

Delayed Gastric Emptying After Laparoscopic Left Hemicolectomy: Incidence, Clinical Course, and Operative-Field Extension

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Abstract

Background Delayed gastric emptying (DGE) after left hemicolectomy is uncommon and may be confused with diffuse postoperative ileus, fixed mechanical obstruction at the Treitz region, or an occult local functional disturbance. We characterised its incidence and clinical course and examined associations with tumour-location-related extension of the operative field and pathological features.

Methods This single-centre retrospective cohort included consecutive adults who underwent laparoscopic left hemicolectomy between August 2019 and June 2026. DGE required persistent upper gastrointestinal symptoms, intolerance of oral intake, or a need for gastric decompression, together with computed-tomography exclusion of fixed mechanical obstruction and upper gastrointestinal contrast evidence of delayed gastric emptying. The proximal–extended operative-field phenotype comprised a tumour in the distal transverse colon near the splenic flexure, at the splenic flexure, or in the middle/upper descending colon, together with splenic-flexure mobilisation, inferior mesenteric vein root/high ligation, and Treitz-adjacent dissection. Fisher’s exact test, Wilson confidence intervals, and Firth penalised-likelihood logistic regression were used. Pathological and molecular analyses were exploratory.

Results Among 379 eligible patients, 9 developed DGE (2.37%; 95% CI, 1.25%–4.45%); the median duration was 24 days (interquartile range, 18–26 days). All 9 events occurred in the proximal–extended operative-field group: 3.69% (9/244) versus 0% (0/135), yielding an absolute risk difference of 3.69 percentage points (95% CI, 0.42–6.86; $P = 0.030$). DGE occurred in 8.43% (7/83) of patients with pathological T4 disease and 0.68% (2/296) of those with T0–T3 disease ($P < 0.001$). In a Firth model containing both variables, the proximal–extended operative-field phenotype was associated with DGE (adjusted odds ratio [aOR], 14.75; 95% CI, 1.79–1922.10; $P = 0.007$), but the extremely wide interval indicates substantial uncertainty in the magnitude of this association. Pathological T4 stage was also associated with DGE (aOR, 14.01; 95% CI, 3.59–77.11; $P < 0.001$).

Conclusions DGE after left hemicolectomy was uncommon and occurred only among patients with an extended operative field. The imprecise model estimate requires validation in larger cohorts and should not be interpreted as an independent causal effect of a single manoeuvre. Pathological T4 stage may mark local tumour burden and operative complexity rather than constitute a direct mechanism. Postoperative gastric retention should be differentiated among mechanical “pseudo-gastroparesis”, occult functional obstruction at the Treitz region, and stomach-predominant DGE.

Introduction

Contemporary definitions of gastroparesis require compatible symptoms, objective evidence of delayed gastric emptying, and exclusion of mechanical obstruction; a 4-h gastric-emptying test is the standardised method for chronic gastroparesis [1–4]. After colorectal surgery, however, gastric retention lies at the intersection of several diagnostic concepts. Stomach-predominant dysmotility, diffuse

postoperative ileus (POI), impaired transit at the duodenojejunal flexure, and overt mechanical obstruction can all cause nausea, vomiting, gastric dilatation, and prolonged nasogastric drainage [5–8].

Most colectomy studies combine passage of flatus or stool and dietary recovery into a composite POI outcome. A systematic review suggested that prolonged POI is more common after right- than left-sided colectomy, although definitions varied substantially [11], and a single-centre study found greater nasogastric-tube use after right-sided surgery [12]. Evidence specifically addressing DGE after left hemicolectomy is sparse; Sarli et al. [13] reported two DGE events among 54 laparoscopic left hemicolectomies for cancer. Its incidence, clinical boundary, and operative anatomical context therefore remain incompletely defined.

Left hemicolectomy has a distinctive anatomical feature. Tumours in the distal transverse colon near the splenic flexure, at the splenic flexure, or in the middle/upper descending colon often require splenic-flexure mobilisation and division of the inferior mesenteric vein (IMV) adjacent to the ligament of Treitz. By contrast, lower descending or descending–sigmoid junction tumours may not require splenic-flexure mobilisation when the sigmoid colon is sufficiently long. A randomised study of Treitz-ligament reconstruction, reports of superior mesenteric artery syndrome after colectomy, and a recent systematic review indicate that displacement, angulation, or compression at the duodenojejunal flexure can produce upper gastrointestinal retention resembling “gastroparesis” [14–17].

Accordingly, we restricted the outcome to an acute postoperative DGE phenotype, described its incidence and course, and examined associations with tumour-location-related operative-field extension, TNM stage, histopathology, and molecular markers. We further propose that between fixed mechanical obstruction and conventional motility-predominant DGE lies a clinically relevant intermediate phenotype: occult functional obstruction at the Treitz region caused jointly by local inflammation, oedema, and mild or intermittent angulation.

Methods

Study design and patients

This single-centre retrospective cohort study included consecutive adults who underwent laparoscopic left hemicolectomy in the Department of Gastrointestinal Surgery, Minhang Hospital, Fudan University, between August 2019 and June 2026. Patients undergoing concomitant gastric, duodenal, or pancreatic resection and those with a recognised preoperative mechanical gastric-outlet or duodenal obstruction were not included. The database was locked and the final analyses were performed in July 2026. A total of 379 patients met the study criteria (Fig. 1).

The study was approved by the Ethics Committee of Minhang Hospital, Fudan University (approval no. 202105301K). Individual informed consent was waived because of the retrospective, de-identified design. Reporting followed the STROBE recommendations [34].

Operative exposure and anatomical classification

Tumour location was classified anatomically as the distal transverse colon near the splenic flexure, splenic flexure, middle/upper descending colon, lower descending colon, or descending–sigmoid junction. Splenic-flexure mobilisation, the IMV ligation level, and Treitz-adjacent dissection were recorded. IMV root ligation was defined as division close to the IMV–splenic-vein confluence with full exposure posterolateral to the ligament of Treitz; high ligation as division at the inferior border of the ligament; and non-high ligation as division more than 2 cm below the inferior border without disturbing the surrounding region.

In this cohort, tumours in the distal transverse colon near the splenic flexure, at the splenic flexure, and in the middle/upper descending colon were uniformly accompanied by splenic-flexure mobilisation, IMV root or high ligation, and Treitz-adjacent dissection. These cases were therefore classified as the proximal–extended operative-field phenotype. Lower descending and descending–sigmoid junction tumours were treated without splenic-flexure mobilisation, with non-high IMV ligation and no Treitz-adjacent dissection, and were classified as non-extended. Because the components coincided completely, they were not entered simultaneously into regression models and no causal effect was assigned to an individual operative manoeuvre.

DGE outcome and competing diagnoses

The primary outcome was an acute postoperative DGE phenotype, defined by persistent upper abdominal distension, nausea or vomiting, intolerance of oral intake, or a need for continued or repeated gastric decompression, together with abdominal computed tomography excluding fixed mechanical obstruction and an upper gastrointestinal contrast study supporting delayed gastric emptying. DGE duration was measured from first fulfilment of these criteria until decompression was no longer required and oral intake had resumed. This postoperative phenotype was not equated with chronic gastroparesis diagnosed by a standardised 4-h gastric-emptying test [1–4].

