Review of pathological findings in laparoscopic sleeve gastrectomy specimens performed for morbid obesity

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ABSTRACT

Background Bariatric surgical procedures are employed when there is a failure of lifestyle modification in arresting obesity. Laparoscopic sleeve gastrectomy (LSG) is quickly becoming the bariatric surgical procedure of choice. LSG results in a gastric remnant that is subject to pathological examination. The objective of this paper is to review the literature in regard to histological findings identified in gastric remnants post-LSG and identify the most pertinent histological findings.

Materials and methods A literature search was performed to identify relevant case series. Data gathered from relevant case series then underwent statistical analysis.

Results The most common histological findings in an LSG specimen were clinically indolent findings such as no pathological abnormalities identified followed by non-specific gastritis. A minority of cases demonstrated clinically actionable findings for which *Helicobacter pylori* represented the majority of these findings.

Conclusion There is a broad spectrum of pathological findings in LSG specimens, ranging from clinically indolent to clinically actionable. The most common histological findings are clinically indolent and only a small portion are of clinical significance and, hence, actionable.

INTRODUCTION

In 2016, the WHO reported that more than 1.9 billion adults, 18 years and older, were overweight, and of these, over 650 million were obese.[1] Furthermore, obesity has been associated with adverse medical outcomes such as cardiovascular disease, diabetes mellitus, degenerative joint disease and malignancy. Initial management includes lifestyle and dietary modifications. Failure of initial management of obesity results in treatment with adjunct pharmacotherapy or bariatric surgery.[2]

A variety of bariatric surgical procedures exist and include, but not limited to, Roux-en-Y gastric bypass (RYGB) and laparoscopic sleeve gastrectomy (LSG). In 2011, LSGs represented 17.8% of all metabolic and bariatric procedures performed in the USA, whereas, in 2016, the number of LSGs represented 58.1% of all metabolic and bariatric procedures performed in the USA.[3] Moreover, a recent meta-analysis has demonstrated laparoscopic RYGB (LRYGB) and LSG to be equivalent for excess weight loss.[4] Patients receiving LSG experienced fewer postoperative complications and reoperation rate than those who underwent LRYGB.[4]

LSG involves taking down the greater curvature of the stomach with the subsequent division of the stomach, in turn, creating a gastric remnant.[5] Currently, it is at the discretion of the institutions to determine whether LSG specimens undergo pathological examination. There have been only a handful of studies describing the histopathological findings encountered in LSG specimens from patients with morbid obesity. The objective of this review is to provide a systematic review of the literature describing the all histopathological findings seen in LSG specimens.

MATERIALS AND METHODS

Criteria for considering studies

i. Inclusion criteria.
   - The inclusion criteria were as follows: (1) case series; (2) study must include patients undergoing LSG for weight loss or comorbidities; (3) all gross and microscopic examination of LSG specimens must be done in the department of pathology; (4) reportage of histological findings and the number patients per histological finding; and (5) published in English.

ii. Exclusion criteria.
   - The exclusion criteria were as follows: (1) experimental trial on animals or non-human study; (2) patients undergoing other bariatric procedure; (3) other diseases that may influence outcome; (4) insufficient data or not meeting inclusion criteria; and (5) not published in English.

iii. Others.
   - If present, the following parameters were recorded: (1) demographic data: number of female and male patients, mean age and mean body mass index (BMI); (2) preoperative investigations including, but not limited to, upper gastrointestinal endoscopy (UGIE) and *Helicobacter pylori* serology; and (3) country where study was performed.

Search strategy

The Cochrane Library and PubMed were searched up to 30 November 2019. Search terms included 'sleeve gastrectomy', 'histology' and 'pathology'. The reference sections of some studies were also

searched. Two assessors independently screened the titles and abstracts of each study. When a relevant study was identified, the full text was obtained for further evaluation.

Statistical analysis

Statistical analysis was performed using the Data Analysis tool within Microsoft Excel. Equality of variance of clinically indolent and clinically actionable findings was determined using Levene's test. The difference between means of the clinically indolent and clinically actionable groups was measured using a t test. One-way analysis of of variance (ANOVA) and regression analysis were performed on the clinically indolent and clinically actionable groups, individually.

RESULTS

Levene's test demonstrated the presence of inequality in variance (p=0.001). Subsequently, a t test assuming unequal variance demonstrated a significant difference (p=1.32E-5) between the means of the clinically indolent group and the clinically actionable group.

