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Benign gastric outlet obstruction by a large phytobezoar

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We read with interest the article of Shetty *et al* [1] on trichotillomania with gastric trichobezoar obstruction. Similarly, Zin *et al* [2] reported on okra phytobezoar-related gastrojejunostomy efferent limb obstruction.

We report a case of a 76-year-old man with a medical history of ulcer disease referred to our hospital due to recurrent vomiting, nausea and inability of food intake for the previous 48 h. On admission, his vital signs were normal (temperature: 36.5°C, blood pressure: 125/80 mmHg, heart rate: 93 bpm). Physical examination revealed mild sensitivity, distension of abdomen with splashing and presence of normal bowel movements. Laboratory tests, compared to previous ones, showed a significant increase in the hematocrit (51.8% from 46.1%) and serum urea (71 mg/dL from 47 mg/dL) implying moderate dehydration. Chest and abdominal X-ray showed a marked gastric air bubble, air-fluid levels located on small intestine and a downward shift of transverse colon (Fig. 1). Abdominal ultrasound demonstrated a stomach full of liquid content. The patient received parenteral hydration and a nasogastric tube was placed. Twelve hours later, approximately 2,500 mL had been drained. Upper gastrointestinal endoscopy revealed a phytobezoar with a diameter of 4 cm, impacted in the pyloric canal. The phytobezoar was captured with a Dormia basket and was extracted (Fig. 2). After the extraction, the antrum was inspected revealing erythema and nodular appearance of the propyloric area. Biopsies were taken to exclude malignancy. Insertion of the endoscope through the pylorus was successful, without any further findings in the duodenum. After the procedure, the patient mentioned that a few days before admission he ate an orange without proper mastication (he did not have his dentures on). The patient started oral feeding with instructions of mindful chewing. Treatment with proton pump inhibitors q.d. for one month was given. To our knowledge, he remains asymptomatic. Histology was negative.

Gastric outlet obstruction (GOO) is the clinical and pathophysiological consequence of any disease process that produces a mechanical impediment to gastric emptying. In the past, when peptic ulcer disease (PUD) was more prevalent, benign causes were the most common, however, one review shows that only 37% of patients with GOO have benign disease and the remaining patients have obstruction secondary to malignancy [3]. The leading causes of benign GOO are PUD and ingestion of corrosives [4]. Non-steroidal anti-inflammatory drugs and opium addiction are rare causes of GOO [5]. Other benign causes are gastric polyps, pyloric stenosis, congenital duodenal webs, gallstone obstruction (Bouveret syndrome), pancreatic pseudocysts and bezoars [4]. Among the various types of bezoars, the most common type is the phytobezoars, composed mainly of undigested vegetable materials [2].

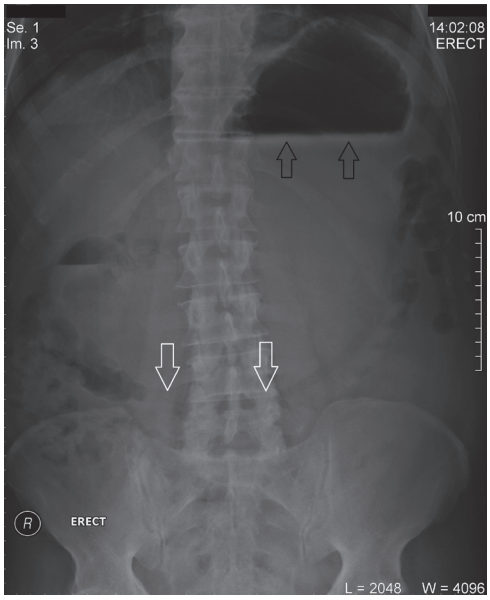

Figure 1 Abdominal X-ray of the patient which showed a marked gastric air bubble (black arrows) and a downward shift of transverse colon (white arrows)

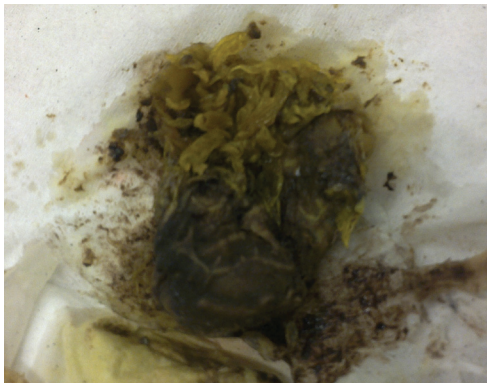

Figure 2 The orange-phytobezoar after it was extracted

In conclusion, proper mastication should always be recommended to people with dentition problems. Treatment of any underlying cause with adequate fluid intake and avoidance of a strictly fibrous diet could prevent recurrence.

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A GIST or not a GIST? That is the question

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Several reports have recently focused on stromal tumors and their acute or chronic complications [1,2]. Gastro-Intestinal Stromal Tumors (GIST) are mesenchymal neoplasms that represent a small percentage of gastric cancers (1-3%) and arise more often from the stomach (60%) [3]. Expression of c-KIT (CD 117) in the tumor cells define their character in a proportion of 95% with the remaining 5% representing c-KIT-negative GISTs associated with platelet-derived growth factor receptor-αmutations [4-6]. Imatinib, an inhibitor of the oncoprotein BCR-ABL is used as an adjuvant therapy in metastatic c-KIT metastatic GISTs and recently in primary resected GISTs and has changed their prognosis.