Imaging review focused on angulation at the ligament of Treitz and the duodenojejunal flexure, a fixed transition point, vascular compression, and proximal dilatation. A definite fixed transition point or vascular compression indicated mechanical “pseudo-gastroparesis”, whereas delayed recovery involving the stomach, small bowel, and colon was more consistent with diffuse POI. When no fixed transition point was present, but local oedema, inflammatory dysmotility, and mild angulation could plausibly reduce dynamic transit at the Treitz region, we considered occult functional obstruction as a testable mechanistic hypothesis rather than assigning a retrospective diagnosis.

Other variables and pathological and molecular assessment

Collected variables included age, sex, body mass index (BMI), type 2 diabetes mellitus, preoperative albumin, previous abdominal surgery, preoperative colonic obstruction, operating time, intraoperative

blood loss, and maximum tumour diameter. Preoperative anxiety was defined as a seven-item Generalized Anxiety Disorder scale (GAD-7) score of at least 10 on the day before surgery [35]. Cumulative net fluid balance over postoperative days 1–7 was classified as net negative by more than 1000 mL, within ± 1000 mL, or net positive by more than 1000 mL. The source dataset contained this cumulative category rather than day-specific input and output; a separate postoperative-day 1–3 exposure could not therefore be reconstructed. Because the 7-day interval overlapped with outcome ascertainment, fluid balance was described exploratorily.

Pathological variables comprised T, N, and M categories; American Joint Committee on Cancer (AJCC) 8th-edition stage [36]; histological type; grade; lymphovascular invasion; and perineural invasion. Molecular-pathology variables comprised mismatch-repair status (deficient or proficient mismatch repair), HER2 immunohistochemistry, and the Ki-67 proliferation index. HER2 was grouped as 0/1 + versus 2+/3+, and Ki-67 as $< 70\%$ versus $\geq 70\%$.

Statistical analysis

Continuous variables are presented as median (interquartile range) and were compared using the Mann–Whitney U test. Categorical variables are presented as n (%) and were compared using Fisher’s exact test. Incidences are accompanied by Wilson 95% confidence intervals (CIs), and absolute risk differences by Newcombe–Wilson intervals. No missing values were imputed. Analyses of variables with missing data used patients with non-missing values for all variables included in that analysis (available-case analysis), with effective sample sizes reported in the Results and table notes; Firth models used complete records for their specified covariates. Firth penalised-likelihood logistic regression was used [37]. A focused model included both the proximal–extended operative-field phenotype and pathological T4 stage; within the extended-field group, the association with T4 stage was assessed with adjustment for age. Two-sided $P < 0.05$ was considered statistically significant. Analyses were performed using R version 4.4.2.

Results

Incidence and clinical course of DGE

The cohort comprised 379 patients, including 242 men (63.9%), with a median age of 69 years. Nine patients developed DGE, corresponding to an incidence of 2.37% (95% CI, 1.25%–4.45%). All 9 underwent abdominal computed tomography and an upper gastrointestinal contrast study on postoperative day 6; no fixed mechanical obstruction was identified on computed tomography, and the contrast study supported delayed gastric emptying. DGE lasted 15–31 days, with a median of 24 days (interquartile range, 18–26 days) (Fig. 2).

Clinical and perioperative characteristics

Age, BMI, operating time, blood loss, preoperative albumin, maximum tumour diameter, sex, type 2 diabetes mellitus, preoperative anxiety, previous abdominal surgery, and preoperative colonic obstruction did not differ between patients with and without DGE. Net-positive fluid balance greater than 1000 mL over postoperative days 1–7 was more frequent in the DGE group (44.4% vs 19.2%; $P = 0.080$), but this variable overlapped temporally with outcome ascertainment and was not interpreted causally (Table 1).

Table 1
Clinical and perioperative characteristics of the study cohort

Variable	Overall (n = 379)	No DGE (n = 370)	DGE (n = 9)	P value
Age, years	69.0 [59.0–76.0]	69.0 [59.0–76.0]	67.0 [65.0–75.0]	0.958
BMI, kg/m ²	23.2 [21.1–25.2]	23.2 [21.2–25.2]	22.1 [20.7–23.9]	0.484
Operating time, min	132.0 [118.5–147.0]	132.0 [118.2–147.0]	136.0 [126.0–146.0]	0.576
Intraoperative blood loss, mL	90.0 [60.0–120.0]	90.0 [60.0–120.0]	100.0 [80.0–120.0]	0.699
Preoperative albumin, g/L	38.0 [33.0–43.0]	38.0 [33.0–43.0]	36.0 [32.0–41.0]	0.582
Maximum tumour diameter, cm	4.5 [3.0–5.5]	4.2 [3.0–5.5]	4.5 [4.0–5.5]	0.419
Male sex	242 (63.9%)	236 (63.8%)	6 (66.7%)	1.000
Type 2 diabetes mellitus	129 (34.0%)	126 (34.1%)	3 (33.3%)	1.000
Preoperative anxiety (GAD-7 ≥ 10)	120 (31.7%)	117 (31.6%)	3 (33.3%)	1.000
Previous abdominal surgery	130 (34.3%)	126 (34.1%)	4 (44.4%)	0.500
Preoperative colonic obstruction	65 (17.2%)	62 (16.8%)	3 (33.3%)	0.188
Net-positive fluid balance > 1000 mL, postoperative days 1–7	75 (19.8%)	71 (19.2%)	4 (44.4%)	0.080

Data are median [interquartile range] or n (%). Continuous variables were compared using the Mann–Whitney U test and categorical variables using Fisher’s exact test. Postoperative fluid balance was an exploratory time-overlapping variable.

Tumour location, extended operative field, and DGE

DGE decreased across progressively distal tumour locations: 4/86 (4.65%) in the distal transverse colon near the splenic flexure, 3/85 (3.53%) at the splenic flexure, 2/73 (2.74%) in the middle/upper descending colon, and no events in either the lower descending colon or descending–sigmoid junction

(overall $P = 0.192$). DGE occurred in 3.69% of the proximal–extended operative-field group (9/244; 95% CI, 1.95%–6.86%) and 0% of the non-extended group (0/135; 95% CI, 0%–2.77%), an absolute risk difference of 3.69 percentage points (95% CI, 0.42–6.86; $P = 0.030$).

Incidences were 5.17% (6/116) after IMV root ligation, 2.34% (3/128) after high ligation, and 0% (0/135) after non-high ligation (overall $P = 0.015$). Because IMV ligation level was tightly coupled to tumour location, splenic-flexure mobilisation, and Treitz-adjacent dissection, this comparison describes an anatomical exposure gradient and does not demonstrate an independent effect of any ligation technique (Table 2; Fig. 3).

