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CASE REPORT

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Giant exophytic gastrointestinal stromal tumor (GIST) causing gastric outlet obstruction: case report and review of literature

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Abstract

Background: Gastrointestinal stromal tumors (GISTs) are rare mesenchymal tumors, mostly located within the stomach. About 30% of GISTs are incidentally diagnosed and as they become symptomatic may be associated with bleeding, bowel obstruction or spontaneous rupture. *Case presentation*: We present the case of a middle-aged patient diagnosed with a giant gastric GIST, which presented for intermittent gastric outlet obstruction symptoms, and emphasize the major imagistic, histopathological, and therapeutic challenges that may be encountered. There are only several cases of gastric exophytic gastric GIST provoking intermittent gastric outlet obstruction. Tumor resection should be adapted to every patient's status, focused on en bloc extraction, with preservation of invaded organs as much as possible.

Keywords: gastric gastrointestinal stromal tumor, immunohistochemistry, surgery.

Introduction

Gastrointestinal stromal tumors (GISTs) are rare mesenchymal tumors (0.1% to 3% of all malignant GI tumors) that originate within the interstitial Cajal cells [1, 2]. With an estimated incidence of 10 to 20 patients per million diagnosed every year their most common location is the stomach (>60%) followed by small intestine (25–30%) and colon (5–15%) [3–5]. About 30% of GISTs are incidentally diagnosed because most of them are asymptomatic [6]. When they reach a significant size, abdominal GISTs may be associated with complications, such as bleeding, bowel obstruction or spontaneous rupture.

While the curative option remains surgical resection, therapeutic management should be followed by neoadjuvant Imatinib to avoid possible relapse and to ensure a long-term disease-free progression. Histopathological (HP) examination along with immunohistochemistry (IHC) and molecular tests are necessary for differential diagnosis of mesenchymal tumors. More than 95% of GISTs harbor *c-Kit* mutation, which can be assessed by positive IHC staining for cluster of differentiation (CD)117. In addition, GISTs are usually positive for CD34 and discovered on GIST1 (DOG1) [7]. They are frequently negative for S100, smooth muscle actin (SMA), vimentin and desmin, which are more specific to other stromal tumors like leiomyoma, melanoma, rhabdomyosarcoma or leiomyosarcoma [8]. Risk stratification for aggressive behavior of primary GISTs is established by tumor size, high mitotic rate and high Ki67 index [9].

Aim

We report the case of a middle-aged patient diagnosed with giant gastric GIST, which presented for intermittent gastric outlet obstruction symptoms and emphasize the major diagnostic and therapeutic challenges that may be encountered.

Case presentation

A 47-year-old man, with a history of appendicectomy as a child and a severe trauma of the left hemithorax, left hypochondrium and left flank three years from the current presentation was admitted in the Department of Gastroenterology for nausea, repeated vomiting, epigastric pain, and intermittent constipation for over a month. He also had acid regurgitation and pyrosis, as well as loss of appetite and a significant weight loss in the past six months. Physical examination revealed a firm, palpable abdominal mass in the left hypochondrium and left flank.

Laboratory tests showed low iron serum level (46 μg/dL) and leukocytosis. Upper endoscopy described a deformed stomach with an extrinsic compression especially on the antrum, which made difficult the passage through the pylorus. There was no significant distention of the gastric body on insufflation and no mucosal abnormality.

We performed an abdominal ultrasound (US) and highlighted a heterogeneous mass with tissular and cystic components, extended from left hepatic lobe (LHL) to the spleen's hilum. Contrast-enhanced ultrasonography (CEUS)

pointed out that the tissular component of the tumor was enhanced in arterial phase and presented a wash-out phenomenon in the late phase. The cystic and necrotic components were unenhanced (Figure 1, A and B).

