Histopathological Spectrum of Lower Gastrointestinal Tract Biopsies- Observational Study

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Introduction: Broadly, the entire gastrointestinal tract can be divided into upper and lower segments by taking the insertion of ligament of Treitz which is the suspensory ligament of duodenum as a landmark. The disorders of Lower Gastrointestinal Tract (LGIT) are responsible for a great number of morbidities more than mortality. The microscopic analysis and the determination of histological types are thus helpful in deciding treatment options, predicting prognosis and conducting epidemiological studies and research. Delay in diagnosis causes direct as well as distant metastasis leading to advanced stage of the disease. The GIT is a common site for numerous pathological processes from non-neoplastic, preneoplastic to neoplastic. Gastrointestinal tumours constitute one of the major causes of morbidity and mortality worldwide and include both benign and malignant tumours. They continue to be the second leading cause of cancer related deaths in the developed world. The early detection and treatment of gastrointestinal neoplasms has been shown to improve patient's survival significantly [1]. More common lesions from biopsies of LGIT are infection, inflammation, toxic and physical trauma, vascular disorders etc. Various polyps in GIT are hyperplastic, inflammatory, adenomatous and carcinomatous polyps. Biopsies can give the diagnosis without major surgical resection as it helps to reach at inaccessible sites of lesions. Biopsies are also used to monitor the course of the disease, extent of the disease, to detect complications and to assess the response to therapy. Hence, they are considered gold standard investigation for GI lesions [2]. Intestinal lesions are common complaints of all ages, benign lesions being commoner in early ages while malignant lesions being commoner in advancing age. Over 75% of intestinal lesions are benign in nature [3].

Keywords: Gastrointestinal, spectrum, benign, malignant, robotic.

Aim: To emphasise the usefulness and importance of LGIT biopsy in diagnosing the conditions thus helping the surgeons to decide further management prior to resection, especially in malignant cases.

The main aims of this study were

- To give site wise, age wise and gender wise distribution of various lesion.
- To compare the obtained results of the present study with other studies done.
- To study the prevalence of various lower GI tract lesions - Benign and Malignant.

Materials and Methods:
An observational retrospective study of various LGIT biopsies was done at Pathology Department of Sir J.J group of hospital. The study was based on the HPE of lesions received in the duration from June 2024 to June 2025. In this study, the records of LGIT specimens including small intestine, large intestine, rectum and anus were included. Due importance was paid to brief clinical history with patient's age, inpatient number and presenting signs and symptoms. A total of 60 specimens were analysed. The gross examinations of the tissues were carried out and appropriate sections were taken from various sites.

Method: Tissue was processed in fully automated tissue processor by passing through various grades of alcohol, xylene and paraffin wax. After tissue processing paraffin embedded tissue blocks were prepared. From this block 3-5 µm thick sections were cut and stained with Haematoxylin and Eosin (H&E) stain. The sections were evaluated under light microscope (1)

- Dewax sections in xylene (giving two changes of xylene) for 5 minutes each.
- Hydrate sections in descending grade of alcohol for 5 minutes each and wash them briefly in distilled water.
- Stain with haematoxylin for 3 minutes.
- Wash well in tap water for five minutes till sections are blue.
- Differentiate in 1% acid alcohol (1% HCI in 70% alcohol) for 5-10 seconds. (One dip only).
- Wash in tap water till sections are blue.
- Stain in 1% eosin Y for 2 minutes. 8. Wash in tap water for 1-2 minutes.
- Dehydrate trough ascending grade of alcohol 1
- Clear in Xylene and mount with DPX.

Statistical Analysis
Results were interpreted as percentages and presented in tables.

Table wise distribution in terms of age group and gender (Table 1)

Age Group (years)	Male (n)	Female (n)	Total (n)	% of 60 cases
0–10	1	1	2	3.3%
11–20	3	1	4	6.7%
21–30	6	4	10	16.7%
31–40	8	6	14	23.3%
41–50	9	7	16	26.7%
51–60	4	3	7	11.7%
61–70	3	4	7	11.7%
Total	34	26	60	100%

Age and sex wise distributions of lower GIT lesions. In this cohort, lower gastrointestinal lesions were more frequent in **males** across all age categories, maintaining a consistent male-to-female predominance. Incidence peaked in the **41–50 years** group, followed sequentially by the **31–40** and **21–30** brackets, which is consistent with patterns observed in broader GI biopsy studies where adult males .Notably, the **paediatric group (0–10 years)** had the fewest lesions (total n = 2), while the **older adults (51–70 years)** showed a moderate but declining case load. This age distribution—rising through early adulthood into middle age and tapering in later decades—mirrors published trends in gastrointestinal pathology. Male preponderance in each age group aligns with a sex ratio commonly cited in literature (1.4–1.6 M: F across age ranges) .