Table 2
DGE incidence according to anatomical location and operative exposure

Domain	Stratum	DGE/total	Incidence, % (95% CI)	Overall P
Tumour location	Distal transverse colon near the splenic flexure	4/86	4.65 (1.83–11.35)	0.192
	Splenic flexure	3/85	3.53 (1.21–9.87)	
	Middle/upper descending colon	2/73	2.74 (0.75–9.45)	
	Lower descending colon	0/65	0 (0–5.58)	
	Descending–sigmoid junction	0/70	0 (0–5.20)	
Proximal–extended operative field	Yes	9/244	3.69 (1.95–6.86)	0.030
	No	0/135	0 (0–2.77)	
Splenic-flexure mobilisation	Yes	9/244	3.69 (1.95–6.86)	0.030
	No	0/135	0 (0–2.77)	
IMV ligation level	Root ligation	6/116	5.17 (2.39–10.83)	0.015
	High ligation	3/128	2.34 (0.80–6.66)	
	Non-high ligation	0/135	0 (0–2.77)	
Treitz-adjacent dissection	Yes	9/244	3.69 (1.95–6.86)	0.030
	No	0/135	0 (0–2.77)	

Confidence intervals are Wilson 95% CIs and overall P values are from Fisher's exact test. The proximal-extended operative-field phenotype, splenic-flexure mobilisation, and Treitz-adjacent dissection coincided completely in this cohort and cannot be attributed separately.

TNM stage, histopathology, and molecular markers

DGE occurred in 8.43% of patients with pathological T4 disease (7/83; 95% CI, 4.15%–16.40%) and 0.68% of those with T0–T3 disease (2/296; 95% CI, 0.19%–2.43%; $P < 0.001$). Within the proximal-extended operative-field group, the corresponding rates were 14.58% (7/48) and 1.02% (2/196; $P < 0.001$). Cross-classification showed that 48 of 83 patients with T4 disease (57.8%) and 196 of 296 patients with T0–T3 disease (66.2%) were in the extended-field group; neither variable was nested within the other. Pathological N category, distant metastasis, and AJCC stage were not significantly associated with DGE (Table 3; Fig. 4).

The histological-type analysis included 354 patients and showed DGE incidences of 6.98% (3/43) in mucinous or signet-ring cell carcinoma and 1.93% (6/311) in conventional or other adenocarcinoma ($P = 0.083$). Effective sample sizes were 225 for lymphovascular invasion, 212 for perineural invasion, 351 for mismatch-repair status, 352 for HER2, and 359 for Ki-67. None of these variables showed a clear association with DGE (Table 3; Fig. 5).

Table 3
DGE incidence according to pathological and molecular characteristics

Domain	Stratum	DGE/n	Incidence, % (95% CI)	P value
Pathological T stage	T4	7/83	8.43 (4.15–16.40)	< 0.001
	T0–T3	2/296	0.68 (0.19–2.43)	
Pathological N stage	N-positive	5/154	3.25 (1.39–7.37)	0.495
	N0	4/225	1.78 (0.69–4.48)	
Distant metastasis	M1	2/56	3.57 (0.98–12.12)	0.627
	M0	7/323	2.17 (1.05–4.41)	
AJCC stage	III/IV	5/167	2.99 (1.29–6.82)	0.515
	0–II	4/212	1.89 (0.74–4.75)	
Histological type (N = 354)	Mucinous or signet-ring cell carcinoma	3/43	6.98 (2.40–18.61)	0.083
	Conventional or other adenocarcinoma	6/311	1.93 (0.89–4.14)	
Lymphovascular invasion (N = 225)	Positive	2/76	2.63 (0.72–9.10)	1.000
	Negative	3/149	2.01 (0.69–5.75)	
Perineural invasion (N = 212)	Positive	3/88	3.41 (1.17–9.55)	0.651
	Negative	2/124	1.61 (0.44–5.69)	
MMR status (N = 351)	dMMR	1/18	5.56 (0.99–25.76)	0.381
	pMMR	8/333	2.40 (1.22–4.67)	
HER2 (N = 352)	2+/3+	0/70	0.00 (0.00–5.20)	0.365

Domain	Stratum	DGE/n	Incidence, % (95% CI)	P value
Ki-67 (N = 359)	0/1+	8/282	2.84 (1.44–5.50)	0.679
	≥ 70%	6/251	2.39 (1.10–5.12)	
	< 70%	1/108	0.93 (0.16–5.06)	

Because of variable-specific missingness in this retrospective dataset, category totals may be lower than the full cohort of 379 patients. No missing values were imputed; percentages and Fisher’s exact tests are based on available observations, and effective sample sizes are shown in the domain labels. The histological-type comparison excluded other malignant tumours and cases with no residual tumour after treatment.

Multivariable Firth regression

In the Firth model including the proximal–extended operative-field phenotype and pathological T4 stage, the extended-field phenotype was associated with DGE (aOR 14.75; 95% CI, 1.79–1922.10; P = 0.007), but the extremely wide confidence interval indicates substantial uncertainty in the magnitude of this association. Pathological T4 stage was also associated with DGE (aOR 14.01; 95% CI, 3.59–77.11; P < 0.001). Within the extended-field group, the age-adjusted aOR for pathological T4 stage was 13.98 (95% CI, 3.59–76.81; P < 0.001). Given only 9 events, these estimates are hypothesis-generating and do not establish independent causal effects. Regression estimates are presented in Tables S2 and S3.

Discussion

Principal findings and central interpretation

Among 379 patients undergoing laparoscopic left hemicolectomy, 9 developed DGE, for an incidence of 2.37%, and the median course lasted 24 days. Every event occurred in the proximal–extended operative-field group, and 7 of the 9 events occurred in patients with pathological T4 disease. The joint Firth model yielded associations for both variables, but the small event count and especially the very wide confidence interval for the extended-field estimate preclude a precise estimate of effect magnitude or an independent causal interpretation.

The central interpretation is therefore not that a particular IMV ligation technique causes gastroparesis. Rather, DGE after left hemicolectomy is a final common phenotype that requires further classification. In proximal or T4 tumours, broader Treitz-adjacent dissection, local inflammation and oedema, mild

angulation, and systemic stress may jointly produce retention in the stomach and proximal duodenum. This phenotype should not be labelled as chronic gastroparesis without further distinction.

Incidence in context

The incidence observed here is of the same order as that reported in the small left-hemicolectomy series of Sarli et al. [13], in which two DGE events occurred among 54 cancer operations. By contrast, POI rates in colorectal literature frequently exceed 10% and vary with outcome definitions, side of resection, and adherence to enhanced-recovery pathways [5–12]. Our 2.37% estimate represents a persistent upper gastrointestinal phenotype without fixed mechanical obstruction and with supportive contrast evidence; it is not directly interchangeable with POI defined by passage of flatus or stool, dietary recovery, or hospital stay.

Interpretation of pathological T4 stage

Pathological T4 stage should be interpreted as an associated clinical marker rather than a direct mechanism. T4 disease can entail a stronger local tissue reaction, adhesions, involvement of adjacent structures, broader mesenteric dissection, and a greater sterile inflammatory burden. Within the operative anatomy of proximal tumours, these features may amplify oedema, traction, and dysmotility at the Treitz region. The direction of association in the full cohort and extended-field subgroup is compatible with this anatomical–inflammatory model, but requires external validation.

