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.

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Stent placement for delayed conduit obstruction at hiatus after esophagectomy

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We report a case of a 62-year-old male who presented with shortness of breath after having undergone a modified McKeown esophagectomy 6 months after induction chemotherapy for cT3N1 adenocarcinoma of the distal esophagus. A chest computed tomography demonstrated a dilated gastric conduit with evidence of hiatal obstruction and aspiration pneumonia (Fig. 1A). Patient was subsequently intubated for respiratory distress, but eventually extubated three days later. Barium esophagram revealed a dilated gastric conduit with poor emptying (Fig. 1B). Patient underwent endoscopic evaluation revealing a dilated conduit with extensive food and gastric debris. The pylorus was found to be widely patent. However at the level of the hiatus just proximal to the pylorus, there was evidence of obstruction with passage of gastric contents impeded by redundant gastric conduit that formed a shelf at the hiatus with a resulting valve-like effect. A 10 cm × 18 mm Alimaxx™ stent was then placed across the area obstructed by the flap to just proximal to the pylorus (Fig. 1C). The area of obstruction was effectively opened with clearance of gastric content immediately evident. The patient subsequently underwent a barium swallow the succeeding day showing passage of contrast into the duodenum across the hiatus (Fig. 1D). The patient was started on clears and then was eventually discharged tolerating a soft solid diet. The patient remains free of obstructive symptoms for the past 12 months.

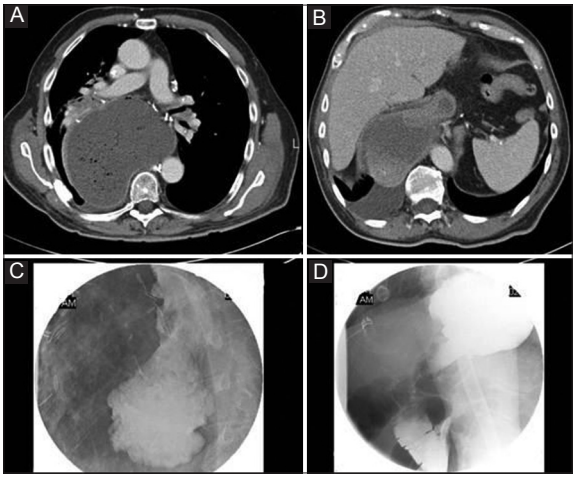

Figure 1 (A) Chest computed tomography illustrating dilated gastric conduit with obstruction at level of hiatus. (B) Barium esophagram demonstrating a dilated gastric conduit with virtually no emptying of contrast into the duodenum. (C) Endoscopic view with stent across area of obstruction at hiatus. (D) Barium esophagram now demonstrating emptying of contrast into the duodenum across the stent