CASE REPORT

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Synchronous gastric tumors – case presentation

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Abstract

Synchronous gastric tumors, and, especially the presence of an adenocarcinoma and gastrointestinal stromal tumors, are less frequent. We present the case of a 75-year-old patient, with no gastrointestinal pathology in the medical history, who was admitted for marked asthenia, nausea, coffee grounds vomiting, inappetence, dizziness, weight loss and periodical epigastralgias. The clinical and imagistic examinations highlighted an ulcerative, infiltrative, bleeding tumor formation, present on the anterior side and subcardially on the small curvature. During the surgery, there was highlighted a second tumor, whitish, of about 2.5 cm, prominent under the peritoneal serous, of firm consistency and with an adherence to the stomach muscles. For removing the two tumors, there was performed total gastrectomy with esophagus-jejunal termino-lateral anastomosis, with jejunum ansa "in omega". The histopathological and immunohistochemical examinations established that the first tumor was a poorly differentiated carcinoma, and the second was a gastrointestinal stromal tumor. The patient's evolution was a good one, both clinically and biologically, the imagistic examinations performed after six and 12 months highlighting the lack of local relapses and absence of metastases.

Keywords: gastric adenocarcinoma, gastrointestinal stromal tumor, synchronous tumors, endoscopy, gastrectomy.

Introduction

Although the gastric cancer incidence decreased a lot in the Western countries, it continues to remain a major source of morbidity and mortality all over the world [1]. Recent estimations indicated that, worldwidely, every year there are recorded about 950 000 new cases of gastric cancer and about 720 000 people die, having as main death cause gastric cancer. All over the world, there is estimated that gastric neoplasms represent the fifth most frequent cancer form and represent the third cause of death by cancer, representing 8.8% of cancer deaths every year [2]. In China and other Asian countries, gastric cancer represents the third most frequent cancer form in both sexes [3, 4]. The most frequent cancer form is adenocarcinoma, representing about 85–90% of gastric tumors [5].

Gastrointestinal stromal tumors (GISTs) are the most frequent mesenchymal tumors of the gastrointestinal tract [6]. They may be found all over the digestive tract, from the esophagus to the rectum, still the most frequent are to be found affecting the stomach (60–70%) and the small intestine (20–30%) [7–10]. Their incidence is estimated to 10–20 per million [11–12], but only 1–2% of them are malignant tumors and metastasize in the liver, peritoneum or lymphatic ganglions [13–15]. Nevertheless, it is considered that the stromal gastrointestinal tumors have an unpredictable and variable behavior [16, 17].

We present the case of a 75-year-old patient with a subcardial, perforated, covered gastric adenocarcinoma, associated with a gastrointestinal stromal tumor localized in the gastric antrum.

Case presentation

The patient MC, aged 75 years, was admitted to the Clinic of Gastroenterology, Emergency County Hospital of Arad, Romania (OS No. 3253/25.01.2015), suffering from coffee grounds vomiting, inappetence, dizziness, weight loss (approx. 10 kg in the last three months) and periodical epigastralgias. The symptoms were progressive in the last 3–4 months, with digestive disconfort, inappetence, asthenia and moderate epigastralgias. Vomiting and hematemesis appeared 2–3 days before, which determined the patient to present to hospital.

In his medical history, the patient was diagnosed with

type 2 diabetes mellitus (for 10 years), ischemic cardiopathy and high blood pressure (HBP), for about eight years, conditions for which the patient constantly received treatment and periodical check-ups. The patient declared that he did not use to consume alcohol or smoke.

The clinical examination at hospital admission highlighted a normosthenic, normoponderal patient (weight 76 kg, height 179 cm), presenting pale skin and mucosa, with persistent skin crease, slightly dehydrated, foul tongue and reduction of muscular mass. The examination of the cardiovascular system showed sinusal rhythm, normal blood pressure (BP) (130/75 mmHg), ventricular allure (VA) 94 beats/minute. The abdomen was light, painful at feeling in the epigastrium, and the liver was slightly enlarged (3 cm below the rib cage), elastic, pain free; the spleen and kidneys were not palpable, and the Giordano technique was bilaterally negative.

Abdominal ultrasounds highlighted a slight enlargement of the liver, with posterior remission, with no other significant changes in the abdominal organs that could be observed in ultrasounds.

Lung X-ray did not highlight the presence of pathological lesions in the lungs or mediastinum, and the electrocardiogram was within normal limits.