Mucinous or signet-ring cell carcinoma had a higher observed incidence, although the difference was not statistically significant and could reflect its relation to T4 stage, peritoneal reaction, or case mix. The absence of clear associations with mismatch-repair status, HER2, or Ki-67 is consistent with DGE being primarily an acute operative–anatomical phenotype rather than a direct molecular phenotype.

Occult functional obstruction at the Treitz region

Postoperative gastric retention after left hemicolectomy can arise from at least three mechanisms. The first is overt mechanical obstruction, including marked angulation or folding at the Treitz region, adhesions, extrinsic compression, or superior mesenteric artery compression [14–17]. The second is stomach-predominant or pan-enteric dysmotility driven by surgical stress, sympathetic activation, opioids, metabolic derangement, and systemic inflammation [25–28]. The third lies between these categories: occult functional obstruction at the Treitz region, in which no stable transition point is visible, yet local oedema, subtle positional change, and inflammatory suppression of bowel-wall motility jointly increase dynamic resistance at the duodenojejunal flexure.

Experimental studies show that local intestinal manipulation activates resident muscularis macrophages, recruits leucocytes, and suppresses smooth-muscle contractility [18, 19]. Selective jejunal manipulation can also induce pan-enteric inflammation and delayed gastric emptying in non-manipulated segments, producing a remote-field effect [20]. Mast cells, the vagal–muscularis macrophage anti-inflammatory pathway, and enteric glial cells participate in this neuroimmune regulation [21–25], while human studies link intestinal handling to local inflammation and ileus [22].

Together, these observations provide biological plausibility for a sequence in which Treitz-adjacent inflammatory ileus reduces dynamic transit through the duodenojejunal flexure and culminates in gastric retention (Fig. 6).

This framework also explains why a patient may have no fixed mechanical transition point on computed tomography yet show pronounced delayed gastric emptying on contrast imaging. A static scan captures anatomy at one moment and may not fully represent intermittent angulation, reduced local compliance, or the dynamic interaction between dysmotility and outflow resistance. Thus, computed-tomography exclusion of an overt fixed lesion does not by itself prove a purely gastric motor disorder. Serial contrast imaging, multiphase assessment focused on the Treitz region, and quantitative measurement of local calibre and angulation are logical next steps.

Clinical implications

For persistent vomiting, gastric dilatation, or high nasogastric output after left hemicolectomy, the diagnostic question should shift from “Is this gastroparesis?” to “Which mechanism is producing gastric retention?” Computed tomography and contrast imaging should first be reviewed specifically for a fixed transition point, marked angulation, proximal dilatation, or vascular compression at the Treitz region and duodenojejunal flexure. Recovery of the small bowel and colon should then be considered to identify diffuse POI. Even without a fixed mechanical cause, occult functional obstruction at the Treitz region should be distinguished conceptually from stomach-predominant DGE. A proximal tumour with pathological T4 disease is a setting for heightened vigilance, but it is not a diagnostic criterion.

Perioperative care should continue to follow multimodal enhanced-recovery principles, including opioid-sparing analgesia, rational fluid therapy, early mobilisation, and individualised nutritional support [9, 10, 27–33]. Net-positive postoperative fluid balance was more frequent among patients with DGE in this cohort. However, day-specific postoperative input and output were not available for a separate postoperative-day 1–3 analysis, and the recorded 7-day window overlapped with outcome ascertainment. The observed difference may therefore reflect tissue oedema, increased intravenous support after poor oral intake, or both, and should not be treated as a causal factor.

Strengths and limitations

This study established a dedicated left-hemicolectomy cohort, described tumour location and operative exposure using explicit anatomical terms, required computed tomography and upper gastrointestinal contrast evidence for every DGE event, and examined TNM stage, histopathology, and molecular markers systematically. The association with pathological T4 stage and the hypothesis of occult Treitz-region functional obstruction provide a focused direction for combined imaging, inflammatory, and motility studies.

The study was retrospective and conducted at a single centre, and a standardised 4-h gastric-emptying test was not used; the acute postoperative DGE phenotype therefore cannot be equated with chronic

gastroparesis. Tumour location, splenic-flexure mobilisation, IMV ligation level, and Treitz-adjacent dissection were interrelated in this cohort, so the findings reflect the extended operative field as a whole. Although cross-classification confirmed that pathological T4 stage and extended-field status were not nested, pathological stage, local tumour burden, and operative complexity remain clinically related constructs; residual confounding cannot be fully excluded. With only 9 events, the extremely wide confidence interval around the extended-field estimate further indicates substantial uncertainty and warrants validation in larger cohorts. Opioid exposure, reconstructed bowel-limb course, Treitz-region angulation, local bowel-wall oedema, and inflammatory markers were not quantified using standardised methods; occult functional obstruction consequently remains a mechanistic hypothesis.

Conclusions

DGE occurred after approximately 2.4% of laparoscopic left hemicolectomies but could persist for several weeks. Events occurred only among patients with a tumour-location-related extended operative field; pathological T4 stage may mark greater local tumour burden and operative complexity rather than constitute a direct mechanism. Because the extended-field estimate was highly imprecise, its magnitude requires confirmation in larger cohorts. Postoperative gastric retention should be differentiated among fixed mechanical “pseudo-gastroparesis”, occult functional obstruction at the Treitz region, stomach-predominant DGE, and diffuse POI. Prospective studies should standardise measurement of Treitz-region angulation, local oedema, and inflammatory markers and combine these assessments with dynamic contrast imaging or a 4-h gastric-emptying test.

Declarations

Ethics approval and consent to participate

The study was approved by the Ethics Committee of Minhang Hospital, Fudan University (approval no. 202105301K). Individual informed consent was waived because of the retrospective, de-identified design.

Consent for publication

Not applicable.

Availability of data and materials

De-identified data and analysis code are available from the corresponding authors upon reasonable request and subject to institutional and ethical requirements.

Competing interests

The authors declare that they have no competing interests.

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Authors' contributions

FT conceived the study, curated the data, performed the statistical analysis, and drafted the manuscript. ML verified the results and critically revised the manuscript. CYY curated the pathological data and contributed to methodology. PHW conducted case investigation and prepared the visualisations. YOC conceived and supervised the study and critically revised the manuscript. SQL conceived and supervised the study, acquired funding, contributed to methodology, and critically revised the manuscript. All authors read and approved the final manuscript.