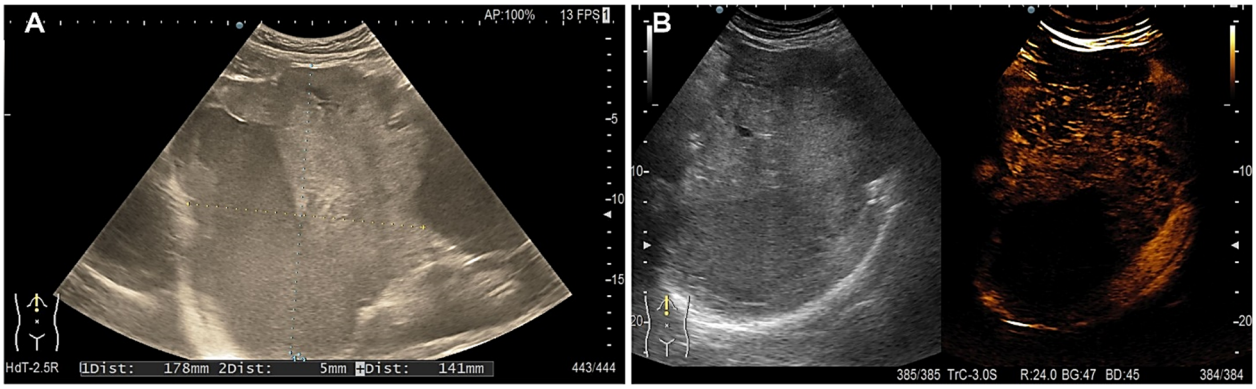

Figure 1 – ***(A) US image of the tumor as a heterogeneous mass with tissular and cystic component extended from left liver lobe to the spleen's hilum; (B) CEUS showed unenhanced areas represented by the cystic and necrotic components, while the tissular part of the tumor was enhanced in the arterial phase with secondary wash out in the late phase. CEUS: Contrast-enhanced ultrasonography; US: Ultrasound.***

Since we could not define the point of origin, we scheduled a contrast-enhanced abdominal computed tomography (CT). The CT scan described a heterogeneous tumor of 17×15×14 cm containing numerous necrosis and cystic areas with origin in the fundus and greater curvature of the stomach, with extraluminal development (Figure 2). The tumor was causing a mass effect on the stomach, spleen, splenic vein, and enveloping the body and pancreatic tail. Thus, we suspected it was a GIST with necrotic areas.

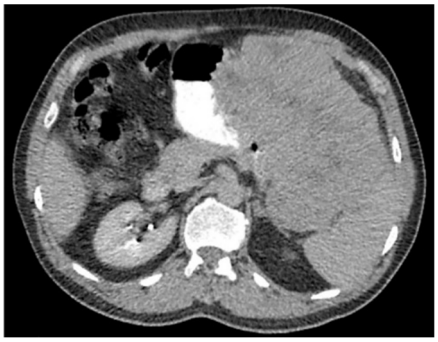

Figure 2 – ***Abdominal CT scan showed a large mass of 17×15×14 cm, with numerous necrosis and cystic areas, causing a mass effect on the spleen. Also, it suggested that the point of origin might be the stomach wall. CT: Computed tomography.***

Due to tumor size and gastric outlet obstruction symptoms, we proposed the patient for surgery. A xypho-umbilical approach was chosen, and the peritoneal cavity was opened. The giant tumor occupied most of the upper abdominal cavity with adherence to the gastric wall. After the gastro-colic ligament was sectioned, tumor appurtenance of the great gastric wall, and infiltration of the tail of the pancreas and splenic pedicle were confirmed. No hepatic or peritoneal metastases were found, and the rest of the abdominal organs seemed macroscopically normal. En bloc surgical removal of the tumor was decided including with adjacent lymph nodes, spleen, and pancreas tail. The pancreas tail was sectioned 5 cm proximal from the splenic pedicle. The tumor block included the greater curvature of the stomach, which was consecutively sealed with a linear stapler.

Macroscopically, the resected piece measured 23×17×10 cm, while the tumor had 22×10 cm (Figure 3, A and B) with many cystic and necrotic areas. For HP examination, tumor fragments were embedded in 10% formalin for 24 hours and included in paraffin according to current protocols. Classical staining with Hematoxylin–Eosin (HE) described similar characteristics of a mesenchymal tumor, with spindle-shaped cells with high mitotic rate [over 5 mitoses/50 high-power fields (HPFs)], with an 86% risk of progression (PT4Nx).

