To the Editor,

Gastric cancer patients are first diagnosed with an unresectable tumor in up to 40% of cases (1-3). Gastric antrum obstruction (GAO) is a frequent complication and presents as nausea, vomiting, dehydration and eventually malnutrition.

Furthermore, patients with GOO with duodenal involvement often have coexistent biliary obstructions or require subsequent biliary drainage for a combined biliary obstruction (4-6).

A 19-year-old woman, presenting with a 3-month-long history of ulcer-like dyspepsia not responding to proton pump inhibitors (PPI), microcitic anemia (Hb 9 g%, MCV 73) and weight loss of 7 kg (her body weight at admission was 50 kg) was admitted on October 2011 to our surgery unit due to the occurrence of incoercible nausea and vomiting.

The patient was treated with total parenteral nutrition (TPN), PPI intravenously, and empirical antibiotic therapy. A computed tomography (CT) scan was initially performed, showing inhomogeneous parietal thickening of the ascending colon, longitudinally from the hepatic flexure to 8 cm, with coexistent thickening of the adjacent omentum and the gastric outlet, lymphadenomegaly, and ascitic fluid collection in the pelvis. An upper endoscopy was performed, showing an ulcerating and substenotic lesion surrounding the gastric outlet. At histological examination an undifferentiated gastric adenocarcinoma (G3) was diagnosed. The Gastric Outlet Obstruction Scoring (GOOS) System was 1 (4). Her2/Neu gene amplification was negative at FISH assay. Furthermore, colonoscopy was performed, showing an extrinsic compression in the proximal segment of the transverse colon, while a positron emission tomography showed enhancement of fluorodeoxyglucose in the gastric antrum, without enhancement in the transverse colon.

At abdominal laparoscopy, which the patient initially underwent to resolve the obstruction, the unresectability of the primitive lesion was confirmed, due to the invasion of the omentum, the extrinsic compression of the proximal transverse colon, and the presence of peritoneal carcinosis, as shown histologically.

Therefore, after the laparoscopic intervention the patient was discharged, with the indication of starting neoadjuvant chemotherapy. However, five days after discharge, the patient was admitted to our oncology unit due to the sudden onset of jaundice, vomiting and epigastric pain. At admission, biochemical tests showed hyperbilirubinemia (total bilirubin 5.4 mg/dL, direct bilirubin 3.6 mg/dL), elevated aminotransferases up to 20 times the upper normal limit (ULN), alkaline phosphatases (ALP) 10 x ULN, gamma-glutamyl-transferases (GGT) 8 times the ULN. The patient started TPN, PPI intravenously and empirical antibiotic therapy. Due to the persistence of vomiting and epigastric pain, with confirmation in abdominal X-ray of abdominal distension of the small bowel with air-fluid levels, a nasogastric tube was put in place, but without clinical benefit. Furthermore, a CT scan showed compression of the main hepatic duct by the mass arising from the gastric antrum (type I stricture, according to the classification by Mutignani (7), and confirmed the other findings of the previous CT.

Therefore, a self-expandable metal stent (SEMS) (Covered WallFlex, Boston Scientific) was placed in the proximal transverse colon with clinical (reduction of the abdominal pain and disappearance of vomiting) and radiological (reduction of the air-fluid levels) benefit. Endoscopic retrograde cholangio-pancreatography (ERCP) was planned to resolve the biliary obstruction, but the procedure was unsuccessful owing to the presence

An unusual presentation of unresectable gastric cancer in a young woman, treated with palliative endoscopic treatment with triple metal stents

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Received: May 22, 2013 Accepted: October 03, 2013
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of marked stenosis of the gastric antrum, because the stenosis did not allow progression of the duodenoscope; therefore another SEMS (Covered WallFlex, Boston Scientific) was put in place, through the pylorus, into the duodenum, and the day after a biliary metallic uncovered stent (NITI-ST1010, Taewoong Medical, Gomak-ri, Wolgot Myeon, Gimpo-si, Gyeonggi-do, Korea) was put in place through percutaneous transhepatic cholangiography (PTC).

We did not repeat the ERCP, after the first unsuccessful attempt, because the gastroduodenal stent was not fully expanded, and the narrow lumen of the duodenum, despite the stenting, did not allow progression of the duodenoscope, as shown at the upper gastrointestinal transit performed one day after the procedure; therefore, due to an increase in the bilirubin and cholestatic enzymes, despite medical therapy, PTC was urgently performed.

Figures 1 and 2 show a CT scan showing the simultaneous placement of the triple stent.

Figures 3 show marked gastrectasia with dilatation of the biliary tree, which is resolved after placement of the triple stent (Figure 4).
Six days after, due to normalization of bilirubin (1.5 mg%) and marked reduction in cholestatic markers (aminotransferases 5 x ULN, ALP and GGT within the normal range), the patient started palliative chemotherapy with cisplatin, and was discharged with the instruction to continue this regimen. The Gastric Outlet Obstruction Scoring System was 4.

Unfortunately, the patient died eight months after diagnosis, due to disease progression. The use of SEMS has been shown to be an effective palliative treatment in patients with unresectable malignant gastroduodenal or biliary obstruction (8), but such simultaneous obstructions complicate stent placement. In patients with such combined obstruction, biliary metal stenting is recommended as an initial step due to the difficulty in accessing the papilla through the mesh of the duodenal SEMS (5,8). Endoscopy biliary stenting, however, may be impossible in severe duodenal obstruction due to the difficulty of passing through the duodenal stricture with a duodenoscope. A percutaneous approach to biliary drainage before endoscopic duodenal metallic stenting is traditionally preferred in such cases, even though biliary re-intervention after duodenal metal stent placement may also be undertaken successfully using a percutaneous route (6), as in our patient. Our patient presented a type I biliary and duodenal obstruction, according to the classification proposed by Mutignani and coworkers (7); furthermore, in the case here reported, the placement of a colonic stent was necessary to resolve the extrinsic compression due to the primitive gastric tumor. The patient had an 8-month survival after diagnosis, with a slight improvement in the GOOS up to 5 month after placement of the triple stent.

Considering the optimal performance of stenting, in the setting of palliative care, further studies are needed to evaluate the efficacy and safety of multiple stenting, possibly with a combined percutaneous-endoscopic approach. The continued development of endoscopic procedures and devices is expected to improve the treatment of patients with combined malignant biliary, duodenal, and colonic obstruction.

Conflict of Interest: No conflict of interest was declared by the authors.

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