References

1. Staller K, Parkman HP, Greer KB, et al. AGA Clinical Practice Guideline on Management of Gastroparesis. *Gastroenterology*. 2025;169:828–861.
2. Schol J, Huang IH, Carbone F, et al. Rome Foundation and international neurogastroenterology and motility societies' consensus on idiopathic gastroparesis. *Lancet Gastroenterol Hepatol*. 2025;10:68–81.
3. Camilleri M, Kuo B, Nguyen L, et al. ACG Clinical Guideline: Gastroparesis. *Am J Gastroenterol*. 2022;117:1197–1220.
4. Abell TL, Camilleri M, Donohoe K, et al. Consensus recommendations for gastric emptying scintigraphy. *J Nucl Med Technol*. 2008;36:44–54.
5. Hedrick TL, McEvoy MD, Mythen MMG, et al. Joint consensus statement on postoperative gastrointestinal dysfunction within an enhanced recovery pathway for elective colorectal surgery. *Anesth Analg*. 2018;126:1896–1907.
6. Vather R, Trivedi S, Bissett I. Defining postoperative ileus: results of a systematic review and global survey. *J Gastrointest Surg*. 2013;17:962–972.
7. Gero D, Gié O, Hübner M, et al. Postoperative ileus: in search of an international consensus on definition, diagnosis, and treatment. *Langenbecks Arch Surg*. 2017;402:149–158.
8. Wells CI, Milne TGE, Seo SHB, et al. Post-operative ileus: definitions, mechanisms and controversies. *ANZ J Surg*. 2022;92:62–68.
9. Gustafsson UO, Scott MJ, Hubner M, et al. Guidelines for perioperative care in elective colorectal surgery: ERAS Society recommendations 2018. *World J Surg*. 2019;43:659–695.
10. Irani JL, Hedrick TL, Miller TE, et al. Clinical practice guidelines for enhanced recovery after colon and rectal surgery from ASCRS and SAGES. *Surg Endosc*. 2023;37:5–30.

11. Seo SHB, Carson DA, Bhat S, et al. Prolonged postoperative ileus following right- versus left-sided colectomy: a systematic review and meta-analysis. *Colorectal Dis.* 2021;23:3113–3122.
12. Adamova Z, Filova M, Slovacek R. Factors determining postoperative ileus after surgery for colon cancer: comparison of right- and left-sided resections. *Surg Technol Int.* 2022;40:140–146.
13. Sarli L, Pavlidis C, Cinieri FG, et al. Prospective comparison of laparoscopic left hemicolectomy for colon cancer with laparoscopic left hemicolectomy for benign colorectal disease. *World J Surg.* 2006;30:446–452.
14. Yuan XY, Li RP, Wu ZJ, Li RE, Zhu JQ, Deng ZW. Reconstructing Ligament of Treitz Reduces Postoperative Upper GI Obstructions after Left Hemicolectomy: A Prospective Randomize Controlled Study. *J Dig Oncol (Electronic Version).* 2018;10:214–219.
15. Li X, Tian M, Yang H, et al. Superior mesenteric artery syndrome after colectomy: a case report and literature review. *Medicine (Baltimore).* 2022;101:e30427.
16. Alsaleh N, Yaseen W, Alshamat RA, et al. Superior mesenteric artery syndrome following colorectal surgery: a systematic review. *Ann Med Surg (Lond).* 2025;87:2763–2773.
17. Yerdal MA, Moreira LF, Iwagaki H, et al. Acute superior mesenteric artery syndrome following left hemicolectomy: a case report. *Acta Med Okayama.* 1992;46:479–482.
18. Kalff JC, Schraut WH, Simmons RL, et al. Surgical manipulation of the gut elicits an intestinal muscularis inflammatory response resulting in postsurgical ileus. *Ann Surg.* 1998;228:652–663.
19. Kalff JC, Carlos TM, Schraut WH, et al. Surgically induced leukocytic infiltrates within the rat intestinal muscularis mediate postoperative ileus. *Gastroenterology.* 1999;117:378–387.
20. Schwarz NT, Kalff JC, Türler A, et al. Selective jejunal manipulation causes postoperative pan-enteric inflammation and dysmotility. *Gastroenterology.* 2004;126:159–169.
21. de Jonge WJ, The FO, van der Coelen D, et al. Mast cell degranulation during abdominal surgery initiates postoperative ileus in mice. *Gastroenterology.* 2004;127:535–545.
22. The FO, Bennink RJ, Ankum WM, et al. Intestinal handling-induced mast cell activation and inflammation in human postoperative ileus. *Gut.* 2008;57:33–40.
23. Gao Z, Müller MH, Karpitschka M, et al. Role of the vagus nerve on the development of postoperative ileus. *Langenbecks Arch Surg.* 2010;395:407–411.
24. Matteoli G, Gomez-Pinilla PJ, Nemethova A, et al. A distinct vagal anti-inflammatory pathway modulates intestinal muscularis resident macrophages independent of the spleen. *Gut.* 2014;63:938–948.
25. Mazzotta E, Villalobos-Hernandez EC, Fiorda-Diaz J, et al. Postoperative ileus and postoperative gastrointestinal tract dysfunction: pathogenic mechanisms and novel treatment strategies beyond colorectal ERAS protocols. *Front Pharmacol.* 2020;11:583422.
26. Hussain Z, Park H. Inflammation and impaired gut physiology in post-operative ileus: mechanisms and treatment options. *J Neurogastroenterol Motil.* 2022;28:517–530.

27. Sommer NP, Schneider R, Wehner S, et al. State-of-the-art colorectal disease: postoperative ileus. *Int J Colorectal Dis.* 2021;36:2017–2025.
28. Buscail E, Deraison C. Postoperative ileus: a pharmacological perspective. *Br J Pharmacol.* 2022;179:3283–3305.
29. Rybakov EG, Shelygin YA, Khomyakov EA, et al. Risk factors for postoperative ileus after colorectal cancer surgery. *Colorectal Dis.* 2018;20:189–194.
30. Lee MJ, Vaughan-Shaw P, Vimalachandran D, et al. A systematic review and meta-analysis of baseline risk factors for postoperative ileus in gastrointestinal surgery. *Ann R Coll Surg Engl.* 2020;102:194–203.
31. Quiroga-Centeno AC, Jerez-Torra KA, Martin-Mojica PA, et al. Risk factors for prolonged postoperative ileus in colorectal surgery: a systematic review and meta-analysis. *World J Surg.* 2020;44:1612–1626.
32. Namba Y, Hirata Y, Mukai S, et al. Clinical indicators for the incidence of postoperative ileus after elective surgery for colorectal cancer. *BMC Surg.* 2021;21:80.
33. Myles PS, Bellomo R, Corcoran T, et al. Restrictive versus liberal fluid therapy for major abdominal surgery. *N Engl J Med.* 2018;378:2263–2274.
34. von Elm E, Altman DG, Egger M, et al. The STROBE statement: guidelines for reporting observational studies. *Lancet.* 2007;370:1453–1457.
35. Spitzer RL, Kroenke K, Williams JBW, et al. A brief measure for assessing generalized anxiety disorder: the GAD-7. *Arch Intern Med.* 2006;166:1092–1097.
36. Weiser MR. AJCC 8th Edition: Colorectal Cancer. *Ann Surg Oncol.* 2018;25:1454–1455.
37. Heinze G, Schemper M. A solution to the problem of separation in logistic regression. *Stat Med.* 2002;21:2409–2419.

