

Original Article

Clinicopathological Features and Treatment Outcomes for Colorectal Gastrointestinal Stromal Tumor

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Key Words

Colorectal gastrointestinal stromal tumor;

Tyrosine kinase inhibitor;

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Purpose. Lower gastrointestinal tract gastrointestinal stromal tumors, including those originating in the appendix, colon, and rectum, are rare and remain poorly characterized. This study aimed to investigate their clinicopathological features, treatment approaches, and outcomes over a 20-year period using a single-center database.

Methods. A retrospective review was performed using a prospective pathology database at National Taiwan University Hospital. Patients diagnosed with lower gastrointestinal tract gastrointestinal stromal tumors between 2004 and July 2023 were included. Clinical and pathological variables were collected, and outcomes were assessed with Cox regression analysis. Follow-up continued until December 2024.

Results. Thirty patients were identified. The median age was 61.5 years, and the median tumor size was 6.3 cm. Tumor sites included the appendix (n = 2), colon (n = 7), and rectum (n = 21). Twenty-seven patients underwent surgery; four received neoadjuvant tyrosine kinase inhibitor therapy, and 15 received adjuvant therapy. Among the nine patients who underwent lymph node sampling, none had nodal metastasis. Eight patients developed recurrence, all with tumors classified as intermediate or high risk by National Institutes of Health criteria. However, this was not statistically significant for recurrence-free survival ($p > 0.99$). Six patients died, including one from a non-gastrointestinal stromal tumor-related cause. Patients who required neoadjuvant therapy had poorer overall survival (hazard ratio = 1.16, $p = 0.04$).

Conclusions. Lower gastrointestinal tract gastrointestinal stromal tumors demonstrate distinct clinical features. Existing risk stratification systems may not fully reflect their prognostic heterogeneity. Multicenter studies are needed to refine management strategies for these rare subgroups.

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Gastrointestinal stromal tumors (GISTs) are the most common mesenchymal neoplasms of the gastrointestinal (GI) tract, arising from the interstitial cells of Cajal or their precursors. These tumors are usually driven by activating mutations in the *KIT* or

PDGFRA genes and are immunohistochemically characterized by expression of CD117 (KIT), DOG1, and frequently CD34.¹

Although GISTs most commonly develop in the stomach (approximately 60%) and small intestine

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(around 30%), their occurrence in the lower GI tract, including the colon, rectum, and appendix, is rare, accounting for only about 5% of cases.² Within colonic locations, the sigmoid colon appears to be the most frequently involved segment. Notably, lower GI GISTs have been reported to display more aggressive biological behavior than gastric GISTs, with a higher proportion classified as high-risk and associated with poorer disease-specific survival.³

The clinical presentation of lower GI GISTs is heterogeneous, ranging from abdominal pain, gastrointestinal bleeding, or bowel obstruction to incidental detection during imaging, endoscopy, or surgery.^{4,5} Colonic GISTs are often transmural and may be complicated by perforation or hemorrhage.¹ Accurate diagnosis and preoperative assessment generally depend on imaging modalities such as computed tomography, magnetic resonance imaging, and, when feasible, endoscopic ultrasound (EUS).²

Risk stratification of GISTs is primarily determined by tumor location, size, and mitotic rate, with mitotic rate serving as the most significant independent predictor of prognosis, particularly in colonic GISTs.^{3,6} The gold standard of treatment is complete surgical resection with negative margins. For tumors classified as high risk, unresectable, or anatomically complex, neoadjuvant or adjuvant therapy with tyrosine kinase inhibitors (TKIs), such as imatinib, is used to improve outcomes.^{7,8} Previous studies have demonstrated that surgical intervention is associated with improved cancer-specific and overall survival in colonic GISTs.⁹

Given their rarity and distinct clinicopathological features, lower GI GISTs require a multidisciplinary management approach. In this study, we present a 20-year single-institution case-control analysis evaluating the clinicopathological characteristics and treatment outcomes of colorectal GISTs.

