

Which is your diagnosis?

Pedro José de Santana Júnior¹, Ana Caroline Vieira Aurione², Rafael Dangoni², Roberta Rodrigues Monteiro da Gama³,
Tiago Marinho Almeida Noletto³, Kim-Ir-Sen Santos Teixeira⁴

Study developed at Department of Radiology and Imaging Diagnosis of Hospital das Clínicas – Universidade Federal de Goiás (UFG), Goiânia, GO, Brazil. 1. Titular Member of Colégio Brasileiro de Radiologia e Diagnóstico por Imagem (CBR), Substitute Professor at School of Medicine – Universidade Federal de Goiás (UFG), Goiânia, GO, Brazil. 2. MDs, Residents, Department of Radiology and Imaging Diagnosis, Hospital das Clínicas – Universidade Federal de Goiás (UFG), Goiânia, GO, Brazil. 3. Trainee

Physicians, Department of Radiology and Imaging Diagnosis, Hospital das Clínicas – Universidade Federal de Goiás (UFG), Goiânia, GO, Brazil. 4. PhD, Associate Professor and Head of Department of Radiology and Imaging Diagnosis, Hospital das Clínicas – Universidade Federal de Goiás (UFG), Goiânia, GO, Brazil. Mailing Address: Dr. Pedro José de Santana Júnior. Avenida T4, nº 1616, ap. 402 B, Setor Serrinha. Goiânia, GO, Brazil, 74835-090. E-mail: pedrojoseradihocufg@gmail.com.

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A 62-year-old male patient complaining of asthenia, anorexia, nausea and vomiting for two months, with weight loss of 20 kg in this period. He brought the results from high digestive endoscopy performed in another service, with report of ulcerated gastric lesion. Pallor and weight deficit

were observed at physical examination. The patient denied previous history of diseases and surgeries. Computed tomography (CT) (Figures 1 and 2) was performed and subsequently the patient was submitted to laparotomy with gastroenteroanastomosis and jejunostomy with biopsy.

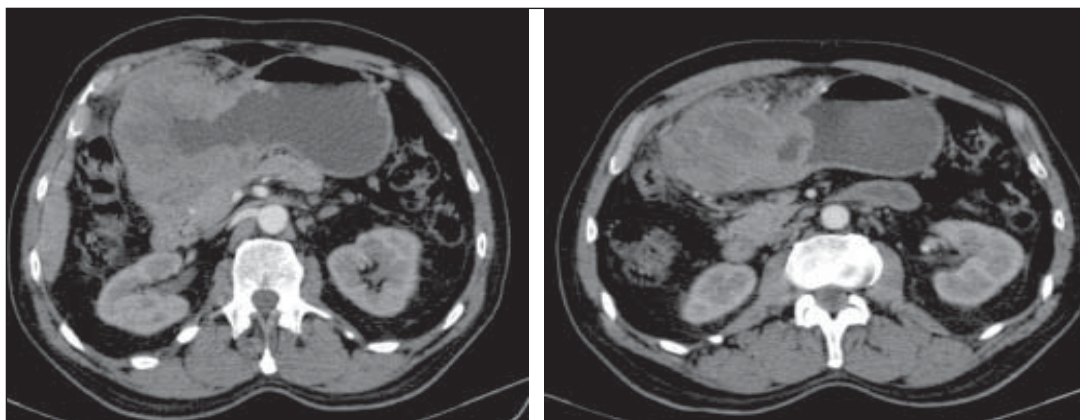


Figure 1. Contrast-enhanced whole abdomen CT, axial sections.