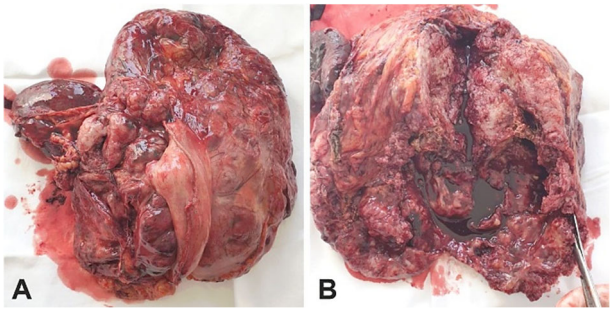

Figure 3 – ***(A and B) Macroscopic image of the resection specimen (22×10 cm) with hemorrhage spots, cystic and necrotic areas.***

In the thickness of the muscular tunic of the gastric wall, a partially encapsulated tumor proliferation was noted, with a pseudonodular appearance, composed of fusiform cells, with hypertrophic nuclei, with unevenly arranged chromatin with karyorrhexes and mitoses. The cells were arranged in a fasciculate pattern, with myxoid, hemorrhage, and necrosis areas (Figure 4, A–D). Neoplastic proliferation invaded only the submucosa, did not affect the gastric mucosa, and did not invade adjacent splenic or pancreatic parenchyma. The mitotic rate was high, >5 mitoses/50 HPFs. Free margins (R0), no evidence of perineural invasion, lymph node, or peritoneal metastasis was noted.

For IHC, we used an antibody algorithm by the labeled Streptavidin–Biotin and horseradish peroxidase (HRP) methods (Table 1). IHC staining revealed that tumor cells were strongly diffuse positive for CD117/c-kit (Figure 5, A and B), CD34 (Figure 6, A and B), and DOG1 (Figure 7). SMA and neuron-specific enolase (NSE) were variable focal positives in tumor proliferation (Figures 8 and 9). The Ki67 proliferation rate was positive in more than 5% of the tumor cells in 50 HPFs (Figure 10) and the S100 protein was negative.