Demographics

A total of 12 923 patients were included in this review; however, not all studies published demographic data such as sex, age or BMI (table 1).

Summary of histological findings

A total of 13 173 histological diagnoses were made. The spectrum of the most frequent findings included no pathological findings (n=5648; 42.9%), gastritis (n=4574; 34.7%) and *H. pylori* (n=1913; 14.5%) (table 2).

Clinically Indolent histological findings

Clinically indolent findings were histological findings that did not require subsequent medical intervention and represented 10 947 (83.1%) of the total 13 173 histological diagnoses made. These findings were further subdivided into non-neoplastic and neoplastic lesions. Clinically indolent non-neoplastic findings, in descending order of frequency, included no pathological findings (n=5648; 42.9%), gastritis (n=4574; 34.7%), lymphoid aggregates (n=354; 2.7%), fundic gland polyps (FGPs) (n=212; 1.6%), proton pump inhibitor (PPI) effect (n=69; 0.52%), benign non-neoplastic polyps (n=29; 0.22%), benign non-specific findings (n=28; 0.21%), non-necrotising granulomatous inflammation (n=9; 0.07%), ulceration/necrosis (n=6; 0.05%) and pancreatic ectopic tissue (n=5; 0.04%) (table 2). Benign neoplasms represented 13 of all histological diagnoses made (0.10%) (table 2).

Regression analysis demonstrated a correlation between the number of patients and the number of clinically indolent histological findings, R squared value of 0.88 (figure 1). One-way ANOVA failed to demonstrate a difference between and within the groups (p=0.98).

Clinically actionable histological findings

Clinically actionable findings were histological findings requiring subsequent medical intervention and represented 2226 (16.9%) of the total 13 173 histological diagnoses made. Clinically actionable findings, in descending order of frequency, included non-neoplastic lesions (n=1916; 14.5%), premalignant lesions (n=260, 2.0%) and malignant lesions (n=50; 0.4%) (table 2).

Regression analysis demonstrated a weak correlation between the number of patients and the number of clinically actionable

Table 1 Demographic data of patients undergoing laparoscopic sleeve gastrectomies

Study	Year study was published in print	Country in which study was conducted	Length of time that data were collected (days)	Patients in study (n)	Females (n)	Males (n)	Mean age (years)	Mean preoperative body mass index (kg/m^2)	Patients who underwent preoperative UGEI (n)	Patients that underwent preoperative *Helicobacter* screening other than UGEI (n)
Al Saady R *et al*	2019	Qatar	2921	342	241 (70.5%)	101 (29.5%)	34.7	Not reported	Not reported	Not reported
Demibras B *et al*	2019	Turkey	1766	253	183 (72.3%)	70 (27.7%)	38.5	47.7	253	253
Di Palma *et al*	2019	Canada	2464	222	178 (80.2%)	44 (19.8%)	48.1	51.2	50	222
Erkinuresin T *et al*	2019	Turkey	2190	151	134 (88.7%)	17 (11.3%)	36.0	43.9	Not reported	Not reported
Ge L *et al*	2019	USA	790	649	468 (72.1%)	181 (27.9%)	46.2	45.2	649	649
Komaei I *et al*	2019	Italy	2249	474	349 (73.6%)	125 (26.4%)	40.5	43.3	474	Not reported
Turan G *et al*	2019	Turkey	1825	1257	940 (74.8%)	317 (25.2%)	37.3	43.8	1257	Not reported
Aljerian K	2018	Saudi Arabia	2190	602	336 (55.8%)	266 (44.2%)	31.4	Not reported	Not reported	Not reported
Canil AM *et al*	2018	Italy	1703	925	743 (80.3%)	182 (19.7%)	44.1	44.5	925	925
Dogan U *et al*	2017	Turkey	Not reported	291	225 (77.3%)	66 (22.7%)	42.0	Not reported	291	291
Hansen SK *et al*	2017	USA	3256	351	277 (78.9%)	74 (21.1%)	48.6	43.6	Not reported	63
Kopach P *et al*	2017	USA	2555	511	338 (66.1%)	173 (33.9%)	51.3	44.3	Not reported	Not reported
Saafan T *et al*	2017	Qatar	1276	1555	1084 (69.7%)	471 (30.3%)	35.5	46.8	1555	1555
Yardmci E *et al*	2017	Turkey	2160	755	496 (65.7%)	259 (34.3%)	39.6	42.6	755	755
Kinsinger LA *et al*	2016	USA	1976	241	183 (75.9%)	58 (24.1%)	46.0	47.0	Not reported	241
Miller GC *et al*	2016	Australia	2646	1463	1112 (76.0%)	351 (24.0%)	43.0	Not reported	Not reported	Not reported
Abdull Gaffar *et al*	2015	United Arab Emirates	2921	546	351 (64.3%)	195 (35.7%)	33.0	Not reported	63	Not reported
Clapp B	2015	USA	1613	145	97 (66.9%)	48 (33.1%)	43.1	47.5	Not reported	Not reported
Lauti M *et al*	2015	New Zealand	2648	976	Not reported	Not reported	Not reported	Not reported	21	Not reported
Ohanessian S *et al*	2015	USA	2282	310	236 (76.1%)	74 (23.9%)	45.0	49.9	8	310
Raess PW *et al*	2015	USA	1522	248	172 (69.4%)	78 (31.5%)	43.0	Not reported	Not reported	Not reported
Almazeedi S *et al*	2013	Kuwait	1246	656	480 (73.2%)	176 (26.8%)	33.6	Not reported	656	656
Total number of patients in all studies	12 923									

