MAR Gastroenterology (2026) 5:02

Case Report

Coexistence of Gastric Gastrointestinal Stromal Tumor and Colon Cancer: A Case Report

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Received: 24 March 2026

Published: 01 April 2026

DOI: https://doi.org/10.5281/zenodo.19678995

Abstract

The prevalence of gastrointestinal stromal tumors (GISTs) remains rare. Few cases of synchronous GIST and colonic adenocarcinoma have been reported. Here we report a case of GIST and colonic adenocarcinoma. The patient presented with the predominant symptoms of epigastric pain and epigastric swelling. Abdominal CT scan and upper GI endoscopy revealed gastric GIST. A colonoscopy was done for the indication of a positive stool occult blood test and found to have sigmoid adenocarcinoma after colonoscopic biopsy. The patient underwent surgical therapy followed by adjuvant chemotherapy for sigmoid cancer and imatinib for the high risk GIST. Though the coexistence of these two tumor types is rare, it is important to be aware of their disease patterns.

Keywords: *Colonic neoplasm, gastrointestinal stromal tumors.*

Introduction

The term "gastrointestinal stromal tumor "(GIST) was introduced by Mazur and Clark in 1983 to differentiate GISTs from leiomyomas (1, 2). It arises from the interstitial cells of Cajal, which are responsible for autonomous GI movement (3). Although GIST represents the commonest mesenchymal tumors of the gastrointestinal tract, the incidence ranges between 0.1–1 percent of all gastrointestinal malignancies, predominantly occurring in adults 55–65 years of age (4, 5).

The driver mutations for the development of GIST are tyrosine kinase receptor c-kit (c-KIT) or Platelet-Derived Growth Factor Receptor Alpha (PDGFR-α) gene mutations. Diagnosis is usually based on the expression of CD117 (90–95%), and CD34 (70–80%) (6).

The most common location of GISTs is the stomach (50–60%), followed by the small intestine (30–40%), the colon-rectum (5–10%), and rarely the esophagus (< 5%) (4, 7). GISTs may also develop in extra-GI sites, mainly the mesentery, the omentum, and the retroperitoneum.

GISTs can appear as small and benign nodules to frankly malignant tumors.

The synchronous occurrence of GISTs with other malignancies varies from 4.5% to 33%, with the most frequent localization of GIST associated malignancies in the GI tract and the urogenital and female genital tract (8).

Colorectal adenocarcinoma is a significant clinical problem and the third most common neoplasm worldwide, making up around 10% of all cancer diagnoses (9, 10). In Ethiopia, colorectal cancer is one of the commonest

cancers in men and the third most common cancer in women and overall the third most common cancer, accounting for 8.2% of all cancer diagnosis (11). Unlike GIST, colonic adenocarcinomas are epithelial tumors that arise from the colonic mucosa, classically because of alterations in the APC tumor suppressor gene, resulting in overactivation of the Wnt/β-catenin signaling pathway. Here we present a 52-year-old male with a simultaneous occurrence of a gastric GIST and colonic adenocarcinoma.

Case Presentation

A 52-year-old male patient presented with epigastric pain of one year duration and recently started to have epigastric swelling. He had also experienced significant weight loss. He had a history of bowel habit change and was found to have intestinal amebiasis in stool examination at a nearby center, and then, after treatment, it resolved. He had fatigue and loss of appetite. He had a history of hemorrhoids on lifestyle modification. He had a history of seasonal bronchial asthma, and the last attack was one year ago. No rectal bleeding history. No family history of CRC. No history of DM or HTN. On physical examination he has a raised blood pressure of 150/90 mmHg with the rest of vital signs within normal range. His ECOG performance was 0. There was a palpable mass in the epigastric region. On laboratory investigations, the stool occult blood test was positive, and the other tests, such as CBC, OFT, serum electrolytes, HA1c, uric acid, lipid profile, Stool examination, H. pylori stool antigen, CEA, and CA 19-9 checkup, were all in the normal range. Chest, abdominal and pelvic CT scan done with a standard protocol showed a heterogeneous, large, exophytic gastric mass likely to be a GIST (Figure 1). Esophagogastroduodenoscopy revealed an extramucosal tumor (Figure 2). Colonoscopy revealed sigmoid cancer (Figure 3) and confirmed it with a biopsy (Figure 4).