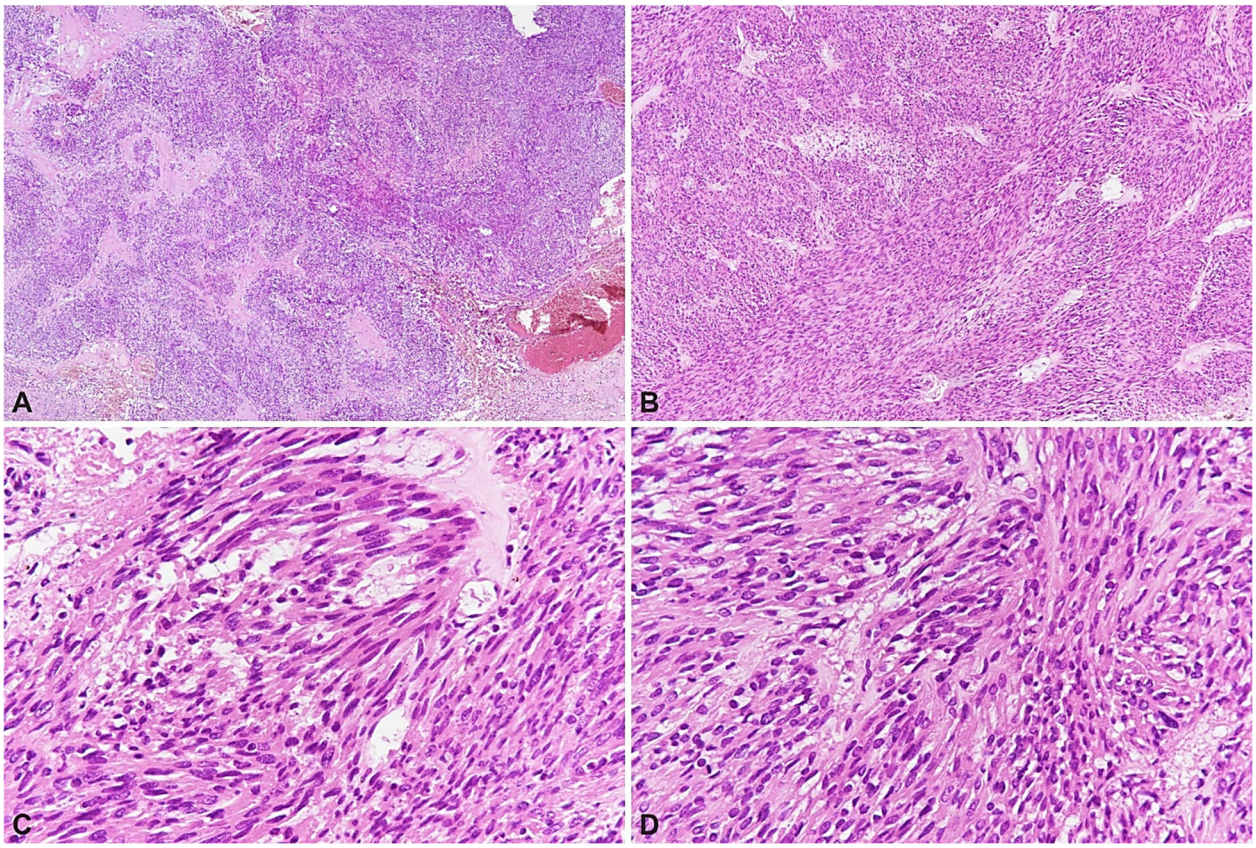

Figure 4 – ***Histological aspect of the tumor: (A) The visible capsule with hemorrhage area; (B) The cells are arranged in a fasciculate pattern, with myxoid areas, hemorrhage, and necrosis; (C and D) The cells have a fusiform epithelioid shape with hypertrophic nuclei and unevenly arranged chromatin (C), as well as karyorrhexes and mitoses (D). HE staining: (A) ×40; (B) ×100; (C and D) ×400. HE: Hematoxylin–Eosin.***

Table 1 – ***Specific antibody characteristics***

Antibody	Specificity	Clone	Dilution	Producer	Microwave oven pretreatment
CD117	Membranous and cytoplasmatic, Cajal cells, melanocytes, germinal cells	T595	1:40	LEICA	Seven cycles citrate buffer
CD34	Membranous, blood cells, endothelial cells	QBend10	1:100	DAKO	Five cycles citrate buffer
DOG1	Cytoplasmatic, membranous, GIST	K9	RTU	LEICA BOND	20 minutes EDTA-TS buffer
SMA	Cytoplasmatic, smooth muscle cells	1A4	1:50	DAKO	Three cycles citrate buffer
S100	Cytoplasmatic, Schwann cells, myoepithelial, mesenchymal	Polyclonal	1:500	DAKO	
Ki67	Nuclear	MIB-1	1:20	DAKO	Seven cycles citrate buffer
NSE	Cytoplasmatic, membranous, neuroendocrine	BBS/NC/V1-H14	1:200	DAKO	Seven cycles citrate buffer

CD: Cluster of differentiation; DOG1: Discovered on GIST1; GIST: Gastrointestinal stromal tumor; NSE: Neuron-specific enolase; SMA: Smooth muscle actin; RTU: Ready-to-use; EDTA-TS: Ethylenediaminetetraacetic acid tetrasodium salt.