Materials and Methods

Study design and patient selection

This retrospective study was conducted using a prospectively maintained pathology database at National

Taiwan University Hospital. Patients with a histopathological diagnosis of GIST arising from the lower intestinal tract (appendix, colon, or rectum) between January 2004 and July 2023 were identified. Clinical follow-up was extended through December 2024. Relevant patient data, including demographic characteristics, clinical presentation, imaging findings, operative details, and histopathological features, were retrieved from electronic medical records and systematically reviewed.

Data collection and variables

Clinical and pathological variables collected included age, sex, tumor location (appendix, colon, or rectum), tumor size, mitotic rate (≤ 5 vs. > 5 per 50 high-power fields [HPF]), operative method (anatomical resection vs. tumor excision), pathological subtype (spindle, epithelioid, or mixed), presence of necrosis, margin status, lymph node metastasis, and risk classification according to the National Institutes of Health (NIH) consensus criteria (very low, low, intermediate, or high risk).

Statistical analysis

Continuous variables, including age and tumor size, were summarized as median with interquartile range (Q1-Q3) and compared between groups using the Mann-Whitney U test. Categorical variables were analyzed using Fisher's exact test.

Univariate analyses of potential prognostic factors for disease-free survival (DFS) and overall survival (OS) were performed with the Cox proportional hazards regression model. Variables with a p -value < 0.5 in univariate analysis were subsequently included in the multivariate analysis to identify independent predictors.

All statistical tests were two-tailed, and a p -value < 0.05 was considered statistically significant. Statistical analyses were performed using GraphPad Prism version 10.4.1 (GraphPad Software, LLC, CA, USA).

Results

A total of 43 patients were initially identified.

Thirteen were excluded owing to either a primary tumor origin outside the lower gastrointestinal tract (e.g., small bowel or mesentery with secondary involvement of the lower GI tract) or insufficient clinical data to establish the primary site. Ultimately, 30 patients with primary GISTs arising from the lower GI tract were included in the analysis.

The median age at diagnosis was 61.5 years, and the median tumor size was 6.3 cm. Tumor locations included the appendix ($n = 2$), colon ($n = 7$), and rectum ($n = 21$). Twenty-seven patients underwent surgical intervention, whereas three patients received only an endoscopic biopsy for pathological confirmation.

Among the 27 surgically treated patients, four received neoadjuvant TKI therapy with imatinib (Glivec), and 15 received adjuvant TKI therapy. All three patients who did not undergo surgery received systemic therapy with either TKI or chemotherapy.

Of the 21 patients with rectal GIST, 11 underwent local tumor excision, while 7 underwent anatomical resections, including low anterior resection, total mesorectal excision, intersphincteric resection, or abdominoperineal resection. Among the 9 patients with colonic or appendiceal GIST, 8 underwent anatomical resection and 1 underwent local excision. Lymph node dissection was performed in 9 patients, none of whom had nodal metastasis. Detailed patient characteristics are summarized in Table 1.

At the end of the follow-up period, 8 patients experienced tumor recurrence, either local or distant. The median follow-up duration was 98 months (range: 43-150.5 months), with the longest follow-up extending to 203 months. Six patients died during the study period, including one patient whose death was attributed to colon cancer, unrelated to GIST.

Comparison between patients with and without recurrence is summarized in Table 2. No significant differences were observed in clinical or pathological variables between the two groups, except for NIH risk classification. All 8 patients who developed recurrence had primary tumors classified as intermediate or high risk. In contrast, among the 17 patients without recurrence, only 9 (52.9%) were intermediate or high risk, while all 8 patients with very low or low risk tumors remained recurrence-free.

Univariate and multivariate Cox regression analyses were conducted to evaluate predictors of recurrence-free survival (RFS) and OS. No independent risk factor for RFS was identified. However, for OS, neoadjuvant TKI therapy was associated with worse prognosis (hazard ratio = 1.16, $p = 0.04$; Table 4).