The computed tomography (CT) examination with contrast substance, i.v. administered, highlighted a heterogeneous thickening of the gastric wall on the anterior side and on the small curvature, and the presence of 8–10 mm adenopathies in the small curvature. There were not highlighted any nodular formations suspect of karyokinetic lesions in the liver, kidneys, spleen or adrenals. Also, there was not highlighted the presence of liquid in the peritoneal cavity.

For establishing the etiology of hematemesis, there was performed a gastroscopic examination, showing the presence of a normal esophagus, with a permeable cardia foramen. At about 6 cm below the cardia foramen, on the stomach anterior side and on the small curvature, there was highlighted an ulcerative, infiltrative, bleeding tumor formation, approx. 4.5 cm in diameter; in the pyloric antrum, there was highlighted a papular erosive, intensely congested lesion; the pylorus was permeable, and the duodenal bulb had a normal aspect.

The biological examinations showed the presence of a severe anemia (hemoglobin 8.8 g/100 mL, red blood cells 3.14 million/mm^3, hematocrit 26.2%), leukocytosis (leukocytes 13 700/mm^3) with neutrophilia (neutrophils 84%), mild thrombocytemia (thrombocytes 475 000/mm^3), hyperglycemia (glycemia 486 mg/dL), moderate increase of urea (urea 158 mg/dL) and serum creatinine (creatinine 1.24 mg/dL).

Based on the clinical examination and paraclinical investigations, there was established the diagnosis of gastric neoplasm associated with secondary anemia, unbalanced diabetes mellitus.

After the biological rebalancing, the patient was transferred to the Ist Clinic of Surgery, of the same Hospital, for a surgical intervention.

After the usual preoperatory preparation, there was surgically opened the abdominal cavity, where there was identified the presence of a tumor formation on the anterior wall of the stomach and small curvature, with an infiltrative, necrosing, bleeding characteristic, covered with false membranes and with the inferior side of the liver. On the small curvature, there were identified various lymphatic ganglions of large dimensions and consistency. Perigastrically, there was highlighted a small quantity of puss liquid, that was harvested for bacteriological and biochemistry studies.

In the gastric antrum, on the anterior side, there was identified another oval, whitish, 2.5 cm tumor formation, prominent under the peritoneal serous, adherent to the stomach muscles.

The lesion evaluation imposed the performance of total gastrectomia with esophagus-jejunal termino-lateral anastomosis, with transcolic jejunal ansa "in omega" and Brown anastomosis at the jejunal ansa foot.

Afterwards, the entire resection piece was sent to the Laboratory of Pathological Anatomy for establishing the histopathological diagnosis.

After surgery, the patient's evolution was a favorable one, and the clinical and imagistic controls after six and 12 months since surgery did not identify any local relapses or presence of metastases.

The histopathological examination of gastric tumors was performed on pieces fixed in formalin, included in paraffin and stained with Hematoxylin–Eosin (HE). The tumor present on the anterior wall and on the small gastric curvature was a poorly differentiated adenocarcinoma (Figure 1), penetrating in the muscular tunica, associated with atrophic chronic gastritis (Figure 2) and intestinal metaplasia (Figure 3).

The tumor with the antral localization was formed of fusiform cells, organized in fascicles with variable orientation (Figure 4), mainly developed in the stomach muscular tunica, infiltrating the submucosa. Peritumorally and even intratumorally, there were highlighted microhemorrhagic foci (Figure 5). In the tumor area, the gastric mucosa presented areas with normal histological aspect (Figure 6), and also areas with gastric mucosa erosions associated with vascular congestion.

For the positive and differential diagnosis and for the evaluation of the tumor aggression, there were performed various immunohistochemical investigations, using the following antibodies: CD117 (polyclonal rabbit anti-human CD117 c-Kit, code A4502, 1:100 dilution, Dako), CD34 (monoclonal mouse anti-human CD34 class II, clone QBEnd 10, 1:50 dilution, Dako), anti-vimentin (monoclonal mouse anti-vimentin, clone V9, M0725, 1:50 dilution, Dako), anti-alpha-smooth muscle actin (α-SMA) (monoclonal mouse anti-human muscle actin, clone HHF35, M0635, 1:100 dilution, Dako), anti-p53 (monoclonal mouse anti-human p53 protein, clone DO-7, M7001, 1:50 dilution, Dako), anti-Ki67 (monoclonal mouse anti-human Ki67 antigen, clone MIB-1, M7240, 1:50 dilution), anti-cytokeratin (CK) 5/6 (monoclonal mouse anti-human CK5/6, clone D5/16 B4, M7237, 1:50 dilution, Dako), anti-CK8 [monoclonal mouse anti-human CK8 (low molecular weight – LMW), clone 35betaH11, Dako], anti-CK19 (monoclonal mouse anti-human CK19, clone RCK 108, M0888, Dako).