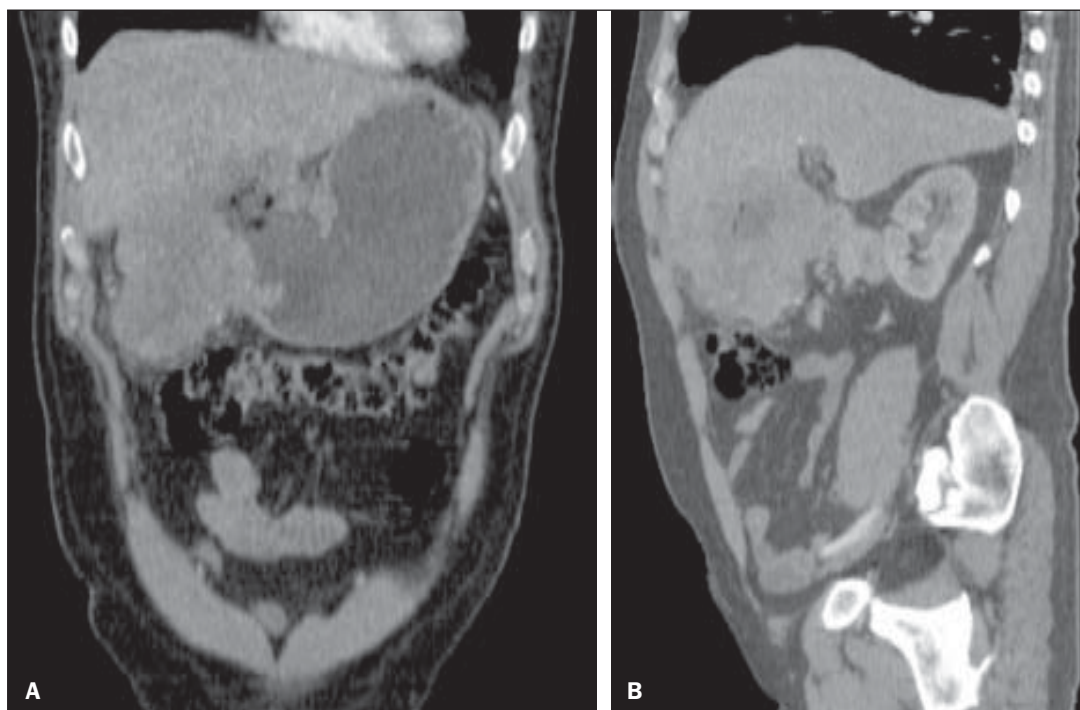


Figure 2. Contrast-enhanced whole abdomen CT, coronal (A) and sagittal reformation (B).

Images description

Figure 1. Contrast-enhanced whole abdomen CT, axial sections. A large lesion involving the gastric antropyloric region and the first duodenal portion, characterized by remarkable concentric irregular parietal thickening, with hypoattenuating central regions (liquefaction necrosis).

Figure 2. Contrast-enhanced whole abdomen CT, coronal (A) and sagittal reformation (B). A large lesion involving the gastric antropyloric and the first duodenal portion, characterized by remarkable concentric irregular parietal thickening, with hypoattenuating central regions (liquefaction necrosis). There are signs of invasion of hepatic segments III and IV.

Diagnosis: Type III gastric neuroendocrine tumor.

COMMENTS

The imaging evaluation of the digestive system has been approached by a series of recent publications in the Brazilian literature⁽¹⁻⁸⁾.

Neuroendocrine tumors originate in different organs, most commonly in the gastrointestinal tract (GIT) and secondly in the bronchopulmonary tract^(9,10). More rarely, such tumors affect the liver, gallbladder, ovaries, testicles and thymus, sites which also contain neuroendocrine system cells⁽⁹⁾.

Neuroendocrine gastric (carcinoid) tumor is a rare neoplasia originating from enterochromaffin cells, corresponding to 1.2–1.5% of all stomach tumors, with an incidence of 1.6-2.0 cases per 100,000 patients/year⁽¹¹⁾. Stomach is the least common site of GIT tumors, losing in frequency for small bowel, rectum, colon, cecum and appendix⁽¹²⁾. The mean age of diagnosed patients is 60 years, with predominance in female individuals (2:1)⁽¹²⁾.

Gastric carcinoid tumors are usually classified into three types. Type I (70–85% of cases) develops with hypergastrinemia and is related to atrophic gastritis and pernicious anemia. It occurs in mid aged women, with multiple, small polypoid lesions and rarely produces metastases. Type II (5–10% of cases develops with hypergastrinemia related to Zollinger-Ellison syndrome, in most of cases in a context of type I multiple endocrine neoplasia (MEN I). Type III (15–25%), where the presently described lesion is included, presents a poor prognosis. There is no relation with hypergastrinemia and it is sporadic. Its highest incidence occurs mainly in men at the sixth decade of life, with metastases at diagnosis (frequently nodal and hepatic metastases). Such lesion is solitary, larger than 2 cm, presenting an aggressive behavior and dissemination by contiguity to the liver, many times with tendency to necrosis⁽¹¹⁻¹³⁾.

The differential diagnosis for types I and II lesions includes adenomatous or hamartomatous polyps⁽¹³⁾. Kaposi sarcoma and metastases may also manifest as small gastric parietal lesions. Adenocarcinoma, lymphoma and gas-

trointestinal stromal tumors are considered as differential for type III lesions⁽¹³⁾, substantially more frequent, which highlights the relevance of the present report.

The diagnosis of neuroendocrine gastric tumors involves high digestive endoscopy, endoscopic ultrasonography, computed tomography or magnetic resonance imaging, besides biopsy with immunohistochemical analysis.

The surgical treatment depends on the lesion type and extent, and corresponds to local resection, antrectomy or total gastrectomy, either associated or not with lymphadenectomy. The clinical treatment involves the use of somatostatin for symptoms management, in addition to complementary chemotherapy and/or radiotherapy as necessary.

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