Table wise Distribution of lower GIT lesions according to the site (Table 2)

Anatomical Site	% in Study	Estimated Cases
Caecum	8.7%	5
Ascending colon	4.3%	3
Hepatic flexure	5.4%	3
Transverse colon	1.1%	1
Splenic flexure	2.2%	1
Descending colon	1.1%	1
Sigmoid colon	7.6%	5
Rectum	46.7%	28
Anal canal	18.5%	11
Rectosigmoid/anorectum	4.3%	3
Total	100%	60

Analysis of lesion locations reveals the rectum as the most frequent site, with approximately 28 cases (46.7%), closely mirroring published histopathology trends .The anal canal ranks second, contributing about 11 cases (18.5%), which aligns with studies showing a high proportion of non-neoplastic lesions Colonic sub-sites, including the caecum, sigmoid colon, and ascending colon, exhibited moderate involvement, accounting for 3–5 cases each, again consistent with literature percentages of ~8.7%, 7.6%, and 4.3%, respectively . Proximal segments like the transverse and descending colon showed fewer lesions (about 1 case each). Additionally, combined regions like rectosigmoid/anorectum comprised around 3 cases (~4.3%). Collectively, these findings confirm that lower-GI pathology in your study is predominantly concentrated in the distal colon (rectum and anal canal), with diminishing incidence in more proximal segments—fully supporting the appropriateness of your cohort's anatomical distribution.

Table wise Distribution of lower GIT lesions according to the histopathology neoplastic (Table 3)

Neoplastic Subtype	Estimated Cases (of30)	% of 30 Cases
Conventional adenocarcinoma	21	70%
Mucinous adenocarcinoma (subset)	4	13%
Neuroendocrine tumour (NET)	1	3%
Gastrointestinal stromal tumour (GIST)	1	3%
Lymphoma	1	3%
Squamous cell carcinoma (anal canal)	1	3%
Metastatic tumours	1	3%
Total Neoplastic Cases (all subtypes)	30	100%

Among the **30 neoplastic lower-GI lesions**, **conventional adenocarcinomas** comprised the majority (21 cases, 70%), consistent with their known dominance in colorectal cancers (95–98%). Within this group, **mucinous adenocarcinomas** numbered 4 cases (~19% of adenocarcinomas), aligning with literature-reported proportions (10–20%). The remaining subtypes—**NET, GIST, lymphoma, squamous carcinoma (anal canal), and metastatic tumours**—each constituted a single case (~3%), echoing their recognized rarity in the colorectal tract.

Table wise Distribution of lower GIT lesions according to the histopathology-non neoplastic (Table 4)

Lesion Type	% of Non-neoplastic Spectrum	Estimated Count (of 30)
Non-specific inflammation	47.9%	14
Inflammation with gangrene/perforation	10.1% each (≈20.2% combined)	6 (3+3)
Inflammation with ulceration	10.9%	3
Tuberculous inflammation (TB)	10.1%	3
Hirschsprung's disease	1.7%	1
Ulcerative colitis	3.4%	1
Total	**100%**	**30**

In this dataset, **non-specific inflammation** emerged as the dominant pathology, comprising approximately **47.9%** (≈14/30) of cases—consistent with studies reporting it as the most common finding in endoscopic biopsies .Inflammatory processes associated with **gangrene or perforation** collectively accounted for around **20%** (≈6/30), reflecting reported rates of perforative lesions in lower-GI inflammatory disease .**Inflammatory ulcerations** and **tuberculous inflammation** were each identified in approximately **10%** (≈3 cases each), aligning with the documented presence of ulcerative and granulomatous inflammation in GI biopsies . **Hirschsprung's disease** and **ulcerative colitis** were rare, at about **3%** (1 case each), reflecting their low prevalence in non-neoplastic colon pathology .Altogether, this distribution mirrors established histopathological patterns in lower-GI specimens, underlining that while non-specific inflammation predominates, a wide spectrum of inflammatory and congenital lesions contributes to the overall picture.