The immunohistochemical study showed that the tumor localized in the antrum was intensely positive for the following antibodies anti-CD117 (Figure 7), anti-CD34 (Figure 8) and anti-vimentin (Figure 9). The Ki67 cellular

proliferation index was quite low, about 1–2% of the tumor cells being positively marked in the nuclei (Figure 10), and the reaction to p53 was negative (Figure 11) and anti-α-SMA antibody (Figure 12). Also, the immunohistochemical reactions to CK5/6, CK8, CK19 were negative. The histopathological and immunohistochemical aspects showed that the tumor localized in the gastric antrum was a GIST.

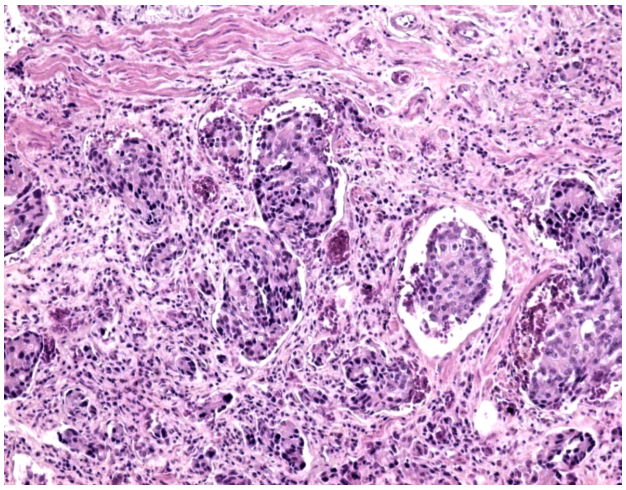

Figure 1 – ***Image of poorly differentiated gastric adenocarcinoma gastric (HE staining, ×100).***

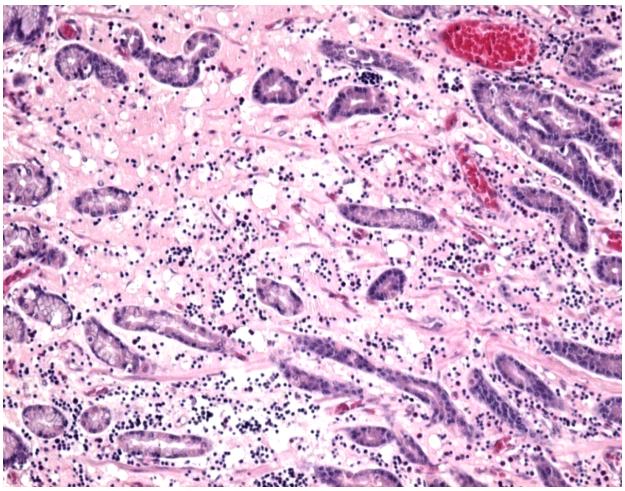

Figure 2 – ***Image of atrophic chronic gastritis (HE staining, ×100).***

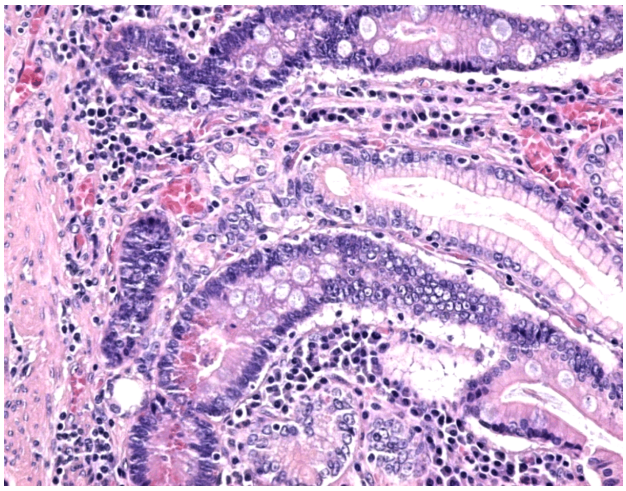

Figure 3 – ***Area of gastric mucosa with intestinal metaplasia (HE staining, ×200).***