Figures

Study design, outcome adjudication, and analytical framework

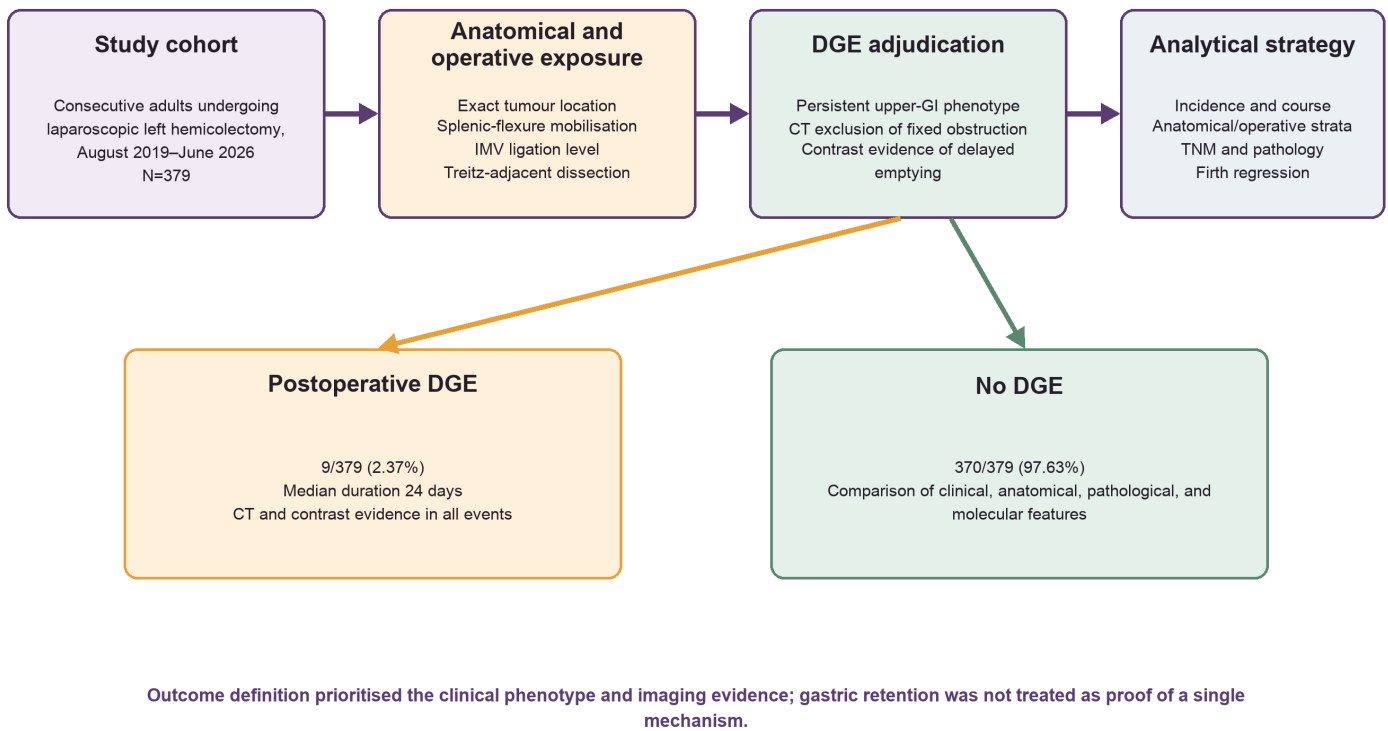


Figure 1

Study design, outcome adjudication, and analytical framework. DGE was defined from a persistent gastrointestinal phenotype, computed-tomography exclusion of fixed mechanical obstruction, and supportive upper gastrointestinal contrast findings.

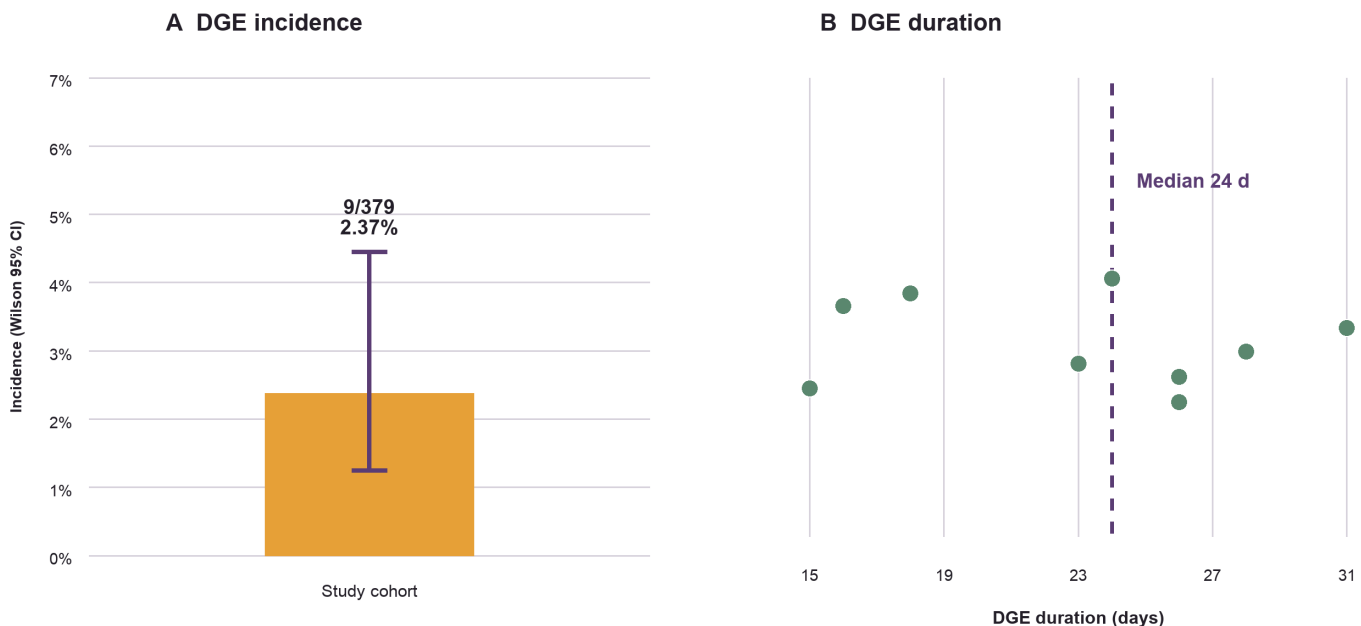


Figure 2

Incidence and duration of postoperative DGE. (A) Observed incidence with Wilson 95% confidence interval. (B) Individual duration among the 9 patients with DGE; the dashed line indicates the median of 24 days.