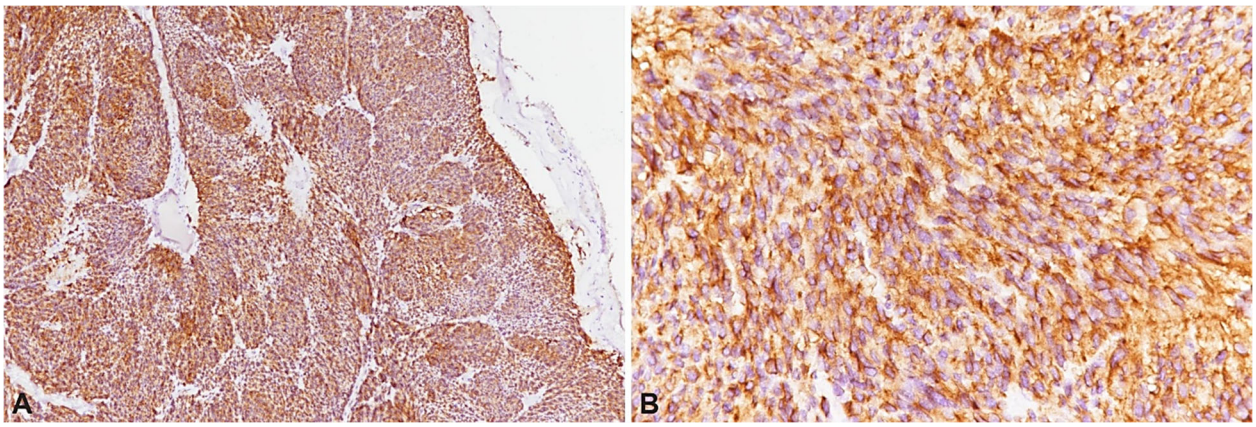

Figure 5 – ***(A and B) IHC reactivity with clear cell change highlighting CD117 intense positive tumoral cells. Anti-CD117 antibody immunostaining: (A) ×40; (B) ×400. CD: Cluster of differentiation; IHC: Immunohistochemistry.***

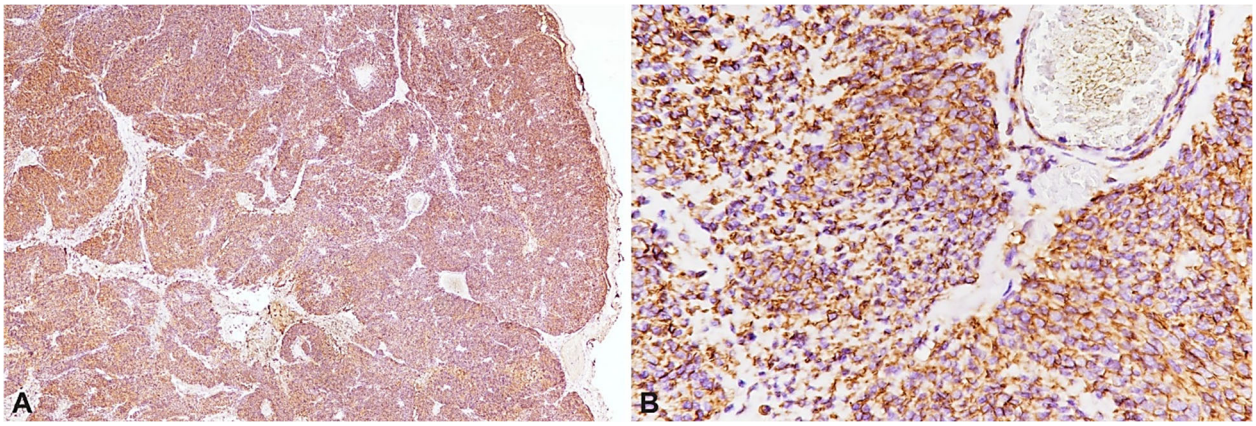

Figure 6 – ***(A and B) IHC images showing intense CD34-positive epithelioid-shaped tumoral cells. Anti-CD34 antibody immunostaining: (A) ×100; (B) ×400.***