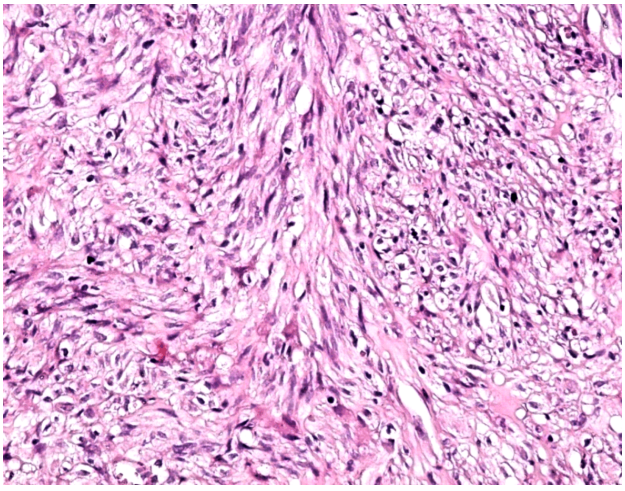

Figure 4 – ***Tumor cells with a fusiform aspect, arranged in fascicles with a varied, poorly vascularized orientation (HE staining, ×200).***

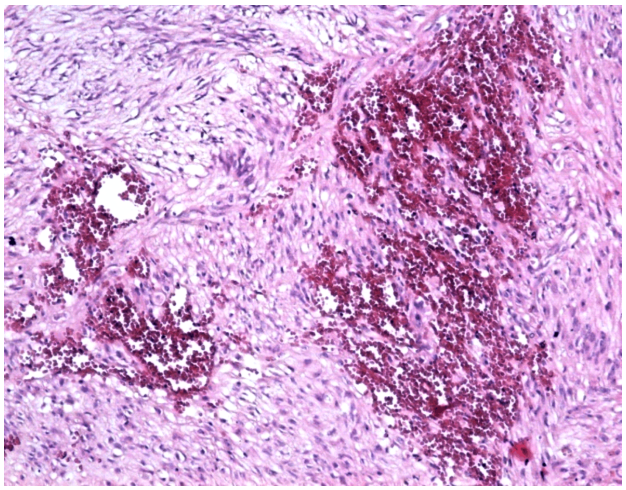

Figure 5 – ***Image of tumor parenchyma with areas of microhemorrhage (HE staining, ×200).***

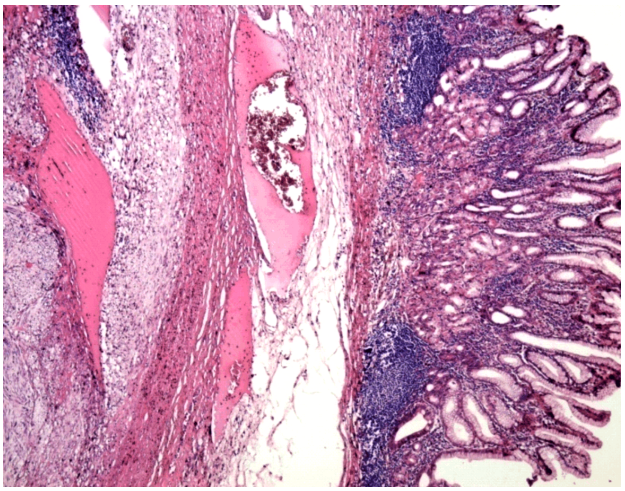

Figure 6 – ***GIST tumor developed in the stomach muscular tunica, invading in the subserous, associated with vascular congestion (HE staining, ×40).***

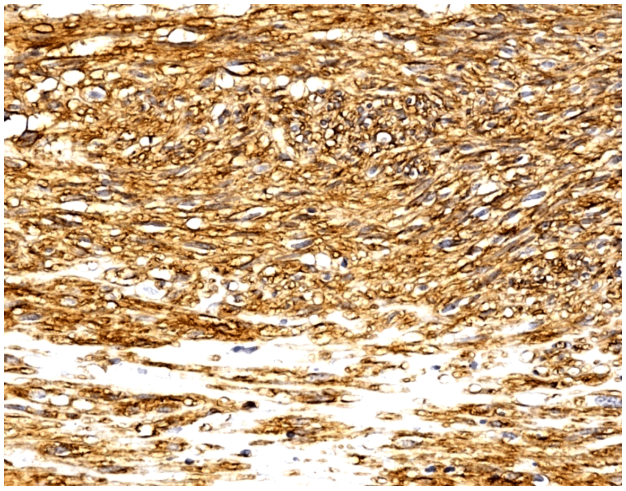

Figure 7 – ***Tumor cells with an intense reaction to the anti-CD117 antibody (Anti-CD117 antibody immuno-marking, ×200).***