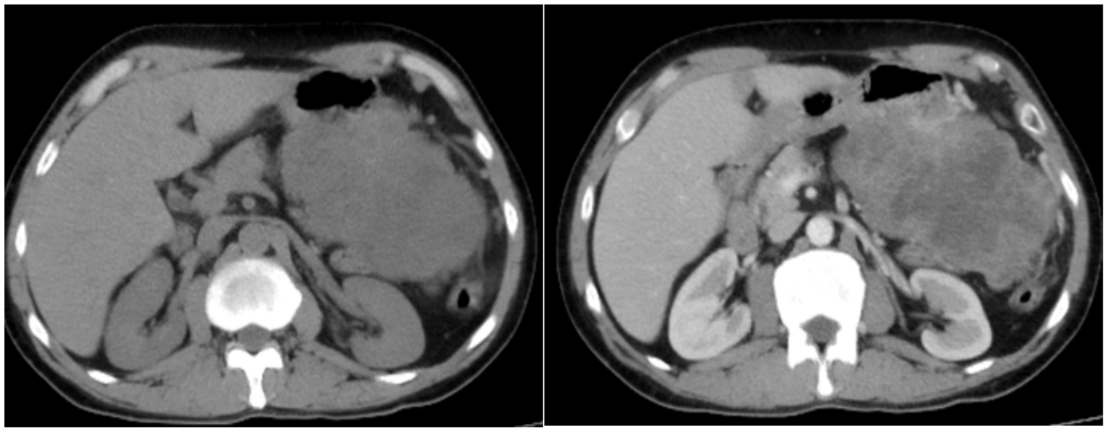

Figure 1: Abdominal CT scan showing a heterogeneously hypo dense exophytic 11*11*9cm mass arising from the body of the stomach, it shows heterogeneous enhancement with hypo enhancing central areas.

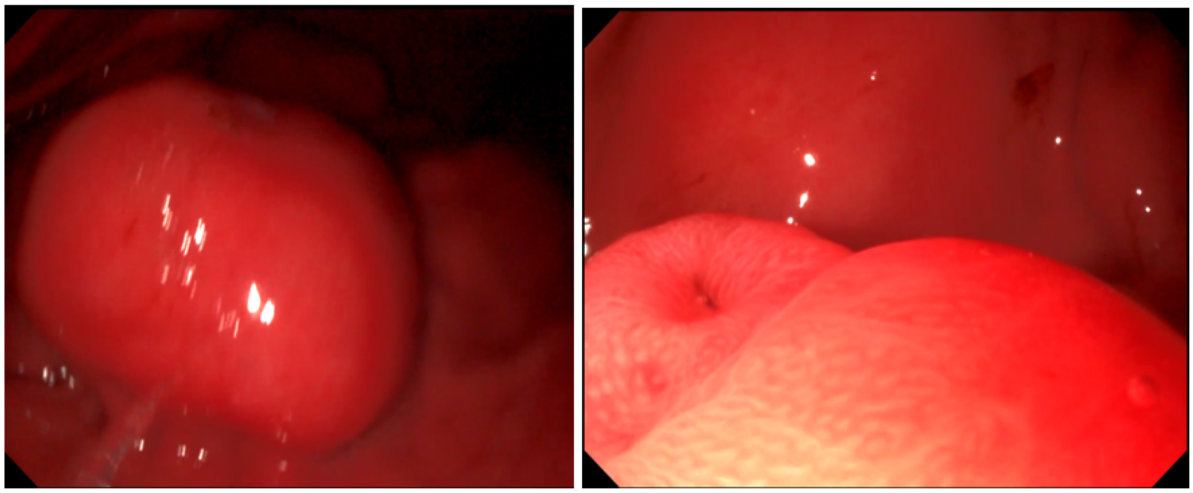

Figure 2: Esophagogastroduodenoscopy revealed a polypoid extramucosal round elevated mass at the junction of gastric body and fundus with central umbilication having extrinsic compression with normal overlying mucosa, suggesting extramucosal mass