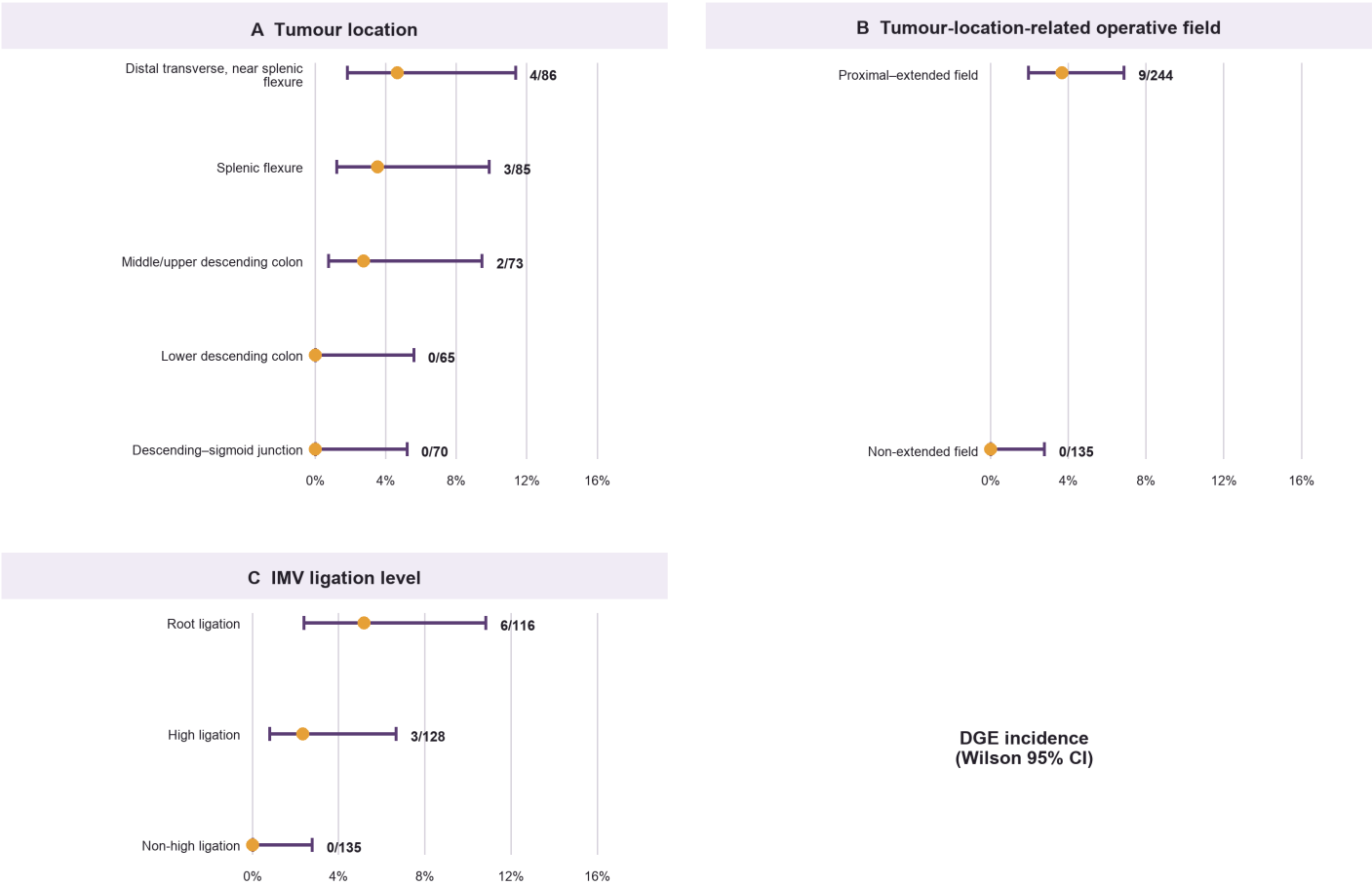


Figure 3

DGE incidence according to tumour location, tumour-location-related operative field, and IMV ligation level. Points show observed incidences, horizontal bars Wilson 95% confidence intervals, and labels the number of events/total patients.

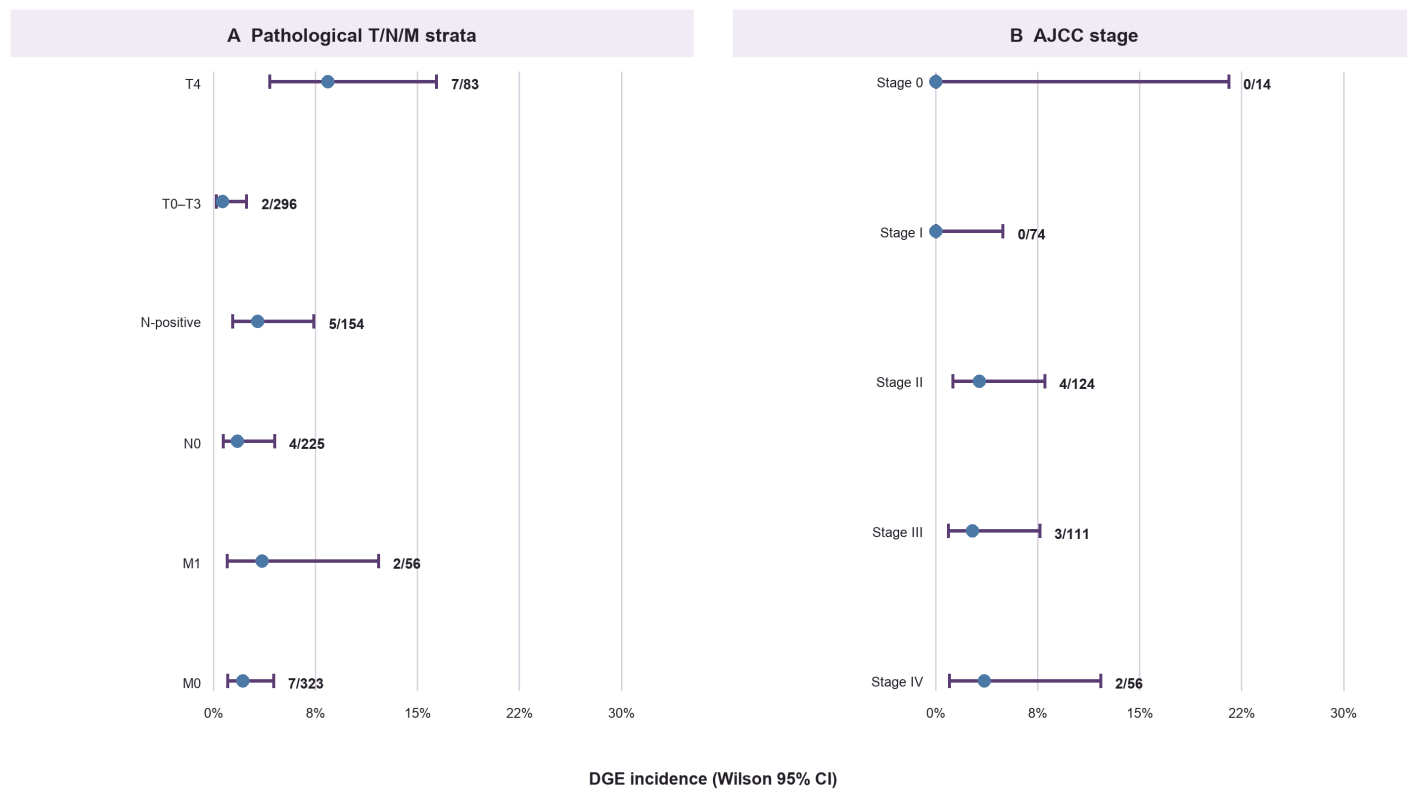


Figure 4

DGE incidence across pathological T, N, and M strata and AJCC stages. DGE incidence was higher in pathological T4 than T0–T3 disease.