UGEI, upper gastrointestinal endoscopic investigation.

Table 2 Summary of all histological findings identified in laparoscopic sleeve gastrectomy specimens

			Frequency	%
Clinically indolent histological findings (frequency)	Non-neoplastic	No pathological findings	5648	42.88
		Gastritis	4574	34.72
		Lymphoid aggregates	354	2.69
		Fundic gland polyps	212	1.61
		Proton pump inhibitor effect	69	0.52
		Benign non-neoplastic polyps	29	0.22
		Benign non-specific findings	28	0.21
		Non-necrotising granulomatous inflammation	9	0.07
		Ulceration and necrosis	6	0.05
		Heterotopic pancreatic tissue	5	0.04
	Neoplastic	Leiomyoma	8	0.06
		Submucosal lipoma	2	0.02
		Neurofibroma	1	0.01
		Glomus tumour	1	0.01
		Other*	1	0.01
	Total frequency of clinically indolent histological findings		10 947	83.10
Clinically actionable histological findings (frequency)	Non-neoplastic	*Helicobacter pylori* gastritis	1913	14.52
		Necrotising vasculitis	2	0.02
		Eosinophilic gastritis	1	0.01
	Premalignant	Intestinal metaplasia	168	1.28
		Atrophic gastritis, not otherwise specified	80	0.61
		Dysplasia	5	0.04
		Neuroendocrine microadenoma	3	0.02
		Atypical lymphoproliferative process	2	0.02
		Enterochromaffin-like cell hyperplasia	2	0.02
	Malignant	Gastrointestinal stromal tumour	44	0.33
		Adenocarcinoma	2	0.02
		Neuroendocrine tumour (well differentiated; G1)	2	0.02
		Extranodal marginal zone lymphoma	1	0.01
		Metastasis†	1	0.01
	Total frequency of clinically actionable histological findings		2226	16.90
Total of histological findings			13 173	

*Benign neoplasm, not otherwise specified.
†Metastatic ovarian mucinous cystadenocarcinoma.

histological findings, R squared value of 0.51. One-way ANOVA failed to demonstrate a difference between and within the groups (p=0.67).

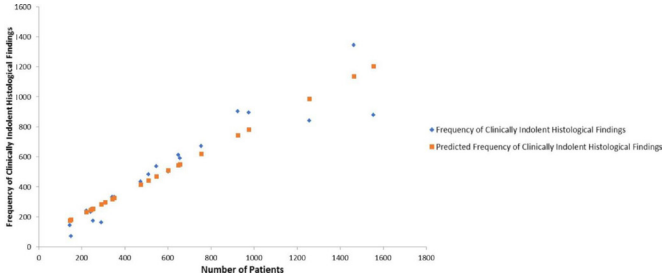

Figure 1 Graph depicting linear regression of clinically indolent histological findings.

Non-neoplastic lesions

Non-neoplastic clinically actionable lesions included, in descending frequency, *H. pylori* (n=1913; 14.5%), necrotising vasculitis (n=2; 0.02%) and eosinophilic gastritis (n=1; 0.01%).