Desmoid tumors are well differentiated, locally aggressive fibrous neoplasms that occur in patients with familiar adenomatous polyposis at an approximately rate of 10%. Histologically their character is benign but could lead to obstruction of vital structures and organs. They tend to recur and their therapeutic management is often very difficult.

An 18-year-old male with free medical history was admitted in our department due to acute abdominal distension. Standard blood tests were normal. Abdominal ultrasound revealed an epigastric mass of 120.8x107.3 mm that repelled spleen, without a central fusion and cystic components with rich neo-vascularization. CT could not establish the origin organ of the lesion. MRI showed that the origin of the mass was from the layers of the stomach (Fig. 1). Upper GI endoscopy and colonoscopy were normal. Endoscopic ultrasound (EUS) revealed in the major arc of the stomach a sizeable hypoechogenic and in homogeneous arrangement of 122.2x108 mm in contact with the fourth sonographic layer (muscularis propria). Due to the size and pressure of the mass EUS could not determine that it originated from the stomach. The patient underwent a surgical resection of a 25x20 cm mass of 4 kg (Fig. 2). The macroscopic diagnosis was as of a GIST. However, the histology revealed a tumor inherent in the stomach with few mitoses, 1-3/50 mitoses per field, asteroid or elongated cells with fusiform nuclei with histological and immunohistochemical characters compatible with intraabdominal fibromatosis. Tumor cells penetrated the main focal muscular tunic but the supernatant submucosa and mucosa of the stomach were intact. The surgical margins as well as the omentum, mesentery lymph nodes and three omentum lymph nodes were free of tumor infiltration. Immunohistochemistry showed positivity for Vimentin. S100 protein was positive in scattered cells and β-catenin positive in isolated plasmatic and nuclear staining of cell damage. SMActin, MSActin and desmin were negative. Finally CD117 and CD34 were also negative. Because of the size of the mass EUS could not establish the origin of the tumor. On the other hand, histology revealed a giant intra-abdominal fibromatosis based on positivity of β-catenin and negativity of CD117, CD34 and although the mass was inherent with muscularis propria of the stomach the origin of the tumor could not be established.

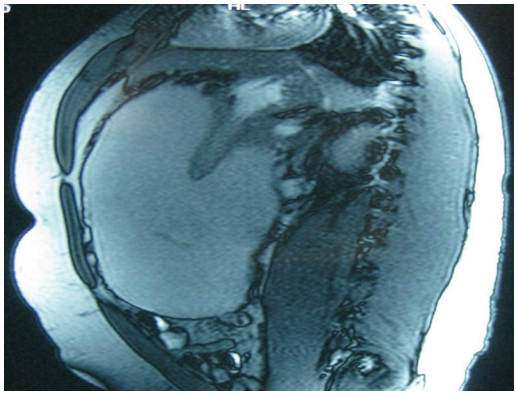

Figure 1 MRI showing the limits between the stomach and mass

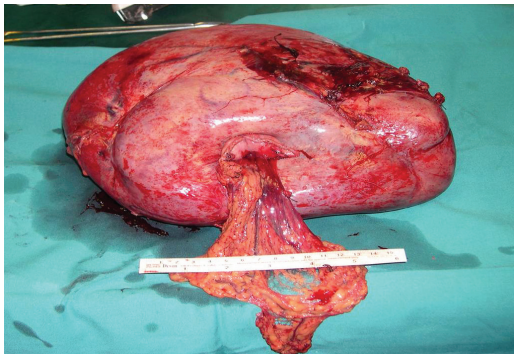

Figure 2 Postsurgical image of the abdominal mass

In vivo results such as paraclinical tests or intraoperative findings do not always correlate with *in vitro* results such as histological findings as in our case, which makes the paraphrase of Hamlet's question opportune.

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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. 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 ^{13}C urea breath test [3,7], are needed.

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Author's reply

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We thank Zullo *et al* for submitting their criticism on our study published in *Annals of Gastroenterology* [1]. A point-by-point response to their comments can be found below.

Firstly, the overall performance of commercially available serology kits that measure IgG antibodies for the diagnosis of *Helicobacter pylori* (*H. pylori*) infection showed that serology has accuracy >90% in diagnosing *H. pylori* infection. These are widely available and cheap, and particularly helpful in cirrhotic patients taking antibiotic and/or antisecretory (proton pump inhibitor, PPI) treatments which may affect culture, histology, rapid urease test, urea breath test or stool test [2]. This is relevant especially in our cirrhotic patients as most of them were on PPI treatment. Our study shows that patients with liver cirrhosis have a seroprevalence of *H. pylori* 35.7%, comparable to the Indian data reported by Batmanabane *et al* [3] in which the prevalence of *H. pylori* was 43%. The decreasing seroprevalence may be due to improved sanitation and hygiene as a result of a rapidly growing economy.

Secondly, this study demonstrates not only a significant association of *H. pylori* with portal hypertensive gastropathy (PHG) in cirrhosis but also with the severity of PHG. Being a retrospective study selection bias cannot be completely ruled out.

Thirdly, one of the major limitations of the study is the absence of histological data for diagnosis of PHG, which was diagnosed on the basis of upper gastrointestinal endoscopic appearance only, an accepted method for the diagnosis of PHG in previous studies as well [3,4]. However this might have been attributed to inclusion of 30 CHILD C cirrhotic patients as controls.

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