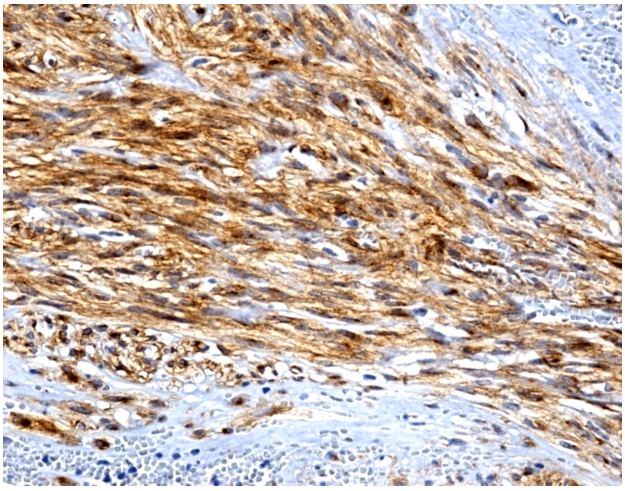

Figure 8 – ***Tumor cells with an intense reaction to the anti-CD34 antibody (Anti-CD34 antibody immuno-marking, ×200).***

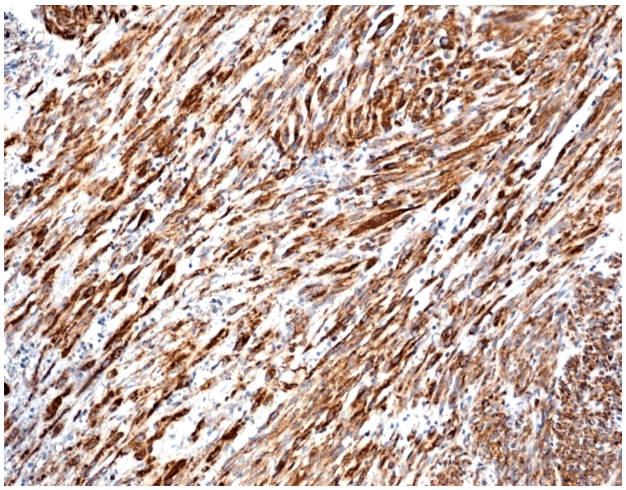

Figure 9 – ***Tumor cells with an intense reaction to the anti-vimentin antibody (Anti-VIM antibody immuno-marking, ×200).***

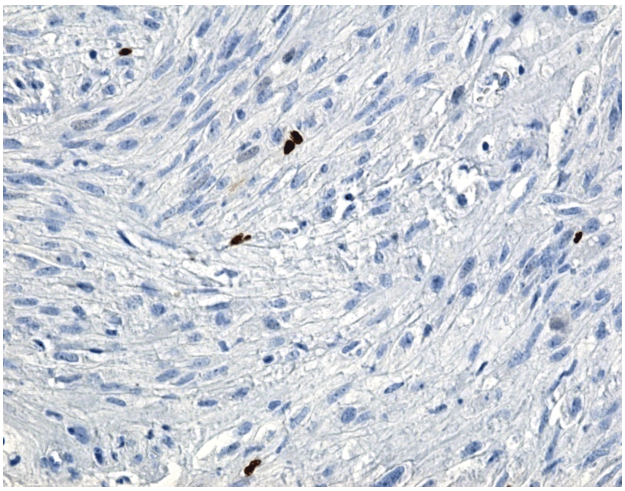

Figure 10 – ***Tumor cells with low immunohistochemical reaction to the anti-Ki67 antibody (Anti-Ki67 antibody immunomarking, ×200).***

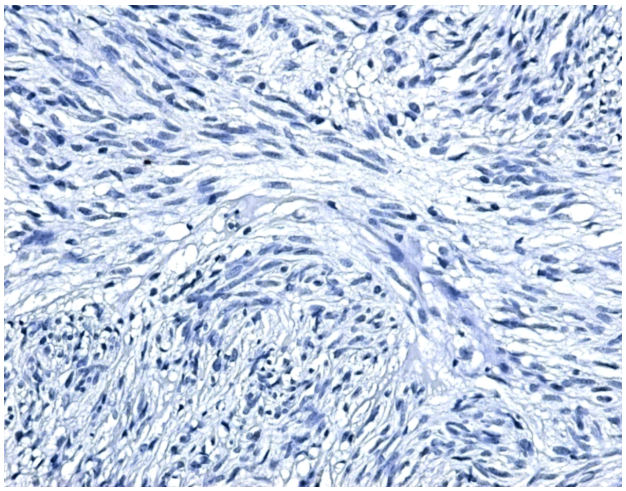

Figure 11 – ***Tumor cells with negative reaction to p53 (Anti-p53 antibody immunomarking, ×100).***