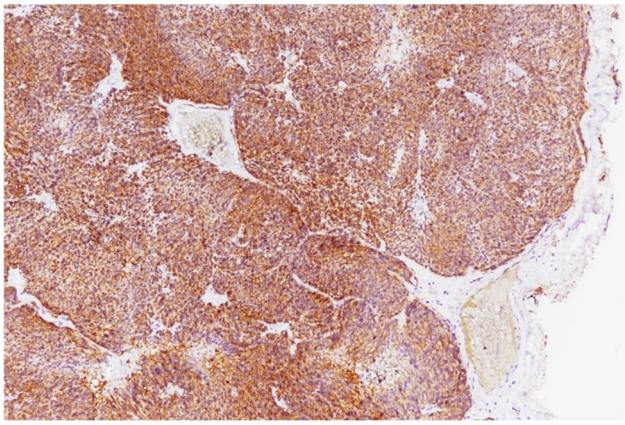

Figure 7 – ***IHC reactivity to the anti-DOG1 antibody showing intense positive tumoral cells. Anti-DOG1 antibody immunostaining, ×400. DOG1: Discovered on gastrointestinal stromal tumor (GIST) 1.***

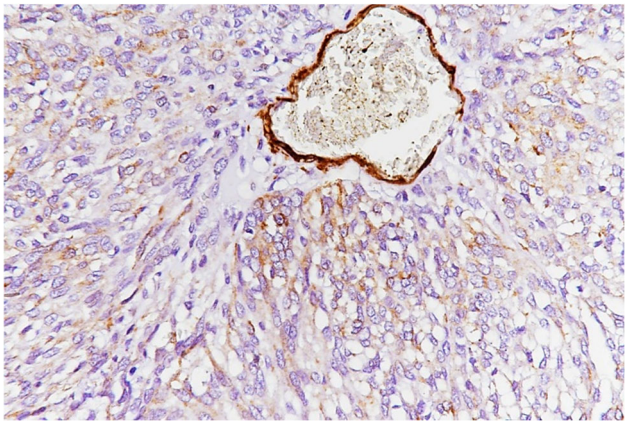

Figure 8 – ***IHC image of anti-SMA antibody showing only focal immunoreaction of the muscle cells. Anti-SMA antibody immunostaining, ×400. SMA: Smooth muscle actin.***

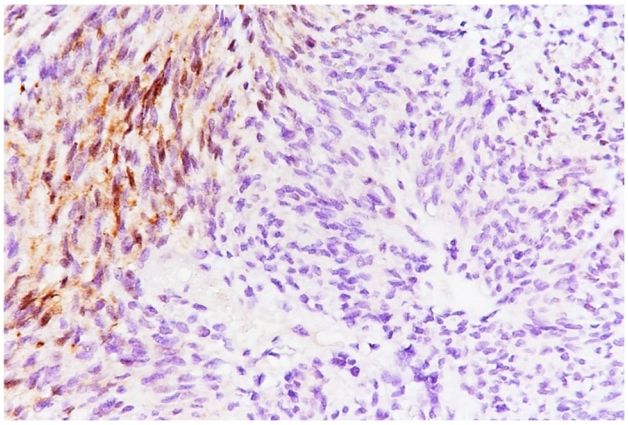

Figure 9 – ***IHC image with positive focal NSE with variable intensity. Anti-NSE antibody immunostaining, ×400. NSE: Neuron-specific enolase.***

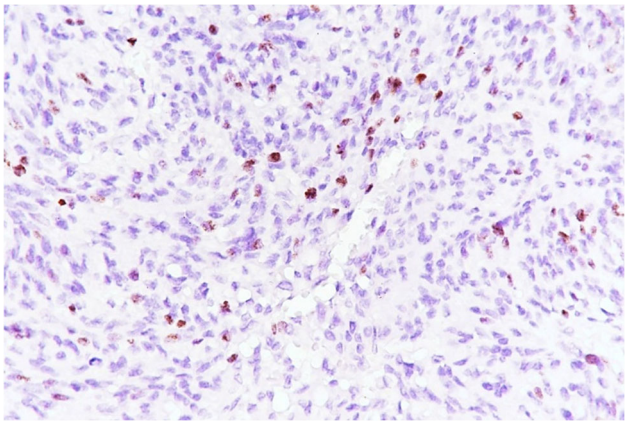

Figure 10 – ***IHC image of Ki67 positivity in 5% of the tumoral cells. Anti-Ki67 antibody immunostaining, ×400.***

The patient was successfully discharged and underwent oncology follow-up with Imatinib, with no relapse a year after surgical resection.