Subgroup analysis compared patients with rectal GISTs to those with colonic or appendiceal tumors. Patients in the colonic/appendiceal group were more likely to undergo anatomical resection and had a higher

Table 1. Patient characteristics

Characteristics	n (%) or median (Q1-Q3)
Sex (male)	21 (70%)
Age (years, median)	61.5 (55.1-69.1)
Size (cm, median)	6.3 (4.6-8.0)
Site	
Appendix	2 (7%)
Colon (cecum, A, T, S-colon)	7 (1, 2, 3, 1) (23%)
Rectum	21 (70%)
Mitosis	
> 5	14 (47%)
≤ 5	16 (53%)
Morphology	
Spindle	29 (97%)
Epithelioid	0 (0%)
Mixed	1 (3%)
NIH classification	
Very low risk	5 (17%)
Low risk	4 (13%)
Moderate risk	9 (30%)
High risk	12 (40%)
Necrosis	
Present	4 (14%)
Absent	24 (86%)
Margin	
Involved	4 (17%)
Free	19 (83%)
Lymph node	
No metastasis	9 (30%)
No LN dissection	21 (70%)
Neoadjuvant therapy	4 (13%)
Adjuvant therapy	18 (60%)
Recurrence	8 (31%)
Local	6 (75%)
Distant metastasis	4 (50%)
RFS (month, median)	78.0 (16.0-123.0)
OS (month, median)	98.0 (43.0-150.5)

NIH: National Institutes of Health; LN: lymph node; RFS: recurrence-free survival; OS: overall survival.

Table 2. Comparison of clinicopathological characteristics between recurrence and non-recurrence groups

	No recurrence (n = 17)	Recurrence (n = 8)	HR	p-value
Sex (male)	12 (70.6%)	5 (62.5%)		> 0.9999
Age (years)	67.47	57.95		0.2380
Size (cm)	3.50	6.95		0.1520
Site (colon/appendix)	7 (41.2%)	2 (25%)		0.6608
OP method (anatomical)	10 (58.8%)	4 (50%)		> 0.9999
Mitosis > 5	6 (35.3%)	6 (75.0%)		0.0968
Morphology				> 0.9999
Spindle	16	8		
Mixed	1	0		
NIH 3 or 4	9 (52.9%)	8 (100%)		0.0261
Necrosis present	2 (12.5%)	1 (12.5%)		> 0.9999
Margin involved	3 (20.0%)	4 (57.1%)		0.1447
LN metastasis	0/5 (27.8%)	0/3 (37.5%)		> 0.9999
Neoadjuvant TKI	3 (17.7%)	0 (0.00%)		0.5270
Adjuvant TKI before recurrence [†]	7 (41.2%)	4 (50.0%)		> 0.9999
Adjuvant TKI overall [‡]	7 (41.2%)	8 (100%)		0.0077
RFS (month)	78.0 (24.0-114.0)	33.5 (13.5-125.3)	22.2 (5.02-98.22)	< 0.0001
OSm (month)	78.0 (24.0-114.0)	171.0 (140.0-184.75)	0.54 (0.05-5.84)	0.2601

NIH: National Institutes of Health; LN: lymph node; TKI: tyrosine kinase inhibitor, HR: hazard ratio, RFS: recurrence-free survival, OS: overall survival.

[†] Patients with adjuvant TKI before recurrence. [‡] All patients with adjuvant TKI.

Table 3. Univariate and multivariate analysis of recurrence-free survival

Variable	Univariate analysis			Cox regression		
	HR	OR (95% CI)	p-value	HR	HR (95% CI)	p-value
Female	1.34	0.27-5.51	0.6871			
Age	1.00	0.94-1.07	0.9694			
Site: colon/appendix	0.55	0.08-2.52	0.4825	1.53	0.12-16.13	0.7178
Anatomical surgery	0.46	0.09-2.13	0.3147	0.07	0.00-1.55	0.1222
Mitosis > 5	3.68	0.84-25.33	0.1132	4.09	0.53-85.40	0.2310
NIH 3 or 4	> 1000	NA	> 0.9999			
Necrosis	3.27	0.16-26.02	0.3091	0.12	0.00-21.67	0.4464
Margin involved	3.21	0.44-16.77	0.1821	1.47	0.12-16.37	0.7429
Size	1.12	0.95-1.28	0.1182	1.40	0.96-2.29	0.1119
Neoadjuvant TKI	0.00	NA	> 0.9999			
TKI [†]	1.00	0.97-1.01	0.7690			

HR: hazard ratio, OR: odds ratio, CI: confidence interval, NIH: National Institutes of Health, TKI: tyrosine kinase inhibitor.