Premalignant lesions

Premalignant clinically actionable lesions included, in descending frequency, intestinal metaplasia (n=168, 1.3%), atrophic gastritis (n=80; 0.61%), dysplasia (n=5; 0.04%), neuroendocrine microadenoma (n=3; 0.02%), atypical lymphoid proliferation(n=2; 0.02%) and enterochromaffin cell-like (ECL) hyperplasia (n=2; 0.02%).

Malignant lesions

Malignant clinically actionable lesions included, in descending frequency, gastrointestinal stromal tumours (GISTs) (n=44; 0.33%), adenocarcinoma (n=2; 0.02%), well-differentiated, G1, neuroendocrine tumours (n=2; 0.02%), extranodal marginal zone lymphoma (n=1; 0.01%) and a metastatic ovarian cystadenocarcinoma (n=1; 0.01%).

DISCUSSION

As the number of the LSG procedures increases, the volume of specimens received by pathologists also increases. It is thus appropriate that the practising pathologist be aware of the spectrum of pathology present in LSG specimens.

The vast majority of histological findings are considered clinically indolent; however, clinically actionable findings do occur.

Levene's test demonstrated the presence of inequality in variance (p=0.001) among the clinically indolent and the clinically actionable groups. Subsequently, a t test assuming unequal variance demonstrated a significant difference (p=1.32E-5) between the means of the clinically indolent group and the clinically actionable group. These results confirm that the mean number of the clinically indolent histological findings and clinically actionable histological findings are not equivalent.

Clinical indolent histological findings

Clinically indolent histological findings represented the majority of findings discovered in LSG specimens, 10 947 (83.1%) of the total 13 173 histological diagnoses rendered. The most common diagnosis made was that of no pathological abnormalities identified (n=5648; 42.9%).

Statistical analysis revealed a positive linear correlation between the number of patients and the number of clinically indolent histological findings, R squared value of 0.88. Furthermore, one-way ANOVA failed to demonstrate a difference between and within the clinically indolent histological findings of each study (p=0.98). These results indicate that the mean

number of clinically indolent histological findings across all studies included in this review are equivalent.

Gastritis and ulceration/necrosis

The second most frequent diagnosis made was gastritis (n=4574; 34.7%). Ulceration/necrosis occurred at a much lower rate (n=6; 0.05%). These findings are not surprising as obesity itself has been found to be a cause of gastritis and ulceration.[6] Yamamoto *et al* demonstrated association of obesity with endoscopic gastritis and gastric ulcers, coining the term obesity-related gastritis.[6] Obesity-related gastritis is a non-*Helicobacter*-associated gastritis which is thought to be associated with decreased levels of adiponectin in obese patients.[6]

Polyps and PPI effect

A total of 212 (1.6%) FGPs and 29 (0.22%) other benign non-neoplastic polyps were reported.[7–21] FGPs are generally considered benign and one of the most common gastric polyps identified during endoscopy.[22] They can be classified as sporadic or to a lesser extent arising in the setting of familial adenomatous polyposis.[23 24] An increased prevalence of FGPs has also been attributed to increased PPI use.[24] On microscopic examination, PPIs have been to cause cystic dilation of fundic glands, parietal cell hyperplasia and cytoplasmic vacuolation.[25]

Lymphoid aggregates

The overall frequency of lymphoid aggregates was 354 (2.7%) per 13 173 diagnoses. This was in keeping with the study performed by Saafan *et al*,[11] which demonstrated the presence of 35 (2.4%) lymphoid aggregates. In their study, Safaan *et al* demonstrated 74.3% (n=26) of patients had a concurrent *H. pylori* infection. Unfortunately, we could not make the same correlation as not all case series reported an association with lymphoid aggregates and *H. pylori*.

Non-necrotising granulomatous inflammation

Non-necrotising granulomatous inflammation was identified at a frequency of 0.07% (n=9).[7 11 15 20 21] This is consistent with literature. Granulomatous gastritis is an uncommon condition, occurring in <1% of all gastric biopsies.[26] It is often inconsequential, and the aetiologies vary from iatrogenic, *H. pylori*-related, foreign body reactions, sarcoidosis and secondary to Crohn disease.[26]