Discussions

Gastric GISTs are the most frequent mesenchymal tumors originating from the GI wall and lately, new studies are considering them as a much more common entity than previously recognized [10]. This may be closely tied to the fact that most patients are asymptomatic and by performing a larger number of endoscopies over the world for various reasons, GISTs are discovered. Also, by widening the bariatric surgery field, incidental GIST has become a more common finding. Mendes *et al.* [11] published their experience on 2655 patients who followed either Roux-en-Y or laparoscopic sleeve gastrectomy and found that 17 patients had gastric GIST with a size smaller than 1 cm. Moreover, many pathological findings have found microscopic GISTS on autopsies, with a higher incidence in the stomach [12].

While there is no rule for gastric GIST's evolution, cross-sectional imaging studies have presented them with an endophytic or exophytic position reported to the gastric wall. Thus, their progression correlates with their symptomatic features. When gastric GISTs protrude within the stomach, the most common symptom is considered to be GI bleeding, which is related to the surface erosions of the lesions, whereas when located exophytically, the most common finding is the palpable abdominal mass and bowel obstruction due to other structure compression caused by the large tumor size [13, 14].

Providing a perioperative diagnosis might be challenging, especially when discussing a large gastric GIST with exophytic distribution. Thus, other tumor origins should be considered, such as colon, ovary, mesenteric, retroperitoneum, or mediastinum. Contrast-enhanced CT scan has a key role for diagnosis in this situation and is also able to assess disease extension [15, 16]. Even though there is no standard characterization for gastric GISTs, CT images usually point out a large inhomogeneous mass with necrosis, as well as possible hemorrhage spots. On the other hand, endoscopic ultrasound (EUS) stands as another valid investigation for these types of tumors, as it can establish the point of origin within the gastric wall and also harvest cytology samples [17]. However, the use of EUS has shown to have a lower correlation for size and malignant prognostic than CT scan. EUS findings, such as cystic spaces have a higher risk for malignant GISTs, whereas the exophytic distribution is better correlated with CT scan for poor prognostic [18].

Given that the standard of care for gastric GISTs should require EUS–fine-needle aspiration (FNA) as a first diagnostic step, biopsies are not required when the tumor is resectable. Also, symptomatic GISTs that are either bleeding or causing gastric outlet obstruction might require surgical resection as a first choice. For exophytic giant gastric GISTs, there is no standardization of surgical procedure, and all interventions should be adapted per patient with the purpose of achieving R0 resection to the greatest extent possible and also preserve the utmost of the nearby organs. Lymph node dissection is considered necessary only if invasion is suspected on clinical pre-operative imaging [19, 20].

Because HP findings are not sufficient for a definitive diagnosis, IHC findings are essential to confirm the diagnosis. While morphology shows spindle-shaped cells and epithelial cells, it is not enough to exclude other mesenchymal tumors, thus, immunostaining is indicated. A positive diagnosis is based on the presence of KIT and CD34, however, if these two are negative, additional staining should be done for desmin, S100 and DOG1 [21].

Adjuvant oncologic therapy is considered the next step after surgical resection for both high and intermediate-risk patients. Available data have defined the patient's risk according to high mitotic rate, tumor size and location, Ki67 expression index, as well as a metastatic presence within diagnosis [22].

Studies published so far on gastric GISTs >10 cm are mostly case reports (Table 2). Hence, therapeutic management is not well defined and requires a multidisciplinary approach to improve survival rates [23–29]. Since our patient presented in a complicated stage with intermittent gastric outlet obstruction symptoms, we chose not to delay the surgical procedure and try to succeed in full surgical resection as the first therapeutic step. Perioperative management should include imaging tests which are imperative to determine the tumor's point of origin. While upper endoscopy only revealed an extrinsic compression, with no lesions within the stomach and US could not provide more information, it was necessary to determine if the obstructive symptoms were caused by an intestinal giant tumor. However, the CT scan suggested that the tumor originated from the gastric wall and causing a mass effect on adjacent organs. Also, it provided important information regarding various invaded structures.

