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Colic adenocarcinoma and gastric stromal tumor's association: a new case

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Abstract

Synchronous development of epithelial and stromal Tumors of the digestive tract was rarely reported in the literature. A case report is described in this article: a 46-yr-old male manifesting a gastric stromal tumor-associated with colic adenocarcinoma

Introduction

GIST (Gastro Intestinal Stromal Tumors) are the most common mesenchymal tumors of the gastrointestinal tract. They are now well highlighted, as far as histology and molecular approach are concerned; gastric localization remains the most frequent in 65% of the cases [1, 2, 3]. Treatment advances have made of it a tumor pathology of interest [4, 5]. Synchronous or metachronous combination of GIST with gastrointestinal carcinoma has rarely been reported in the literature. In this work, we report gastric GIST association to colic adenocarcinoma in a 46-yr-old patient.

Case Report

It is a 46-yr-old patient (smoking 12 PY and chronic alcoholic) who manifests, it's been one year, diffuse abdominal pain of cramps type with early vomiting with no bowel movement disorder, no signs of bleeding disorders, and no other extra-intestinal symptoms; all this is developing into a whole damage of general health. The clinical examination reports a patient with bad condition (IMC 16 kg/m^2, mild abdominal tenderness), with free ganglionic areas.

Performed Laboratory tests underlined hypochromic microcytic anemia 10.8 g/dl. Normal hydro electrolyte check up indicates high tumor markers ACE and CA19,9. A performed abdominopelvic CT shows irregular thickening, stenosis and heterodense of antro pyloric region measuring 12 mm with significant stomach stasis (figure 1), and irregular, not stenosing thickening, including the down cecal bottom, extended to the ileocecal junction (figure 2), with the presence of deep adenopathy.

Figure 1. Scannographic section showing thickening antropyloric stenosis with gastric stasis

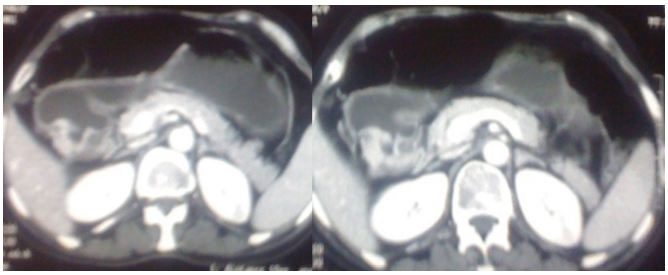
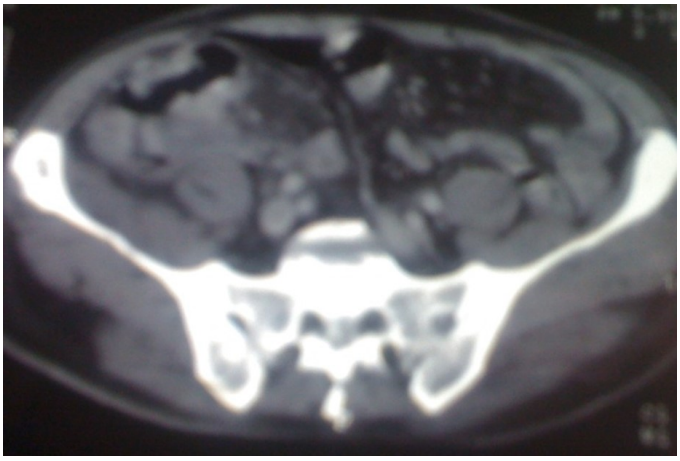


Figure 2. Scannographic section showing thickening of the cecal low bottom



The digestive assessment was completed by an esogastro-duodenal endoscopy showing a very suspicious antropyloric process of malignancy; multiple biopsies were performed. The histological study with immunohistochemical supplement showed malignant mesenchymal tumor proliferation, necrotic with strong, diffuse CD 117 and CD34 positivity and negativity of CK; this helped establish GISTs diagnosis (figure 3).

Performed Colonoscopy showed a hemi-circumferential tumor process, non stenosing cecal with, based on the histological study, a poorly differentiated invasive colic adenocarcinoma (figure 4). The extended assessment did not underline other secondary localizations.

Figure 3. Prolifération fusocllulaire type GIST

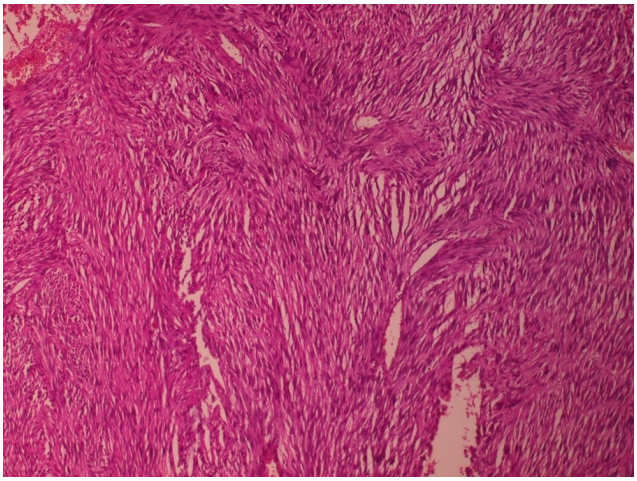
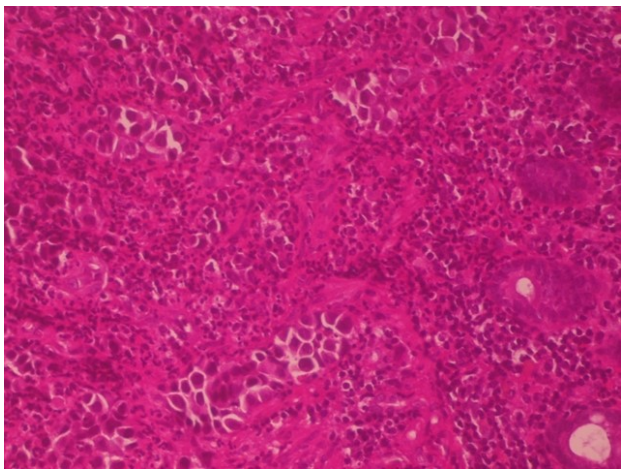