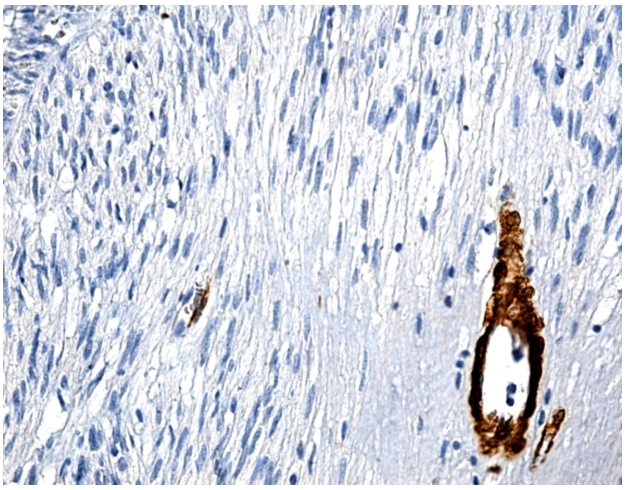

Figure 12 – ***Negative reaction of tumor cells to the anti-α-SMA antibody. Intensely positive reaction to α-SMA of the myocytes form tumor blood vessels (Anti-α-SMA antibody immunomarking, ×100).***

Discussion

The synchronous presence of a gastric adenocarcinoma with GIST is relatively rare, as the two types of tumors develop from different cells, present in the structure of the gastric wall [18]. The gastric adenocarcinoma develops in the mucosa of the gastric epithelium cells (either of the lining epithelium, or of gastric glands), while GIST develops most often in the muscular tunica, subserously, from the interstitial Cajal cells [19, 20]. GISTs are the most frequent non-epithelial tumors of the digestive tract and represent about 0.1–3% of the tumors of the digestive tube [21]. They may develop synchronously with other gastric tumors, such as adenocarcinomas, gastric carcinomas or gastric mucosa-associated lymphoid tissue lymphoma (MALT) [22]. GISTs may also develop outside the digestive tube, the most frequent being found in the caul, mesentery or in the retroperitoneal space [23].

The clinical and paraclinical studies published so far showed that most GISTs were detected by chance during surgeries for other conditions of the digestive tract [12].

In our case, the diagnosis of synchronous gastric tumor was established during surgery when, besides the infiltrative tumor on the anterior side and the stomach small curvature, there was highlighted another tumor with an antral localization, subserously, with macroscopic characteristics different from the first lesion. At first, there was thought that the antral tumor may represent a local tumoral metastasis, a sarcoma or a GIST. Although GIST in our case had relatively large sizes (about 2.5 cm), it was not detected during the CT examination of the abdomen. Numerous studies showed that GISTs had small sizes, some less than 1 cm, and, usually, they cannot be detected after surgery by imagistic examinations. However, they may represent a potential of malignant transformation [24, 25] and, because of this, surgeons should examine very carefully the digestive tract during certain surgeries. Most surgeons recommend the GIST extirpation, even though it apparently does not have any connection to the condition for which the surgery was performed, due to its potential of malignant transformation [26].

An interesting clinical aspect is that GISTs associated with other stomach tumors more frequently appear in the elderly, especially in men [27]. Our case belongs to the general data reported by other studies.

Regarding the clinical symptoms, in the case presented by us, there was determined the presence of gastric adenocarcinoma, GIST not having any symptoms, in our opinion. It is possible that the clinical manifestations of GIST could be masked by the ones of the adenocarcinoma. Some studies showed that large size GISTs manifest by abdominal pain and bleeding, most often expressed by melena [28]. Still, we believe that the clinical symptoms are correlated with the tumor size and localization.

For the positive and differential diagnosis of GIST, we performed various immunohistochemical investigations, because these tumors have extremely variable clinical and biological expressions [29]. The tumor cells were intensely positive to CD117, CD34 and vimentin, which indicates their mesenchymal origin. Numerous studies showed that the GIST cells are positive for CD117 in 80–95% of cases and for CD34 in 60–70% of cases [30, 31]. The positive expression of CD117 allows the differential diagnosis of GISTs, of leiomyoma, leiomyosarcoma or other mesenchymal origin tumors developed in the digestive tube [7, 9, 32].

In our study, the poor reaction of the GIST cells to the anti-Ki67 antibody showed a low capacity of low proliferation and a benign behavior.