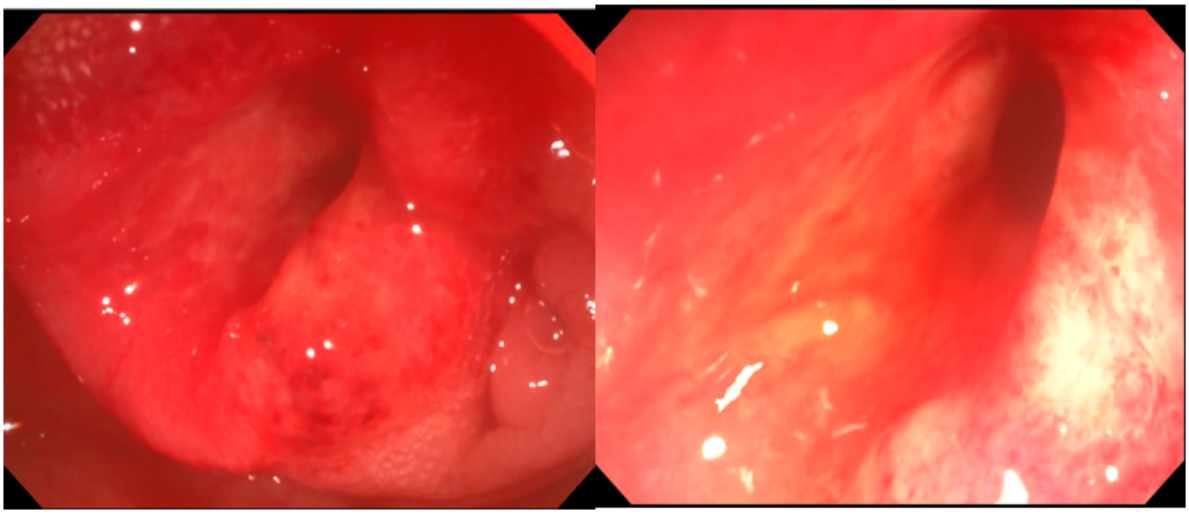

Figure 3 Colonoscopy examination revealed obstructive circumferential ulcerating fragile mass at 30cm from anal verge.

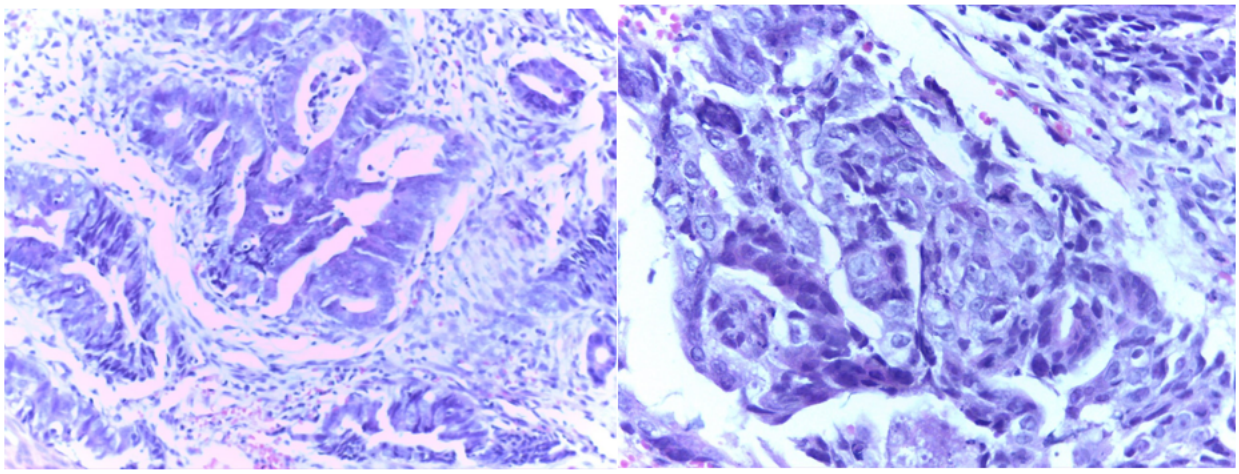

Figure 4 Invasive irregular confluent glands lined by hyperchromatic pleomorphic cells with occasional prominent nucleoli (H&E stain, 20x and 40x respectively)

Intraoperative findings (IOP) (figure 5) were:

1) Gastric body mass without lymphadenopathy or adjacent organ involvement

2) Sigmoid colonic mass with only a few intermediate lymph nodes, with T3/4 clinical stage

Then, the following were done;

1) Gastric wedge resection with about a 2cm gross margin and repaired in layers

2) Sigmoidectomy done with D2 dissection and primary anastomosis

The surgical pathology result from the gastric mass showed high-risk spindle cell type GIST (accordingly to both NIH/ Fletcher and AFIP/ Miettinen Criteria), which is strongly and diffusely positive for CD117 (Figure 6). The colonic mass was histologically diagnosed as adenocarcinoma, moderately differentiated, with a TNM stage of pT3N0Mx (Figure 7).