[†] Patients with adjuvant TKI before recurrence.

Table 4. Univariate and multivariate analysis of overall survival

Variable	Univariate analysis			Cox regression		
	HR	OR (95% CI)	p-value	HR	HR (95% CI)	p-value
Female	0.00	0.00-0.76	> 0.9999			
Age	1.13	1.04-1.25	0.0077	1.26	1.05-1.79	0.0578
Site: colon/appendix	1.08	0.15-5.60	0.9282			
Surgery	7.70	1.00-47.61	0.0275	10.54	0.04-> 1000	0.4962
Anatomical surgery	> 1000	1.36-NA	> 0.9999			
Mitosis > 5	0.85	0.16-4.61	0.8406			
NIH 3 or 4	1.10	0.16-21.70	0.9319			
Necrosis	10.35	1.18-91.02	0.0239	16.80	0.00-> 1000	0.5740
Margin involved	0.00	NA-4.56	> 0.9999			
Size	1.10	0.95-1.23	0.1270	1.00	0.61-1.64	0.9997
Neoadjuvant TKI	1.05	0.97-1.11	0.0946	1.16	1.04-1.48	0.0426
TKI [‡]	1.00	0.98-1.01	0.9109			

HR: hazard ratio; CI: confidence interval; NIH: National Institutes of Health; TKI: tyrosine kinase inhibitor.

[‡] All patients with adjuvant TKI.

rate of margin-negative resections. This was accompanied by a lower recurrence rate among patients with colonic/appendiceal GISTs, although the difference did not reach statistical significance. There were no significant differences in tumor size, NIH classification, RFS, or OS between the two groups. (Table 5). Kaplan-Meier survival curves for RFS and OS are shown in Figs. 1 and 2, respectively, demonstrating no statistically significant difference between the groups.

Discussion

Gastrointestinal stromal tumors (GISTs) arise from

the interstitial cells of Cajal and may occur throughout the gastrointestinal tract. While most cases involve the stomach and small intestine, GISTs of the lower gastrointestinal (GI) tract, including the appendix, colon, and rectum, are relatively rare. Among these, colonic GISTs are particularly uncommon, accounting for only 2-9% of all GISTs.^{10,11} Despite advances in molecular characterization and targeted therapy, the clinicopathological features and prognostic factors specific to lower GI GISTs remain poorly defined.

The most widely used risk stratification systems for GISTs, including the NIH consensus criteria and the Joensuu modification, classify recurrence risk according to tumor size, mitotic index, and anatomical

Table 5. Comparison of clinicopathological characteristics between colonic/appendiceal and rectal GISTs

	Colon/appendix (n = 9)	Rectum (n = 21)	HR	p-value
Sex (male)	7 (77.8%)	14 (66.7%)		0.6814
Age (years)	67.75	66.45		0.8590
Size (cm)	5.50	6.00		0.5256
OP method (anatomical)	8 (88.9%)	7 (38.9%)		0.0192
Mitosis > 5	4 (44.4%)	10 (47.6%)		> 0.9999
Morphology				0.3000
Spindle	8	21		
Mixed	1	0		
NIH 3 or 4	5 (55.6%)	16 (76.2%)		0.3888
Necrosis present	2 (22.2%)	2 (10.5%)		0.5741
Margin involved	0 (0.00%)	7 (50.0%)		0.0189
LN metastasis	0/5 (55.6%)	0/4 (19.0%)		0.0816
Recurrence	2/9 (22.2%)	6/16 (37.5%)		0.6608
Neoadjuvant TKI	0 (0.00%)	4 (19.1%)		0.2874
TKI treatment overall	4 (44.4%)	15 (71.4%)		0.2252
RFS (month)	39.0 (15.0-105.0)	82.0 (23.25-123.0)	0.57 (0.14-2.41)	0.4773
OS (month)	96.0 (39.0-178.0)	100.0 (55.0-149.0)	1.46 (0.23-9.15)	0.6598