The simultaneous presence of an epithelial tumor and of a stromal tumor in the stomach raises the question whether there is an incidental association, or, there is an etiopathological relation between the two tumors. Because this association is quite rare, we consider that the simultaneous presence of an epithelial tumor and a GIST in the stomach is poorly incidental. However, there are hypotheses according to which the same carcinogenetic agent may induce simultaneous proliferation and oncogenesis, both of the epithelial and of stromal cells [28, 33].

The treatment solution approached in our patient's case led to very good results, thus, after 12 months since complete gastrectomia, there were not observed any local relapses or presence of metastases in the main abdominal organs, nor adenopathies.

Conclusions

The presence of synchronous tumors in the stomach may be possible. In our case, the detection of GIST in the antral area, synchronous with a gastric adenocarcinoma, was purely incidental, imposing a complete gastrectomy. The histopathological and immunohistochemical examinations were essential in establishing the diagnosis and monitoring the post-surgery treatment. In our case, GIST was intensely positive to CD117, CD34 and vimentin, and negative to α-SMA, p53 and cytokeratins.

Conflict of interests

The authors declare that they have no conflict of interests.

References

[1] Katona BW, Rustgi AK. Gastric cancer genomics: advances and future directions. Cell Mol Gastroenterol Hepatol, 2017, 3(2):211–217.
[2] Torre LA, Bray F, Siegel RL, Ferlay J, Lortet-Tieulent J, Jemal A. Global cancer statistics, 2012. CA Cancer J Clin, 2015, 65(2):87–108.
[3] Chen WQ, Zheng RS, Zhang SW, Zeng HM, Zou XN. The incidences and mortalities of major cancers in China, 2010. Chin J Cancer, 2014, 33(8):402–405.
[4] Cheng XJ, Lin JC, Tu SP. Etiology and prevention of gastric cancer. Gastrointest Tumors, 2016, 3(1):25–36.
[5] Yan Y, Li Z, Liu Y, Zhang L, Li J, Ji J. Coexistence of gastrointestinal stromal tumors and gastric adenocarcinomas. Tumour Biol, 2013, 34(2):919–927.
[6] Feng F, Liu Z, Zhang X, Guo M, Xu G, Ren G, Hong L, Sun L, Yang J, Zhang H. Comparison of endoscopic and open resection for small gastric gastrointestinal stromal tumor. Transl Oncol, 2015, 8(6):504–508.
[7] Miettinen M, Lasota J. Gastrointestinal stromal tumors (GISTs): definition, occurrence, pathology, differential diagnosis and molecular genetics. Pol J Pathol, 2003, 54(1):3–24.
[8] Behranwala KA, Spalding D, Wotherspoon A, Fisher C, Thompson JN. Small bowel gastrointestinal stromal tumours and ampullary cancer in type 1 neurofibromatosis. World J Surg Oncol, 2004, 2:1.
[9] Mehta RM, Sudheer VO, John AK, Nandakumar RR, Dhar PS, Sudhindran S, Balakrishnan V. Spontaneous rupture of giant gastric stromal tumor into gastric lumen. World J Surg Oncol, 2005, 3(1):11.

