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Research Article

# Synchronous GIST and Adenocarcinoma: A Literature Review

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**Abstract:**

Gastrointestinal stromal tumors (GISTs) are uncommon mesenchymal neoplasms arising from the interstitial cells of Cajal, accounting for 0.5–1% of gastric neoplasms, of which only 1–3% are malignant. Gastric adenocarcinoma, in contrast, is a well-characterized epithelial malignancy with extensively documented risk factors. The synchronous occurrence of both tumors within the same stomach is rare and poorly understood, with fewer than two dozen cases individually described in the literature to date. This review synthesizes the available evidence on synchronous gastric GIST and adenocarcinoma, examining their epidemiology, clinical presentation, diagnostic workup — including imaging, endoscopy, endoscopic ultrasound, and immunohistochemistry — and therapeutic approach, using a well-documented illustrative case from the literature to anchor the discussion. Current studies have not identified shared risk factors between the two neoplasms, yet the increasing number of reported synchronous cases suggests the possibility of shared carcinogenic mechanisms acting on histologically distinct cell populations within the same organ. Given the rarity of this association, heightened clinical suspicion and systematic use of immunohistochemical techniques are essential for accurate diagnosis and management, which may ultimately improve patient outcomes and survival.

**Key words:** gastrointestinal stromal tumors; synchronous GIST; adenocarcinoma

## 1.Introduction

Gastrointestinal stromal tumors (GISTs) are uncommon neoplasms originating from the mesenchymal muscularis propria, specifically from the interstitial cells of Cajal [2]. Although rare, GISTs may arise anywhere along the gastrointestinal tract, with the stomach being the most common site, accounting for 0.5–1% of gastric neoplasms, of which 1–3% are malignant [2,3]. Most GISTs are associated with activating mutations in the C-KIT proto-oncogene, and immunohistochemical positivity for CD117 (KIT) and/or DOG1 is now considered the diagnostic standard, identified in over 95% and 99% of cases, respectively [3].

Gastric adenocarcinoma, by contrast, is far better characterized in terms of risk factors and predisposing conditions, including Helicobacter pylori infection, chronic atrophic gastritis, and dietary factors, with extensive documentation in the literature [9]. However, the concurrent occurrence of GIST and gastric adenocarcinoma within the same organ is rare and infrequently reported. Fewer than two dozen such cases have been individually described worldwide since the first reports in the early 2000s [1,4,5,6,10,11], and synchronous neoplasms of any type are estimated to represent approximately 6% of all cancer diagnoses [7], making the co-occurrence of these two specific gastric tumors a genuine clinical challenge.

This review aims to synthesize the available literature on synchronous gastric GIST and adenocarcinoma, examining reported clinical presentations, diagnostic strategies, and therapeutic approaches, and to discuss the proposed etiopathogenic mechanisms underlying this rare association.

## 2. Methods

A literature review was conducted using the Medline/PubMed, SciELO, and Cochrane databases, covering publications from 2000 to November 2023, using the search terms “synchronous gastric GIST and adenocarcinoma.” Case reports, case series, and narrative reviews addressing the synchronous or collision occurrence of gastric GIST and adenocarcinoma were considered. A well-documented case originally published in 2007 by Ramos et al. [1] was selected as an illustrative example to anchor the discussion of clinical presentation and diagnostic workup, given the level of detail available regarding imaging, endoscopic, and histopathological findings.

## 3.Results and Discussion

### 3.1 Epidemiology and Clinical Presentation

GISTs are frequently asymptomatic and are often diagnosed incidentally. As the tumor enlarges, clinical manifestations may include abdominal

pain, a palpable abdominal mass, dyspepsia, and weight loss [8]. Gastric adenocarcinoma, when symptomatic, more commonly presents with early satiety, epigastric pain, weight loss, and vomiting [9]. Because the two tumors can produce overlapping or discordant symptom patterns, one lesion may clinically mask or delay recognition of the other — a pattern illustrated in the case reported by Ramos et al. [1], in which a 61-year-old woman presented with two years of chronic abdominal pain and a palpable 10-cm upper abdominal mass, symptoms most consistent with the GIST component, while a smaller synchronous adenocarcinoma was identified only secondarily during endoscopic workup.

### 3.2 Diagnostic Approach: Imaging, Endoscopy, and Endosonography

In the illustrative case, computed tomography demonstrated a mass in contact with the gastric curvature, without evidence of regional lymphadenopathy. Upper endoscopy identified a single 2-cm mucosal lesion on the lesser gastric curvature, and biopsy of this lesion confirmed adenocarcinoma. Endoscopic ultrasound subsequently demonstrated that this lesion was restricted to the muscular layer, and additionally identified a second, previously unrecognized lesion on the greater curvature, originating from the fourth layer of the gastric wall (muscularis propria) [1]. This sequence underscores a broader diagnostic principle observed across the reviewed literature: a single lesion identified on initial endoscopic biopsy does not exclude a second, synchronous tumor of a different histological type, and endoscopic ultrasound evaluation of the entire gastric wall is often essential to detect deeper, submucosal GISTs that would not be apparent on standard mucosal biopsy alone [4,5].

### 3.4 Histopathological Diagnosis and Immunohistochemistry

Histopathological analysis of the greater-curvature lesion in the illustrative case confirmed a high-grade GIST, synchronous with the lesser-curvature adenocarcinoma [1]. Across the reviewed literature, accurate histopathological distinction between the two entities relies on immunohistochemistry: GIST is characteristically positive for CD117 (KIT) and/or DOG1, with combined positivity considered highly specific and identified in the large majority of cases, whereas adenocarcinoma is negative for these markers and instead shows epithelial differentiation markers such as cytokeratins [3]. The systematic use of this immunohistochemical panel may facilitate the recognition of additional synchronous cases and help determine whether a true biological association exists or whether the coincidence of two tumors represents an incidental finding [10].