NIH: National Institutes of Health; LN: lymph node; TKI: Tyrosine kinase inhibitor; RFS: recurrence-free survival; OS: overall survival.

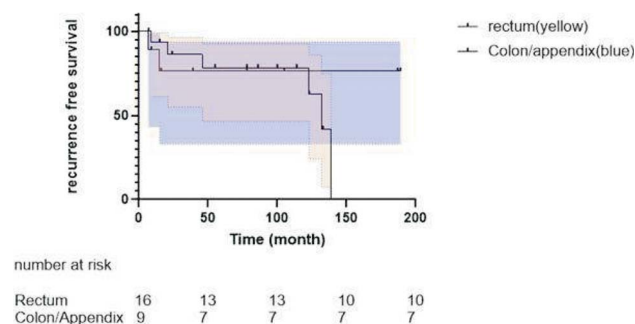


Fig. 1. Kaplan-Meier curve of recurrence-free survival in colonic/appendiceal vs. rectal GISTs.

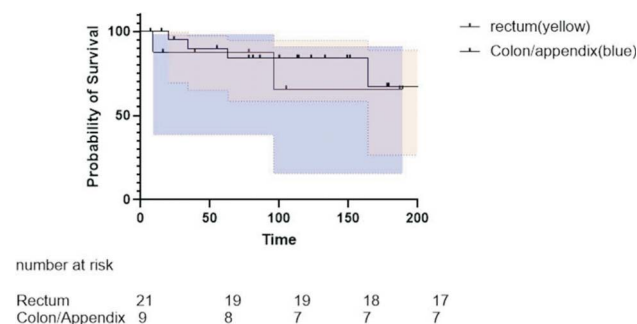


Fig. 2. Kaplan-Meier curve of overall survival in colonic/appendiceal vs. rectal GISTs.

location. However, these systems categorize tumors broadly as gastric versus non-gastric, without specifically distinguishing lower GI GISTs. The MSKCC nomogram and the AFIP-Miettinen classification do recognize rectal GISTs as a distinct subgroup, and the latter further subdivides small bowel tumors into duodenal and jejunal/ileal origins. Nevertheless, a dedicated risk stratification model for colonic GISTs is lacking, most likely because of their rarity and the limited availability of robust clinical data.

Previous studies have demonstrated poorer DFS in patients with colonic or rectal GISTs compared with gastric GISTs. Kukar et al. and Liu et al. reported a significantly higher risk of recurrence for colonic GISTs (HR = 2.24, $p = 0.016$).^{8,9} However, other studies, such as that by Li et al. using propensity score matching, found no significant difference in DFS or OS between colonic and rectal GISTs.¹¹

In our cohort, appendiceal and colonic GISTs accounted for 30% of all lower GI cases, consistent with prior reports. Spindle cell morphology predominated, and only one case exhibited a mixed histological type. About half of the patients underwent anatomical resection, and of those, approximately 64% had lymph node sampling, but none showed nodal metastasis, consistent with the rarity of lymphatic spread in GISTs.

Neoadjuvant TKI therapy was administered in four patients, all with rectal GISTs, using imatinib (Glivec) as first-line treatment. One patient was switched to sunitinib (Sutent) as a second-line neoadjuvant TKI due to progressive disease. Tumor size reduction ranged from 23% to 38% after 3-12 months of therapy. Fifteen patients received adjuvant TKI therapy postoperatively, also initiated with imatinib, with some transitioning to sunitinib or regorafenib as required. Among these, 11 patients had rectal GISTs, while the remaining 4 had colonic or appendiceal GISTs. Decisions regarding neoadjuvant and adjuvant therapy were individualized based on clinical judgment, reflecting real-world practice. This likely contributed to the poorer prognosis observed in patients who received neoadjuvant TKI therapy, which may reflect the selection of individuals with more advanced disease.