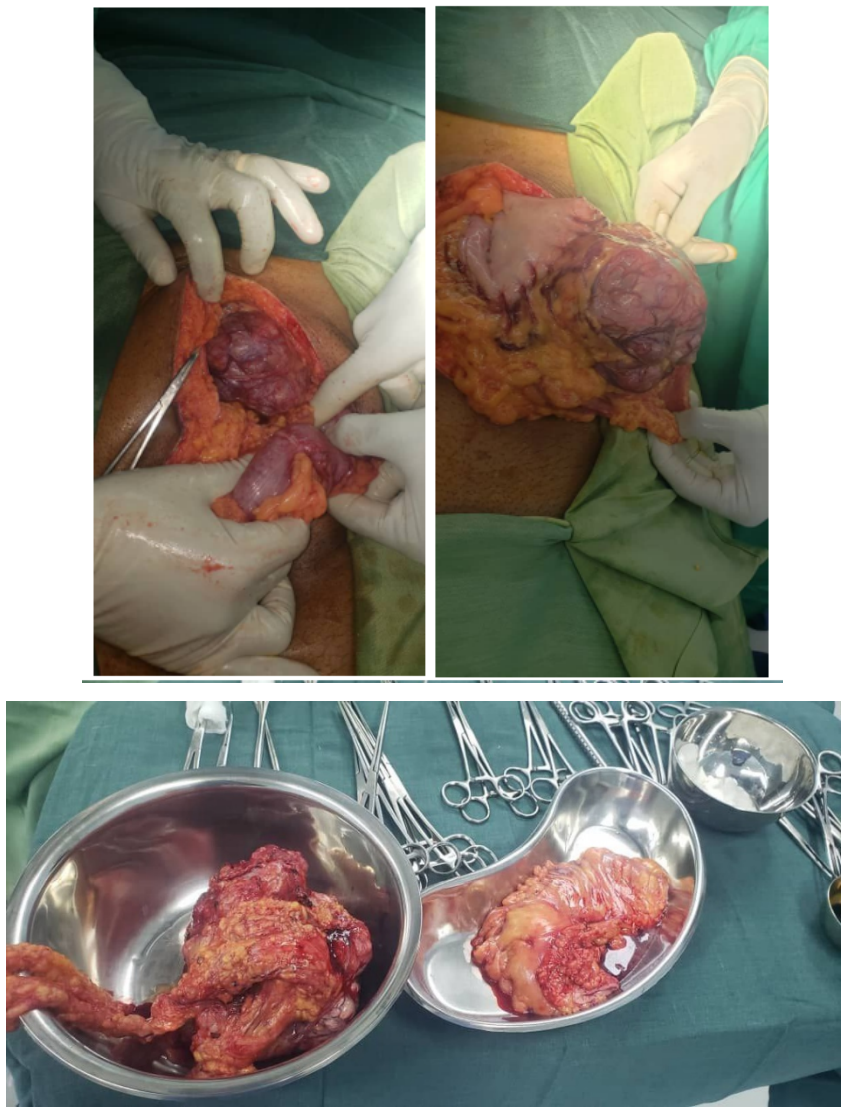

Figure 5: Intraoperative view of gastric GIST and resected sigmoid colonic mass

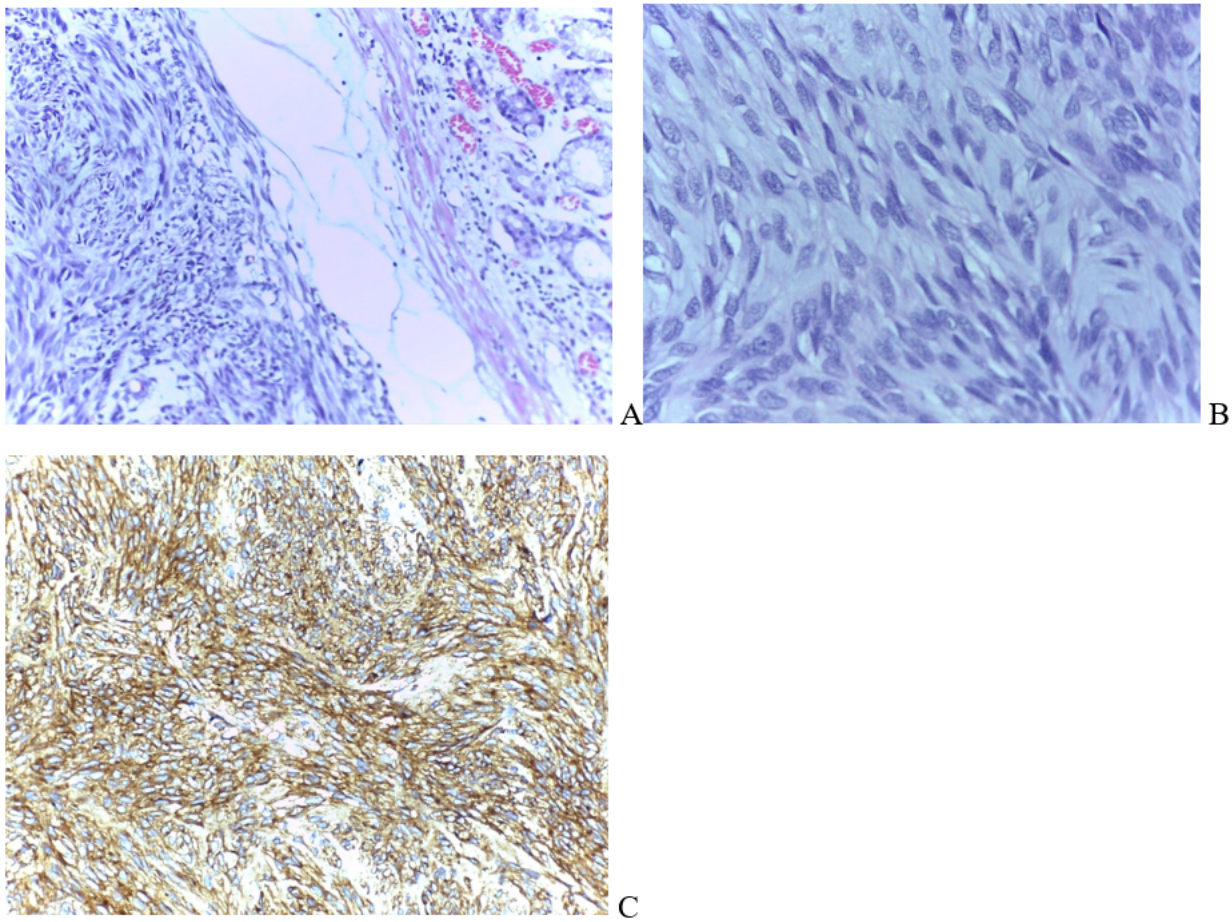

Figure 6 (A & B). Well-circumscribed submucosal tumor having spindle cell proliferation with inconspicuous nuclei and around 6 mitotic activities per 12 HPF (H&E stain, 20X and 40X respectively). **(C).