Helicobacter pylori infection and liver cirrhosis: possible association with hepatic encephalopathy and/or post-hepatic encephalopathy cognitive impairment in patients with portal hypertension

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In their retrospective series, Sathar et al [1] concluded that there is significant association between Helicobacter pylori infection (Hp-I) and portal hypertensive gastropathy (PHG) in cirrhotic patients, also related to PHG severity, thereby necessitating Hp eradication.

Apart from PHG, hepatic encephalopathy (HE) is another complication of portal hypertension that remains a major cause of morbidity in cirrhotic patients [2]. HE encompasses a spectrum of neuropsychiatric disorders related to liver failure and the mechanisms responsible for the neurological alterations in HE begin to emerge [3].

Hepatitis B (HBV) and C (HCV) infections are among the commonest causes of liver cirrhosis worldwide; Hp-I is strongly associated with HBV- and HCV-related cirrhosis in Europe; Hp-I is more common in cirrhotic patients with HE than in those without [4]; HE is not a fully reversible condition and the mechanism behind the lack of reversibility of the neurocognitive status despite the resolution of mental status changes is unclear [5]; and cognitive dysfunction is a factor associated with falls in cirrhotic patients, though further studies are warranted to address the mechanisms implicated in this predisposition and to design preventive strategies [5]. In this regard, Hp-I has been frequently detected in cognitive impairment and Alzheimer’s disease (AD) [6] and we found that Hp eradication may positively influence AD manifestations at five-year clinical endpoints [7], thereby supporting a role for this common infection in the pathobiology of the disease.

Hp may be involved in the pathophysiology of both HE and post-HE persistent cognitive impairment by several mechanisms [7], including the release of proinflammatory/ vasoactive substances, involved, through blood-brain-barrier disruption, in a number of vascular disorders including AD, which can lead to long-term neurologic deficits [4,5]. It is therefore important to know if the authors have considered the association between Hp-I, HE and/or post-HE cognitive impairment in their cirrhotic patients.

References


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