[10] Rebegea LF, Pătraşcu A, Miron D, Dumitru ME, Firescu D. Metachronous gastrointestinal stromal tumor associated with other neoplasia – case presentation. Rom J Morphol Embryol, 2016, 57(4):1429–1435.
[11] Tzen CY, Wang JH, Huang YJ, Wang MN, Lin PC, Lai GL, Wu CY, Tzen CY. Incidence of gastrointestinal stromal tumor: a retrospective study based on immunohistochemical and mutational analyses. Dig Dis Sci, 2007, 52(3):792–797.
[12] Liu Z, Liu S, Zheng G, Yang J, Hong L, Sun L, Fan D, Zhang H, Feng F. Clinicopathological features and prognosis of coexistence of gastric gastrointestinal stromal tumor and gastric cancer. Medicine (Baltimore), 2016, 95(45):e5373.
[13] Miettinen M, Furlong M, Sarlomo-Rikala M, Burke A, Sobin LH, Lasota J. Gastrointestinal stromal tumors, intramural leiomyomas, and leiomyosarcomas in the rectum and anus: a clinicopathologic, immunohistochemical, and molecular genetic study of 144 cases. Am J Surg Pathol, 2001, 25(9):1121–1133.
[14] Grignol VP, Termuhlen PM. Gastrointestinal stromal tumor surgery and adjuvant therapy. Surg Clin North Am, 2011, 91(5):1079–1087.
[15] Kubo N, Takeuchi N. Gastrointestinal stromal tumor of the stomach with axillary lymph node metastasis: a case report. World J Gastroenterol, 2017, 23(9):1720–1724.
[16] Kim HH. Endoscopic treatment for gastrointestinal stromal tumor: advantages and hurdles. World J Gastrointest Endosc, 2015, 7(3):192–205.
[17] Barbu LA, Mărgăritescu ND, Ghiluşi MC, Belivacă D, Georgescu EF, Ghelase ŞM, Marinescu D. Severe upper gastrointestinal bleeding from gastrointestinal stromal tumor of the stomach. Rom J Morphol Embryol, 2016, 57(4):1397–1401.
[18] Shi Z, Zhuang Q. Computed tomography imaging characteristics of synchronous gastrointestinal stromal tumors in patients with gastric cancer and correlation with clinicopathological findings. Mol Clin Oncol, 2015, 3(6):1311–1314.
[19] Nowain A, Bhakta H, Pais S, Kanel G, Verma S. Gastrointestinal stromal tumors: clinical profile, pathogenesis, treatment strategies and prognosis. J Gastroenterol Hepatol, 2005, 20(6):818–824.
[20] Telugu RB, Pushparaj M, Masih D, Pulimood A. Synchronous appearance of adenocarcinoma and gastrointestinal stromal tumour (GIST) of the stomach: a case report. J Clin Diagn Res, 2016, 10(2):ED16–ED18.
[21] Rabin I, Chikman B, Lavy R, Sandbank J, Maklakovsky M, Gold-Deutch R, Halpren Z, Wassermann I, Halevy A. Gastrointestinal stromal tumors: a 19 year experience. Isr Med Assoc J, 2009, 11(2):98–102.
[22] Al-Brahim N, Radhi J, Gately J. Synchronous epithelioid stromal tumour and lipoma in the stomach. Can J Gastroenterol, 2003, 17(6):374–375.
[23] Dimofte MG, Porumb V, Ferariu D, Bar NC, Luncă S. EGIST of the greater omentum – case study and review of literature. Rom J Morphol Embryol, 2016, 57(1):253–258.
[24] Maiorana A, Fante R, Cesinaro AM, Fano RA. Synchronous occurrence of epithelial and stromal tumors in the stomach: a report of 6 cases. Arch Pathol Lab Med, 2000, 124(5): 682–686.
[25] Kawanowa K, Sakuma Y, Sakurai S, Hishima T, Iwasaki Y, Saito K, Hosoya Y, Nakajima T, Funata N. High incidence of microscopic gastrointestinal stromal tumors in the stomach. Hum Pathol, 2006, 37(12):1527–1535.
[26] Liu YJ, Yang Z, Hao LS, Xia L, Jia QB, Wu XT. Synchronous incidental gastrointestinal stromal and epithelial malignant tumors. World J Gastroenterol, 2009, 15(16):2027–2031.
[27] Shen C, Chen H, Yin Y, Chen J, Han L, Zhang B, Chen Z, Chen J. Synchronous occurrence of gastrointestinal stromal tumors and other digestive tract malignancies in the elderly. Oncotarget, 2015, 6(10):8397–8406.
[28] Lin YL, Tzeng JE, Wei CK, Lin CW. Small gastrointestinal stromal tumor concomitant with early gastric cancer: a case report. World J Gastroenterol, 2006, 12(5):815–817.
[29] Cai R, Ren G, Wang DB. Synchronous adenocarcinoma and gastrointestinal stromal tumors in the stomach. World J Gastroenterol, 2013, 19(20):3117–3123.
[30] Lai EC, Lau SH, Lau WY. Current management of gastrointestinal stromal tumors – a comprehensive review. Int J Surg, 2012, 10(7):334–340.
[31] Arabadzhieva E, Yonkov A, Bonev S, Bulanov D, Taneva I, Vlahova A, Dikov T, Dimitrova V. A rare case with synchronous gastric gastrointestinal stromal tumor, pancreatic neuroendocrine tumor, and uterine leiomyoma. World J Surg Oncol, 2016, 14(1):287.
[32] Fletcher CD, Berman JJ, Corless C, Gorstein F, Lasota J, Longley BJ, Miettinen M, O'Leary TJ, Remotti H, Rubin BP, Shmookler B, Sobin LH, Weiss SW. Diagnosis of gastrointestinal stromal tumors: a consensus approach. Hum Pathol, 2002, 33(5):459–465.
[33] Andea AA, Lucas C, Cheng JD, Adsay NV. Synchronous occurrence of epithelial and stromal tumors in the stomach. Arch Pathol Lab Med, 2001, 125(3):318–319.

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