** IHC for CD 117 shows 97% strong cytoplasmic stain (20X).

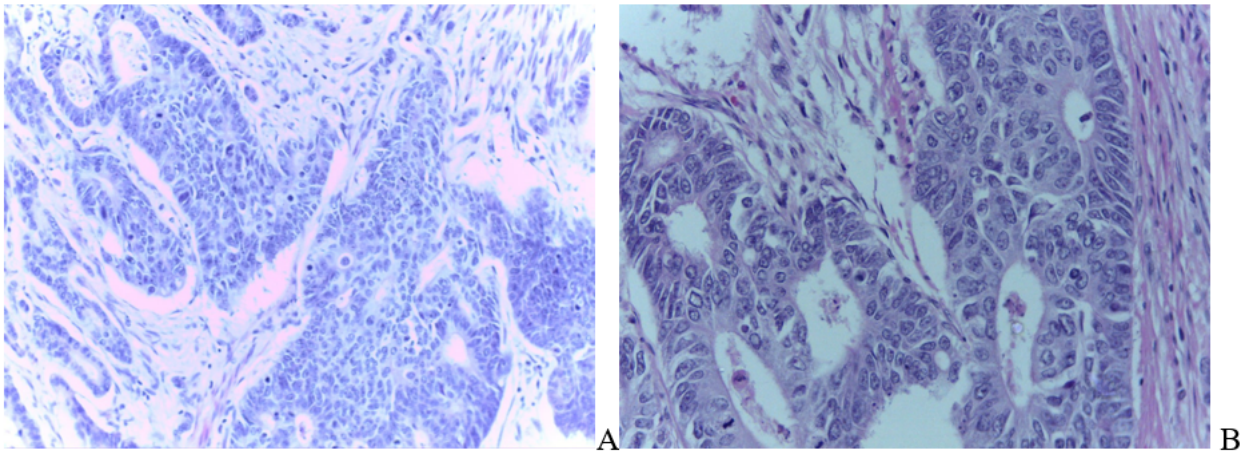

Figure 7 (A & B). Invasive confluent glandular structure lined by hyperchromatic pleomorphic cells, with occasional prominent nucleoli and significant mitotic activity (H&E stain, 20X and 40X respectively)