Figure 4. carcinome à cellules isolées sur polype dénomateux



The treatment consisted of a monoblock resection of the gastric mass which measured 07 × 05 × 03 cm without residue or tumor breaking out and with healthy limits exceeding 2 cm; a right hemicolectomy and a appendectomy to lymphadenecto-my were realised.

Gastric specimen histological check up confirmed GIST diagnosis deemed of low-risk, while the colon surgery specimen showed the presence of adenocarcinoma infiltrating the mucosa without ganglionic affection or vascular emboli with healthy and largely sufficient resection limits.

Given these histological data and the absence of later metastasis, and after a multidisciplinary meeting, the patient did not need adjuvant therapy. After a two-years monitoring, the patient is still in complete remission.

Discussion

Stromal tumors represent less than 1% of gastrointestinal tumors; however, they are the most common mesenchymal tumors of the gastrointestinal tract [1]. The latter is made by a proliferation of spindle cells, sometimes epithelioid expressing Kit protein in most of the cases (more than 90%); other markers are used and necessary, such as the CD34 which can be expressed in 60-70% of the cases. It is also made by a proliferation of smooth muscle actin in 30 to 40% of the cases, of PS100 in 5% of the cases, and of desmin in about 2% of the cases [6].

GISTs are most of the time isolated, but the coexistence of other pathologies, which remains rare, is described in the literature as the triad of Carney. It combines multiple gastric stromal tumors, pulmonary chondroma, and extraadrenal paraganglioma. GIST can be found in Recklinghausen's disease with an estimated prevalence between 5 and 25%. There are also familial forms of GIST associated to hyperpigmentation, urticaria pigmentosa, and/or systemic mastocytosis [7, 8]. Outside these well described associations, the synchronous development of GIST and digestive epithelial tumors were rarely reported in the literature. Various hypotheses have been suggested regarding the simultaneous development, and it is unclear whether or not such an association is a simple coexistence as mentioned in Maiorana and al's study [9], or the two are connected by a secondary causal relationship to a genetic disorder or a common oncogenic factor able to induce tumors of different tissue-types, referring to Cohen and al's study [10].

Conclusion

Between either hypothesis the question remains. For our case, we think it was rather a chance association, due to lack of evidence in our patient which can lead towards the genetic

origin without being able to rule it out definitely.

Conflict of interest: The authors declare no conflict of interest.

References

1. B. Landi, O. Bouché, R. Guimbaud, J.-A. Chayvialle Tumeurs stromales gastro-intestinales (GIST) de taille limitée (inférieure à 5cm): revue de la littérature et propositions pour la prise en charge: Gastroentérologie Clinique et Biologique Volume 34, n. 2 pages 120-133 (février 2010)
2. Miettinen M., Lasota J. Gastrointestinal stromal tumors--definition, clinical, histological, immunohistochemical, and molecular genetic features and differential diagnosis Virchows Arch 2001; 438: 1-12
3. Lasota J., Miettinen M. KIT and PDGFRA mutations in gastrointestinal stromal tumors (GISTs) Semin Diagn Pathol 2006; 23: 91-102
4. Bucher P, Villiger P, Egger J, et al. Management of gastrointestinal stromal tumors: From diagnosis to treatment. Swiss Med Wkly 2004;134:145-53
5. Demetri G, Benjamin R, Blancke C, et al. NCCN task force report: Management of patient with gastro-intestinal stromal tumor (GIST) – update of the NCCN clinical practice guidelines. J Natl Comp Cancer Network 2007; 5(Suppl.2): S1-29.
6. Bennani Guebessi N, Karkouri M, Belhaj. S, Benkirane A, Jabri. L, Azzouzi. S, Zamiati. S: Adénocarcinome du colon et tumeur gastro-intestinale stromale gastrique: association rare: African Journal of Cancer May 2010, Volume 2, Issue 2, pp 120-122
7. M'sakni. I, Rammeh. S, Chelbi. E, Sayari. S, Zaouech. A, Baltagi-Ben Jilani. S, Zermani. R: Adénocarcinome et tumeur stromale digestifs: association fortuite ou mécanisme oncogène commun? À propos de deux observations. Annales de chirurgie, Volume 131, Issue 8, 2006, pp 464–467.
8. Miettinen M., Sobin L., Lasota J. Gastrointestinal stromal tumors of the stomach: a clinicopathologic, immunohistochemical, and molecular genetic study of 1765 cases with long term follow-up Am. J. Surg. Pathol. 2005; 29: 52-68
9. Maiorana A., Fante R., Cesinario A.M., Fano R.A. Synchronous occurrence of epithelial and stromal tumors in the stomach: a report of 6 cases Arch. Pathol. Lab. Med. 2000; 124: 682-686
10. Cohen A., Geller S.A., Horowitz I., Toth L.S., Werther J.L. Experimental models for gastric leiomyosarcoma: the effects of N-methyl-N'-nitro-N-nitrosoguanidine in combination with stress, aspirin, or sodium taurocholate Cancer 1984; 53: 1088-1092