Table 2 – ***Review of case reports with gastric GISTs like our case. We selected only exophytic gastric location, >10 cm, that caused gastric outlet obstruction***

Case No.	Date of publishing	Authors	Age [years]	Sex	Tumor size [cm]	Ki67 index	No. of mitoses/HPFs	IHC	Treatment
1.	10/2019	Mohammed & Arif [23]	65	F	45×21		5/50	Not mentioned	Imatinib + Surgical + Imatinib
2.	02/2018	Matsuo *et al.* [24]	71	F	20×20		3/30	(+) CD117/c-kit (+) CD34 (+) DOG1	Surgical
3.	08/2017	Chen *et al.* [14]	68	F	13×10×10		10/50	(+) CD117/c-kit (+) CD34 (+) DOG1	Surgical
4.	08/2013	Cappellani *et al.* [25]	67	M	37×24×13	>10%	5/50	(+) CD117/c-kit (+) CD34	Surgical + Imatinib
5.	07/2013	Anania *et al.* [26]	63	F	19×11×9		7/50	(+) CD117/c-kit (+) DOG1	Surgical
6.	06/2013	Baskiran *et al.* [27]	38	F	14×13×10		1/50	(+) CD117/c-kit (+) CD34	Surgical + Imatinib
7.	07/2008	Cruz *et al.* [28]	37	M	32×25×21		10/50	(+) CD117/c-kit (+) CD34 (+) SMA	Surgical + Imatinib
8.	06/2006	de Roover *et al.* [29]	66	M	9		14/50	(+) CD117/c-kit	Surgical + Imatinib

CD: Cluster of differentiation; DOG1: Discovered on GIST1; F: Female; GIST: Gastrointestinal stromal tumor; HPF: High-power field; IHC: Immunohistochemistry; M: Male; SMA: Smooth muscle actin.

Surgical management is a complex patient-individualized process that should be initiated in this case due to the complicated status. Multiorgan resection was necessary to achieve full tumor extraction. Another surgical aspect was the careful abdominal exploration to avoid possible tumor rupture or bleeding since it also had necrosis and bleeding spots. Moreover, tumor resection margin is a major challenge for giant GISTs, and cannot be well defined, as tumor resection is individualized by its extension.

The patient fully recovered after surgery and underwent

Imatinib. Some studies have suggested the use of Imatinib in the preoperative stage either for metastatic disease or to reduce the tumor volume and thus to avoid resection of adjacent organs as much as possible. Thus, we suggest that surgery should be the first line of therapy if the giant gastric GISTs are complicated.

Prognostic factors that should be considered are based on Fletcher's risk classification [30] and include tumor size, mitotic index, and primary tumor site. Our patient was directly included in the high-risk group based on the size of the tumor over 20 cm, regardless of the mitotic index, which was also >5, and the gastric tumor site. The post-operative objective is based on early detection of possible recurrence, which usually consists of liver or peritoneal dissemination that may occur during resection [31]. The natural history of gastric GISTs is unknown, with few occurrences after surgery, especially after 10 years of follow-up. In our case, the patient was assessed after four years, and no sign of recurrence was observed. Even though research is ongoing in the field of gastric cancer regarding the tumor microenvironment and treatment, GIST remains a special type of gastric neoplasia with challenging behavior that requires a specific approach [32].

Conclusions

Giant gastric GISTs are different in behavior and their symptomatology may lead to an early surgical management. There are only several cases of gastric exophytic gastric GIST provoking intermittent gastric outlet obstruction. Tumor resection should be adapted to every patient's status, focused on en bloc extraction, with preservation of invaded organs as much as possible. The multidisciplinary approach remains mandatory for better management of giant gastric GISTs.

Conflict of interests

The authors declare that they have no conflict of interests.

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