Subsequently, the patient was given six cycles of adjuvant CAPOX chemotherapy for sigmoid cancer. Imatinib (Gleevec) 400 mg PO daily started after the completion of chemotherapy to avoid overlapping toxicity. Amlodipine 10 mg PO once per day was given for essential hypertension. At follow-up CBC, RFT, LFT, Na, K were checked and all results were normal. The patient is in good health condition continuing his routine activities.

Discussion

The causal association of the concomitant occurrence of GIST with other malignancies, or if this is merely a coincidence, is not yet clear. Up to now, three theories describe the pathogenetic mechanisms underlying the occurrence of synchronous GIST-associated tumors. First, a non-causal relationship suggesting co-occurrence happens incidentally (12). Second, in a non-causal relationship model, exposure to potential carcinogens can trigger oncogenetic pathways in both epithelial and mesenchymal cells (13). Finally, common genetic mutations from both epithelial and stromal cells might be the triggering factor (14).

The clinical presentation of GIST is typically characterized by abdominal pain, GI bleeding, weight loss, anemia, and a palpable mass (15). Our patient had abdominal pain, weight loss, and a palpable mass but no overt GI bleeding or anemia.

The coexistence of GISTs and adenocarcinoma at two separate locations in the GI tract is uncommon (16). Both colon cancer and GISTs are infrequently associated with a genetic disposition, and in this report, the patient did not have a family history of any malignancies.

In synchronous cases, GISTs are usually found incidentally during the surgical operation or the histopathologic examination of the resected tissue (17). In our patient there was a clinical suspicion because of a positive stool occult blood test, and a pre-operative diagnosis was made with a colonoscopy examination.

In a case report of a patient who was treated for GIST and underwent surveillance abdominal imaging, he was having liver masses at follow-up, and finally, the diagnosis was metachronous sigmoid cancer (18). In our case, if a colonoscopy was not done and a diagnosis of sigmoid cancer was not made at the beginning, the patient might end up with metastatic colonic cancer and will be confused with GIST recurrence and metastasis. Therefore, it is imperative to consider the possibility of synchronous malignant lesions.

Surgery is the primary treatment modality for both non-metastatic or resectable metastatic GISTs and colon cancers (19).

Imatinib, a tyrosine kinase inhibitor of KIT, has been approved for the treatment of unresectable or metastatic GIST. It has also shown clinical benefit in the adjuvant setting. Imatinib is recommended for at least 3 years in patients with a high risk of recurrence after surgery (20, 21).

Adjuvant CAPOX followed by imatinib was given to our patient to address both pathologies.

There was no survival difference in single GIST versus concurrent GIST and another primary tumor. However, the recurrence rate was higher in synchronous GISTs and another primary tumor. The size of a single GIST appears larger than tumors in patients with synchronous tumors (22). In contrast, our patient had a large GIST with colonic adenocarcinoma. Our patient was followed for six months, and he is now in good health.

Conclusion

The synchronous co-existence of GISTs and other primary tumors is more common than it has been considered to be rare. In our case, colonic cancer was found before the patient underwent surgical therapy for an initially diagnosed gastric GIST. This presentation of GIST with synchronous colorectal adenocarcinoma can be a learning point so that physicians should be aware of the coexistence of malignancies, evaluating patients more thoroughly to have a better prognosis. Further studies are necessary to know the genetic and molecular mechanisms of carcinogenesis and the progression association of GIST and other synchronous tumors.

Abbreviations;

AFIP: Armed Forces Institute of Pathology, CRC: Colorectal cancer, CT scan: Computed tomography, ECOG: Eastern Cooperative Oncology Group, GI: Gastrointestinal, GIST: Gastrointestinal stromal tumor , IHC: Immunohistochemistry, IOF: intraoperative finding, NIH: National Institutes of Health.

Acknowledgements: Not applicable

Authors' contributions: YG and MK designed the report, collected and assembled the patient data. YG, MK and AM, TJ and AAY wrote the paper. All authors read and approved the final manuscript.

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