When comparing recurrence and non-recurrence groups, individual factors such as tumor size, mitotic

index, necrosis, and margin status did not significantly differ. However, all patients with recurrence were classified as intermediate or high risk by NIH criteria, whereas 47.1% of patients without recurrence were similarly classified. All eight patients with very low or low-risk tumors remained recurrence-free. Despite this trend, univariate analysis did not identify NIH classification as a statistically significant predictor of RFS ($p > 0.99$), most likely due to limited sample size. No similar trend was observed for OS. Among the 8 patients who experienced recurrence, 6 developed local recurrence. All 6 patients underwent surgical resection for the recurrent tumors. Four of these patients also developed distant metastases during follow-up, involving the lungs, bone, liver, peritoneum, and chest wall. Only one patient with peritoneal metastasis underwent peritonectomy along with excision of the locally recurrent tumor. The remaining 3 patients had their metastatic disease controlled with adjuvant TKI therapy.

This study has some limitations. First, the 20-year study period encompassed evolving diagnostic and treatment paradigms, which may have introduced heterogeneity in clinical management. Second, mutational analyses for *KIT*, *PDGFRA*, and other relevant genes were not consistently performed, particularly in earlier cases, limiting molecular correlation. Third, the small sample size may have restricted statistical power to detect meaningful associations and increased the risk of both type I and type II errors. Nevertheless, this study provides valuable insight into a rare subgroup of GISTs through a 20-year single-center experience. Future multicenter or prospective studies with larger cohorts are required to validate these findings and further refine treatment strategies.

Conclusion

This study highlights the distinct clinicopathological features and treatment patterns of lower GI GISTs. While current risk stratification systems provide general guidance, they may not fully capture the heterogeneity and unique clinical behavior of these rare subtypes, particularly those arising in the colon and ap-

pendix. Our findings emphasize the need for multi-center collaboration and prospective studies to enhance understanding of prognosis, refine risk assessment, and guide therapeutic strategies for lower GI GISTs.

Declarations of Interest

None.

Financial Disclosure

None to declare.

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原 著

大腸直腸基質瘤的臨床病理表徵及治療成果

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目的 下消化道的胃腸道基質瘤，包括起源於闌尾、結腸及直腸的病灶，較罕見且其特徵尚未被充分了解。本研究旨在分析這類腫瘤在單一中心 20 年間的臨床病理特徵、治療模式及預後情形。

方法 本研究回溯性分析 2004 年至 2023 年 7 月間，台大醫院病理資料庫中診斷為下消化道胃腸道基質瘤的病人。收集臨床與病理資料，追蹤至 2024 年 12 月，並使用 Cox 比例風險模型進行預後分析。

結果 共納入 30 位病人，年齡中位數為 61.5 歲，腫瘤大小中位數為 6.3 公分。腫瘤發生部位為闌尾 (2 例)、結腸 (7 例) 及直腸 (21 例)。其中 27 位接受手術治療；4 位術前接受酪胺酸激酶抑制劑新輔助治療，15 位術後接受輔助治療。9 位進行淋巴結取樣者皆未發現轉移。共 8 位病人發生復發，皆為 NIH 分級中或高度風險，但無病生存期分析中未達顯著差異 ($p > 0.99$)。共 6 位病人死亡，其中 1 位與 GIST 無關。需要接受前導性輔助治療的病人與較差的總生存期相關 ($HR = 1.16, p = 0.04$)。

結論 下消化道胃腸道基質瘤具有獨特的臨床特性。現行風險分級系統可能無法完整評估其預後差異，需進一步多中心研究以優化此類罕見亞型的治療策略。

關鍵詞 大腸直腸基質瘤、酪胺酸激酶抑制劑、風險分級、大腸直腸腫瘤。