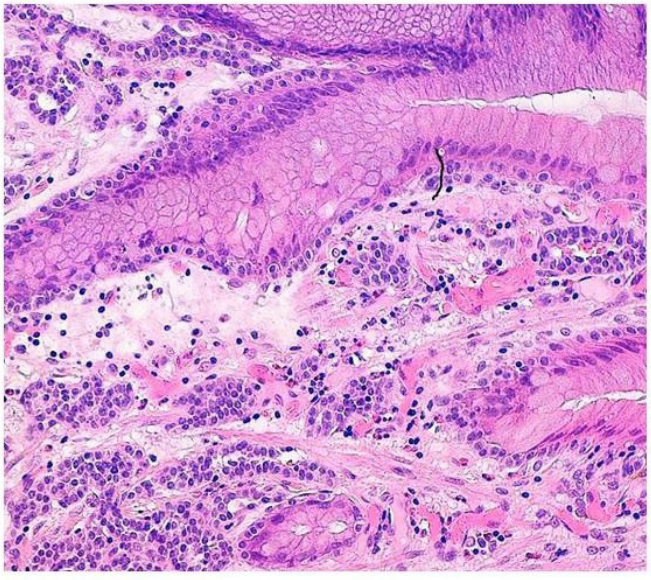

Figure 2 An incidental histological finding in a sleeve gastrectomy specimen: an incidental neuroendocrine tumour (×200).

Benign neoplasms and other benign findings

Other benign findings included non-specific inflammation (n=28; 0.21%) and benign neoplasms such as leiomyomas (n=8; 0.06%),[7–9 11 12 16 18 19 27] lipomas (n=2; 0.02%),[8 11] glomus tumour (n=1; 0.01%),[28] neurofibroma (n=1; 0.01%)[16] and benign neoplasm, not otherwise specified (n=1; 0.01%).[17] Five cases (0.04%) of heterotopic pancreatic tissue were also identified.[11 15–17 19]

Clinically actionable histological findings

Clinically actionable histological findings in this review occurred at a rate of 2226 (17.0%) times. This is higher than the 1.18%–7.8% reported in the literature.[8 10 11 27] This discrepancy is most likely explained by the geographic distribution of *H. pylori* as our review included case series with high incidences of *H. pylori*. The rate of clinically actionable histopathological findings further drops to 2.4% (n=313) if *H. pylori* is excluded.

Regression analysis demonstrated a weak positive linear correlation between the number of patients and the number of clinically actionable histological findings, R squared value of 0.51. One-way ANOVA failed to demonstrate a difference between and within the clinically actionable histological findings of each study (p=0.67). These findings suggest that a positive linear correlation between the number of patients and the number of clinically actionable is unlikely. However, the mean number of clinically actionable histological findings across all studies included in this review are equivalent.

Non-neoplastic lesions

Helicobacter pylori

Helicobacter was identified in 1913 (14.5%) of 13 173 histological findings. There was great variability in the geographical distribution of *H. pylori*, which is in keeping with known literature.[29] The greatest percentage of *H. pylori*-infected patients was seen in Turkey[28 30–33] and Qatar.[11]

It is well known that *H. pylori* infection is a risk factor for the development of gastric adenocarcinoma[34 35] and extranodal marginal zone lymphoma of mucosa-associated lymphoid tissue (MALT).[36] Conversely, *H. pylori* does not influence perioperative complications, postoperative outcomes or weight loss.[37 38]

Our review demonstrated clinically actionable findings at a rate of 17.0% (n=2226). Of the 2226 clinically actionable findings identified, 1913 LSG specimens were positive for *H. pylori*. Although the correlation between *H. pylori* and atrophic gastritis, intestinal metaplasia and adenocarcinoma could not be extrapolated from the current data, *H. pylori* is a known risk factor for the development of these conditions. Consequently, we strongly urge that *H. pylori* screening techniques be further explored as this would alter the rate of clinically actionable findings drastically.

Other

Necrotising vasculitis was reported in two cases.[16 20] A workup for systemic vasculitis is essential in these types of cases. Limited studies exist describing the incidences and pathogenesis of necrotising vasculitis in the setting of LSG.

Eosinophilic gastroenteritis (EG) was described in one case.[16] EG is defined as diffuse or patchy with >20 eosinophils per

