I can’t drink or eat !

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A 38 ys old man presented to our unit by persistant vomiting not responding to treatment (IV metclopromide and ondansteron).

On admission patient was cachectic, hypotensive but his physical examination was otherwise unremarkable.

His past history included fever 11 months ago which was diagnosed as typhoid fever with no improvement after treatment after which patient started to complain of weight loss, dyspnea and bleeding per rectum.

He seeked medical advice at Mansoura Gastroenterology Surgical Center.

Initial lab values:

HB:4 g/dl, MCV: 59,MCHC: 14, PLT: 290,
Cr:0.9 mg/dl,
Bil: 1.2 mg/dl, ALT:24 U/L, AST:8 U/L,
AFP: >1000 ng/dl, CEA: 1.2, CA19-9: 3.7 U/ml.

Abd US:

- Multiple hyperechoic focal hepatic lesions (largest 3.5 cm).
- Multiple para aortic, epigastric and porta hepatis LNs.

Abdominal CT:

- Multiple focal hepatic lesions; no significant enhancement
- Multiple enlarged LNs.
- Intraluminal mass in the antrum and pylorus.

Colonoscopy: Free.

EGD: Dilated stomach, full of food and secretions with large cauliflower antral and pyloric mass, multiple biopsies were taken.

Histopathology: Invasive GII adenocarcinoma.

Patient was diagnosed as:

"Metastatic gastric adenocarcinoma"

He received treatment in form blood transfusion and IV iron.

He was transferred to nuclear medicine for neoadjuvant therapy and received 3 cycles of chemotherapy.

Repeated Lab.:
HB: 10.6 g/dl, Hct: 33.8%, WBCs: 7.100, Plt: 325,
AFP 86 ng/dl, CEA: 37.8 ng/ml, CA19-9: 23 U/ml.

What can we offer to this patient?

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Adv Ther. 2010 Oct;27(10):691-703. doi: 10.1007/s12325-010-0061-2. Epub 2010 Aug 19.

Self-expandable metallic stents for malignant gastric outlet obstruction.

Boškoski I, Tringali A, Familiari P, Mutignani M, Costamagna G.

Catholic University, Rome, Italy.

Abstract

Duodenal self-expandable metal stents (SEMS) are designed for palliation and prompt relief of malignant gastric outlet obstruction (GOO). This mini-invasive endoscopic treatment is preferable to surgery due to its lower morbidity and mortality, shorter hospitalization, and earlier symptoms relief; furthermore endoscopic enteral stenting can be performed under conscious sedation, reducing the risk of general anesthesia in these already fragile patients. The stent placement technique is well established and should be performed in referral centers with adequate materials and equipment. Duodenal stents can be covered and uncovered. Nitinol stents have almost replaced other materials, being more flexible with a satisfactory axial and radial force. Common duodenal SEMS-related complications are recurrence of GOO symptoms due to stent clogging (tissue ingrowth/overgrowth and food impaction) and stent migration. These complications can be usually managed endoscopically. Perforation and bleeding are the most severe, but rare, complications. After stent placement, malignant GOO patients usually have improvement of the GOO symptoms with good resumption of fluids and solids. Choosing the most appropriate type of stent is arduous and should be done mainly in relation to the morphological aspects of the stricture. Endoscopic duodenal SEMS placement is indicated in symptomatic GOO patients suffering from unresectable malignancy or those inoperable due to advanced age or comorbidities. The absence of peritoneal carcinomatosis and multiple small bowel strictures is a key point for the clinical success of duodenal SEMS. Almost all symptomatic malignant GOO patients are candidates for the duodenal SEMS procedure; resolution of GOO, avoiding the need for a permanent naso-gastric or percutaneous endoscopic gastrostomy tube, significantly improves the patients' quality of life and dignity, even if life expectancy is short. Endoscopic duodenal SEMS insertion, after an adequate training, is a reproducible, simple, safe, and cost-effective procedure.

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Gastric Adenocarcinoma

Review and Considerations for Future Directions

An important development in the epidemiology of gastric carcinoma has been the recognition of the association with *Helicobacter pylori* infection.[19] Three independent studies reported a significantly increased risk in subjects who were demonstrated to have had *H. pylori* infection 10 or more years before the cancer diagnosis.[28–30] A follow-up meta-analysis of 42 observational studies carried out by Eslick et al[31] showed a significant relationship between *H. pylori* and gastric cancer (odds ratio [OR], 2.04; CI, 1.69–2.45). *H. pylori* has subsequently been shown to induce changes in the gastric mucosa and the gastric flora predisposing to the development of carcinoma in humans.[19] Furthermore, *H. pylori* is capable of adhering to the Lewis blood group antigen, and may be an important factor facilitating chronic infection and the subsequent increased cancer risk observed in patients with blood group A phenotype.[19]

فَإِنَّهُمْ عَدُوٌّ لِي

إِلاَّ رَبَّ الْعَالَمِينَ

الَّذِي خَلَقَنِي فَهُوَ يَهْدِينِ

وَالَّذِي هُوَ يُطْعِمُنِي وَيَسْقِينِ

وَإِذَا مَرِضْتُ فَهُوَ يَشْفِينِ

وَالَّذِي يُمِيتُنِي ثُمَّ يُحْيِينِ

وَالَّذِي أَطْمَعُ أَن يَغْفِرَ لِي خَطِيئَتِي

يَوْمَ الدِّينِ