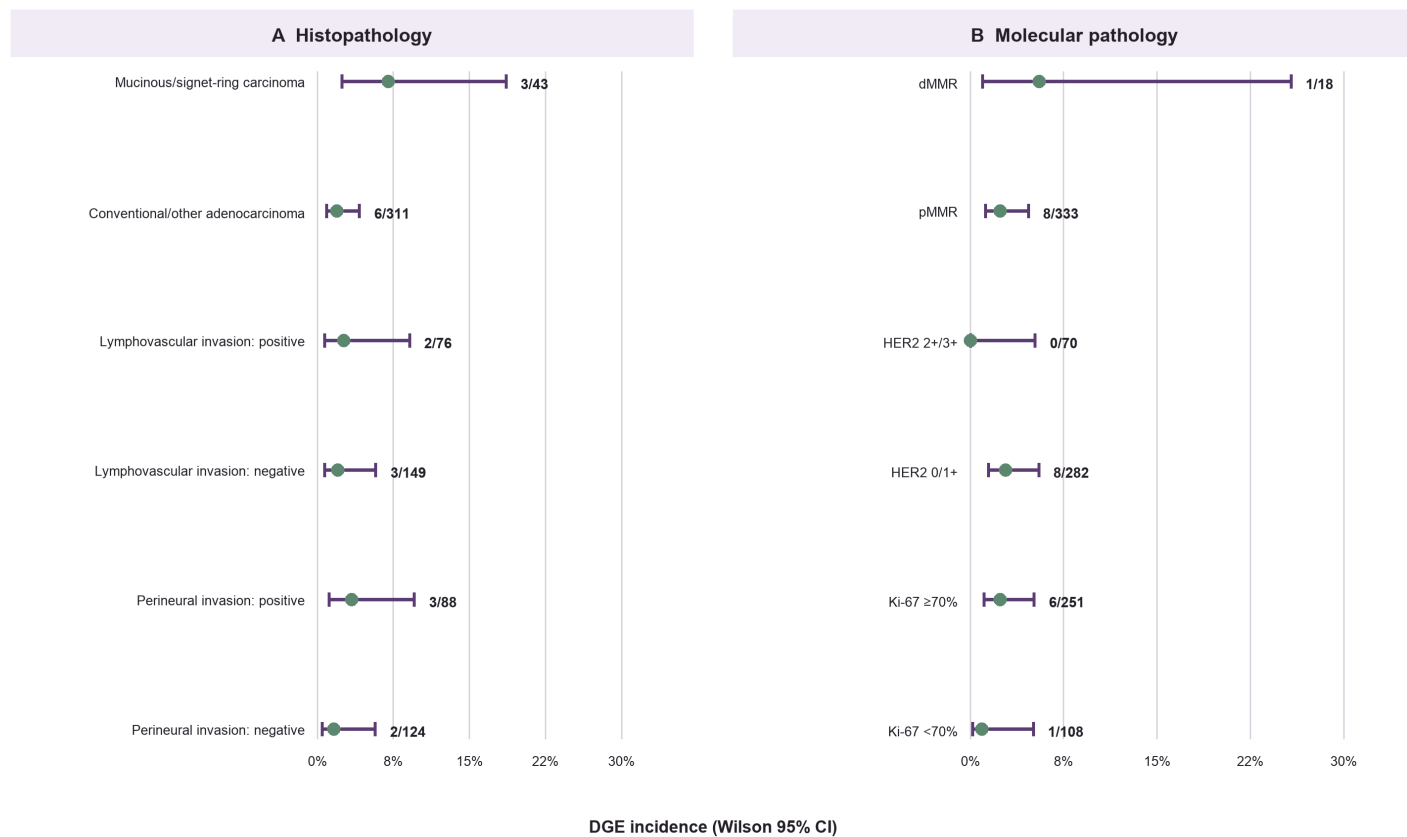
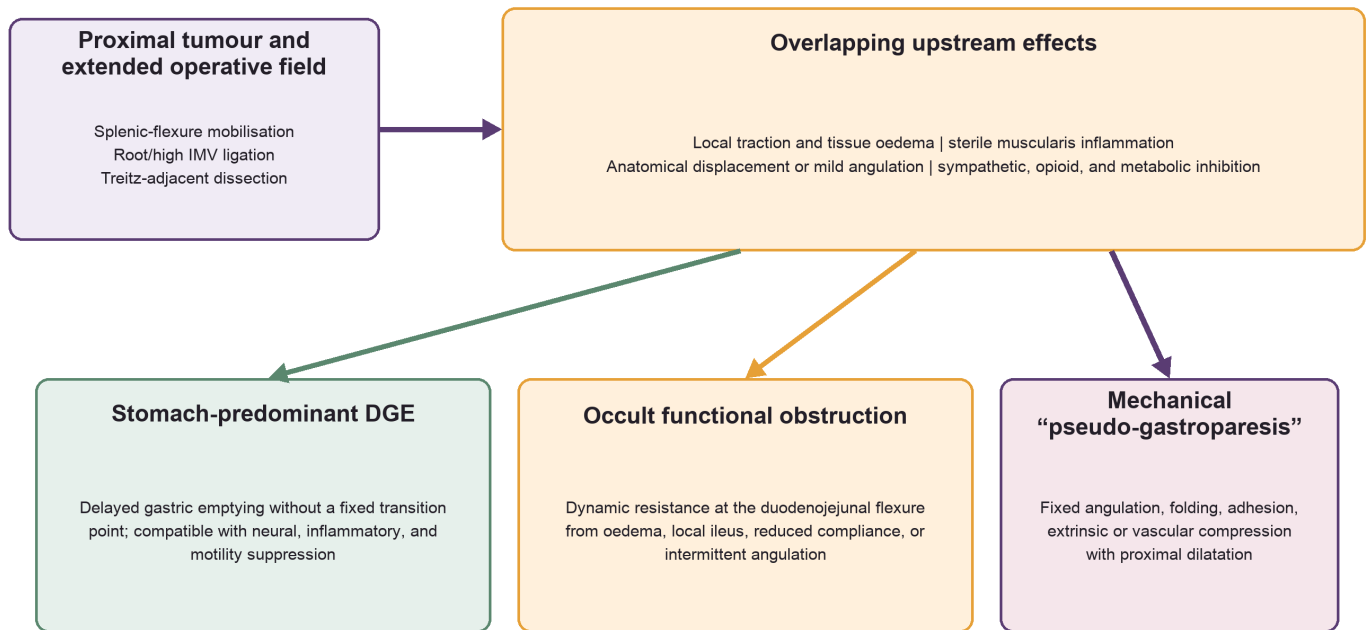


Figure 5

DGE incidence across histopathological and molecular-pathology strata.

Hypothesised Treitz-region pathway to occult functional obstruction and gastric retention



Shared radiographic phenotype: gastric and proximal duodenal retention. A static CT can exclude an overt fixed lesion but cannot fully characterise intermittent angulation or dynamic resistance.

Clinical distinction requires symptoms, contrast transit, targeted Treitz-region review, and assessment of diffuse small- and large-bowel recovery.

Figure 6

Hypothesised Treitz-region pathway to occult functional obstruction. Local tissue oedema or positional change, sterile inflammatory ileus, and systemic neuro-metabolic inhibition may converge on a similar phenotype of gastric and proximal duodenal retention.

Supplementary Files

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