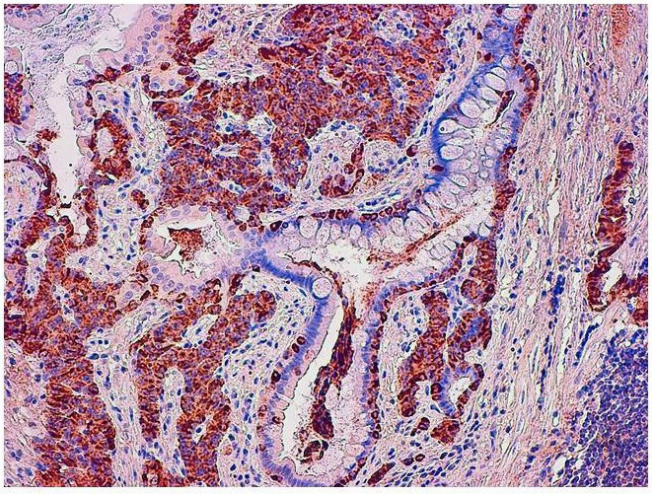

Figure 3 An incidental histological finding in a sleeve gastrectomy specimen: an incidental neuroendocrine tumour, chromogranin (×200).

high-power fields.[39] The differential diagnosis includes parasitic infection, inflammatory bowel disease, allergic granulomatous vasculitis and iatrogenic.[39]

Premalignant lesions

The rate of intestinal metaplasia was 1.3% (n=168),[8 10–17 27 28 30 32 33 40] whereas atrophic gastritis was reported at a rate of 0.6% (n=80).[9–12 16 17 19 20 31 32 40] It was not possible to make the distinction between the frequency of atrophic multifocal autoimmune gastritis and *Helicobacter*-associated atrophic gastritis, as not all the case series reported aetiologies of atrophy. The association between *H. pylori* and premalignant conditions, such as intestinal metaplasia and atrophic gastritis, is well established.[41–44] The highest rates of intestinal metaplasia and atrophic gastritis occurred within the same study,[32] a study where *H. pylori* was 39.4%.

Dysplasia was identified five times (0.04%) within one series.[10] Another study identified two cases of dysplasia on preoperative UGEI only, but not in the resection specimen.[27] Atypical lymphoid hyperplasia was noted twice[8 20]; however, no association with a *H. pylori* infection was reported.

ECL hyperplasia was identified in two cases in one study[8]; nonetheless, it was not described whether the ECL hyperplasia was part of autoimmune atrophic gastritis or secondary to PPI use.

Malignant lesions

GISTs represented the majority of malignant lesions and occurred at a frequency of 0.3% (n=44). A review of literature demonstrated incidence of GISTs in LSG specimens to range from 0.6% to 0.8%.[45–48] Risk stratification was not possible for all case series, as the size and mitotic activity were not reported in all cases diagnosed as GIST. From the studies that did report GIST size and mitotic activity, size ranged from 0.2 to 3.5 cm and none were of high grade.[7 8 10–12 14 15 19 27 33] Of note, gastric GISTs less than 2 cm are considered benign and treated by surgical resection by the National Comprehensive Cancer Network guidelines and subsequently do not require further treatment.[49]

One case (0.01%) of gastric autonomic neural tumour (GANT) was identified.[21] GANT is an extremely rare tumour of the neural plexus and occur at any age, but most commonly in young adults, around 20 years of age,[50] but is more appropriately considered within the remit of gastrointestinal tumours.[51]

Extranodal lymphoma of MALT was identified in one case.[12] Extranodal lymphoma of MALT is known to be associated with *H. pylori*.[36 52] However, on further investigation, the author noted that *H. pylori* was not identified, and the patient's follow-up revealed clinical and radiologic remission of the disease.[12]

Two cases of gastric adenocarcinoma were identified.[8 40] One case represented an intramucosal signet ring adenocarcinoma.[8] Neither of the studies reported *H. pylori* status, nor was it reported whether there was an associated preoperative UGEI.

Two cases of neuroendocrine tumour (figures 2 and 3) were identified,[8 33] as well as a total of three cases of neuroendocrine microadenomas (microcarcinoids) associated with autoimmune atrophic gastritis were reported.[11 20] In addition, Kopach *et al* reported pancreatic acinar metaplasia and microcarcinoids in the setting of autoimmune atrophic gastritis in the study. Currently, there is a paucity of literature documenting the incidence of neuroendocrine proliferations in LSG specimen.

Raess *et al* reported one metastatic ovarian cystadenocarcinoma. Following LSG, the patient was referred to gynaecological oncology service.[20]

Take home messages

- There is a broad spectrum of pathological findings in laparoscopic sleeve gastrectomy specimens, ranging from clinically indolent to clinically actionable.
- The most common histological findings are clinically indolent and only a small portion are of clinical significance and, hence, actionable.
- Major findings were *Helicobacter pylori* gastritis, prominent lymphoid aggregate formation with or without *H. pylori*.

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