointingly low in cirrhotics, with values distinctly lower than controls [2,3]. Therefore, serology is particularly inaccurate for H. pylori infection diagnosis in cirrhotics, preventing a reliable data interpretation. In addition, the overall *H. pylori* seroprevalence detected in this study (only 35.7% on 140 cirrhotics with a mean age of >50 years) appears astonishingly low when considering that the study was performed in India where *H. pylori* prevalence is extremely high in the general population [4]. The evidence that serology in cirrhotics significantly overestimates H. pylori infection, as pointed out in several studies [5], further questions the accuracy of such an unexpected observation.

Secondly, this study found a significantly higher H. pylori seroprevalence rate in cirrhotics with severe PHG (19/24, 79.2%) compared to those with mild PHG (12/46, 26.1%). Consequently, it was concluded that H. pylori infection is not only associated with PHG in cirrhotics, but also with more severe PHG [1]. However, H. pylori prevalence in literature was found to range widely from 23% to 79% and from 22% to 81% in cirrhotics with mild and severe PHG, respectively [5]. Therefore, this finding is not conclusive, and a selection bias cannot be definitely ruled out.

Thirdly and disappointingly, although the authors noted that as many as 18 (75%) cirrhotics with severe PHG were in Child-Pugh class C, a multivariate analysis was not performed to examine whether H. pylori actually plays an independent role. Indeed, a significant correlation between Child-Pugh class C and PHG is widely documented in the literature [6]. Therefore, the inclusion of as many as 30 Child-Pugh class C (as well as 41 cirrhotics with ascites) in the control group without PHG would suggest a remarkable selection of patients.

Based on all these considerations, the data of this study should be considered with caution, and further prospective studies, in which H. pylori infection is correctly diagnosed with either histology or ¹³C urea breath test [3,7], are needed.

Helicobacter pylori and portal hypertensive gastropathy: any new information?

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We read with great interest the study by Sathar et al focused on the role of Helicobacter pylori (H. pylori) infection on portal hypertensive gastropathy (PHG) in cirrhotics [1]. Briefly, this retrospective study compared H. pylori seroprevalence between 70 cirrhotics with PHG (cases) and 70 matched cirrhotics without PHG (controls). The main results were that: a) the prevalence of infection was higher in cases than controls (44.3% vs. 27.1%, P=0.034; OR 2.134, 95% CI 1.052-4.327), and b) the prevalence of severe PHG was higher in the 31 H.

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