### 3.5 Therapeutic Approach

Following diagnosis and staging, the patient in the illustrative case underwent total gastrectomy with D2 lymphadenectomy and Roux-en-Y esophagojejunal anastomosis [1]. Postoperative follow-up data and long-term oncological outcomes were not detailed in the original publication. Across the reviewed cases, surgical approach has varied according to tumor location, size, and extent, ranging from subtotal gastrectomy with Billroth-II reconstruction to total gastrectomy with splenectomy or laparoscopic distal gastrectomy [4,5,6], with treatment decisions generally guided by the adenocarcinoma staging while accounting for the coexisting GIST during resection planning.

### 3.6 Comparative Summary of Reported Cases

Table 1 summarizes the illustrative case alongside comparable reports of synchronous gastric adenocarcinoma and GIST identified in the literature review, including differences in tumor location, GIST risk category, surgical approach, and reported outcomes.

| Reference | Age/Sex | Adenocarcinoma | GIST site/risk | Treatment | Outcome |
|---|---|---|---|---|---|
| Ramos et al., 2007 [1] | 61 / F | Lesser curvature, 2 cm mucosal lesion | Greater curvature, 4th wall layer, high-grade | Total gastrectomy, D2 lymphadenectomy, Roux-en-Y | Not reported in original publication |
| Katsoulis et al., 2007 [4] | 78 / F | Proximal gastric body | Distal antrum, collision tumor, | D2 total gastrectomy with | Not specified |
| Theodosopou los et al., 2011 [5] | 80 / M | Antrum, lesser curvature, 4×6 cm | Lesser curvature, 3 cm proximal to primary lesion, 6.5 | Subtotal gastrectomy, Billroth-II | No lymph node metastasis |
| Ojima et al., 2022 [6] | 71 / M | Concurrent with DLBCL and GIST (triple synchronous) | Concurrent lesion | Laparoscopic distal gastrectomy | 72-month recurrence-free survival |

**Table 1:** Comparative summary of reported cases of synchronous gastric adenocarcinoma and GIST in the literature

The reported incidence of synchronous GIST and gastric adenocarcinoma has been increasing over the past two decades, though it remains a rare event overall [1,4,5,6]. Current studies have not identified shared risk factors between the two neoplasms, and no consistent link to Helicobacter pylori status, smoking, or dietary exposure has been established across reported cases [6].

### 3.6 Proposed Etiopathogenesis

Despite the absence of conclusive research on the subject, the rising number of reported synchronous cases suggests the possibility of shared carcinogenic factors acting on adjacent but histologically distinct cell populations within the same organ [1,12]. The most widely accepted etiopathogenic hypothesis remains that a single carcinogenic agent may act upon neighboring but histologically distinct tissues — the gastric mucosa and the interstitial cells of Cajal — inducing the independent development of tumors of different histopathological types within the same organ [12]. This hypothesis, first proposed in the context of independent early cancer and leiomyosarcoma coexistence, has since been extended to GIST-adenocarcinoma pairs, though it remains supported mainly by observational theories and experimental models rather than confirmed molecular mechanisms [12].

### 3.7 Critical Contributions and Clinical Recommendations

This review reinforces that clinicians evaluating gastric masses should not assume a single unifying diagnosis once one lesion is histologically confirmed. Endoscopic ultrasound should be considered as a routine complementary step whenever a palpable or radiologically evident gastric mass is disproportionately large relative to, or discordant in location with, the lesion identified on standard endoscopic biopsy — as this discrepancy was the key diagnostic clue in the illustrative case reviewed here. When a second lesion is identified, a full immunohistochemical panel (including CD117 and DOG1) should be obtained before finalizing the surgical plan,

since the coexistence of GIST may influence the extent of resection, the sequencing of any neoadjuvant or adjuvant systemic therapy (e.g., tyrosine kinase inhibitors for GIST versus chemotherapy for adenocarcinoma), and postoperative surveillance strategy.

### 3.8 Limitations

This review is limited by the small number of published cases of synchronous gastric GIST and adenocarcinoma, the heterogeneity of reporting standards across them, and the likelihood of publication bias favoring unusual or instructive presentations. The illustrative case anchoring this discussion was originally described in 2007, and several data points relevant to a modern reporting standard — including molecular mutation analysis (KIT/PDGFRA), detailed immunohistochemical panel results, formal GIST risk stratification (e.g., Fletcher/Miettinen criteria), and long-term postoperative follow-up — were not available in the original publication. As such, the conclusions drawn here should be interpreted as hypothesis-generating rather than definitive evidence of a shared etiopathogenic mechanism.

## 4. Conclusion

Synchronous gastric GIST and adenocarcinoma is a rare and likely underdiagnosed entity whose etiopathogenesis remains unclear. Clinicians should maintain a high index of suspicion for coexisting neoplasms during the endoscopic and histopathological workup of gastric masses, as a single lesion identified on initial biopsy does not exclude a second, synchronous tumor.

Systematic use of immunohistochemistry and thorough endoscopic ultrasound evaluation of the entire gastric wall may improve detection rates and guide individualized, combined therapeutic strategies, ultimately contributing to better prognosis and survival for affected patients.

## Declarations

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**Informed Consent:** Written informed consent was obtained from the patient (or legal representative) for the publication of this case report and any accompanying images. [Confirm/attach documentation before submission.]

**Ethical Approval:** Not applicable / institutional review as required. [Confirm institutional requirement before submission.]

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