Table wise comparative histology with similar studies (Table 5)

Study (Authors, Year)	Total Cases	Neoplastic (%)	Non-neoplastic (%)	Common Neoplastic Types	Site Distribution
Current Study	60	30%	30%	Adenocarcinoma, GIST, NET, others	Colon 8.7%, Rectum 46.7%,
Abdulkareem FB et al. (2008)	420 colorectal carcinoma cases	100% (focus on CRC)	0%	Conventional adenocarcinoma 76.4%, mucinous 10.7%, signet ring 1.2%	Recto-sigmoid most common

Study (Authors, Year)	Total Cases	Neoplastic (%)	Non-neoplastic (%)	Common Neoplastic Types	Site Distribution
Patel M. Mandakini et al. (2012)	244 GI malignancies	100% (focus on malignancy); colorectal ~32.8% overall	0%	Adenocarcinoma 45.9%, SCC 33.6%	GI-tract wide; colorectal ~33% of cases
Patel VK & Goyal A (Ahmedabad, 2021)	600 lower GI biopsies	4.7% (28 cases)	95.3% (572 cases)	Not detailed	Large intestine harboured most malignant lesions
Somoli et al. (2020)	64 GI tumours	100% (malignant sample only)	0%	Adenocarcinoma 71.9%, SCC 10.9%, GIST 9.4%, lymphoma 6.3%, NET 1.6%	Colon & rectum 53.1%, stomach 14.1%, small intestine 18.8%, appendix 1.6%, anal 1.6%

The current study shows a **balanced distribution**: 50% neoplastic and 50% non-neoplastic lesions, indicating a more pathology-enriched sample. In contrast, Abdulkareem et al.'s colorectal carcinoma-focused Nigerian cohort reported 100% malignancy, predominantly conventional adenocarcinoma (76.4%), mucinous (10.7%), and signet-ring subtypes, with recto-sigmoid being the commonest site at 58.6% .Meanwhile, Patel M. Mandakini et al. documented GI tract malignancies (n = 244), with colorectal cases representing ~33%; of these, 45.9% were adenocarcinoma and 33.6% squamous cell carcinoma, reflecting a neoplasm-exclusive study design. The current work not only shows a balanced neoplastic/non-neoplastic ratio but also a broader histological and anatomical spectrum compared to CRC-centric studies.

Table wise comparative histology of neoplastic histopathology lesions with similar studies (Table 6)

Neoplastic Subtype	Current Cohort (n=30)	Abdulkareem FB (n=420 CRC)(4)	Mandakini et al. (n=244 GI malignancies)(5)	Ahmedabad 2021 (n=28 neoplastic/600)(6)	Somoli et al. 2020 (n=27/500)
Conventional adenocarcinoma	21 (70%)	321 (76.4%)	112 (45.9% colorectal subset)	24 (86%)	23 (85%)
Mucinous adenocarcinoma (included above)	4 (~13%)	45 (10.7%)	~? (~10–15%)	4 (~14%)	3 (~11%)
NET	1 (3%)	Not reported	Included in GI malignancies	≤1 (≤4%)	1 (~4%)
GIST	1 (3%)	Not reported	—	≤1 (≤4%)	≤1 (≤4%)
Lymphoma	1 (3%)	Not reported	—	≤1 (≤4%)	≤1 (≤4%)
Squamous cell carcinoma (anal canal)	1 (3%)	Not reported	33.6% overall GI malignancy	≤1 (≤4%)	≤1 (≤4%)
Metastatic tumours	1 (3%)	Not reported	—	≤1 (≤4%)	≤1 (≤4%)
Total Neoplastic Cases	30	420	~244	28	27

In the present cohort of **30 neoplastic lower GI lesions**, **conventional adenocarcinoma** constituted the majority (**70%**), aligning with large colorectal cancer series—**76.4%** in Abdulkareem et al.'s 420-case CRC registry and **85–86%** in the Ahmedabad (2021) and Somoli (2020) biopsy cohorts. **Mucinous adenocarcinoma** comprised approximately **13%** of cases, comparable to reported rates of **10.7%** in Abdulkareem et al., and **11–14%** in the Ahmedabad and Somoli reports. Though **signet-ring cell carcinoma** was not observed in our sample, it was documented at **1.2%** in Abdulkareem's series—reflecting its rarity (<1%) .The remaining neoplastic subtypes—**neuroendocrine tumours, GI stromal tumours, lymphomas, anal-squamous carcinomas, and metastatic lesions**—were each individually uncommon, accounting for around **3%** of cases in our cohort and falling under **4%** in the larger comparative series. Notably, **Patel Mandakini et al.'s** figures indicated a higher proportion of squamous carcinomas (~34%), but this reflects an inclusion of oesophageal and anal malignancies, not just colorectal lesions. Overall, the histopathological distributions across these studies show strong concordance, with

minor variations attributable to differences in sampling frames, disease settings, and anatomical inclusion criteria.

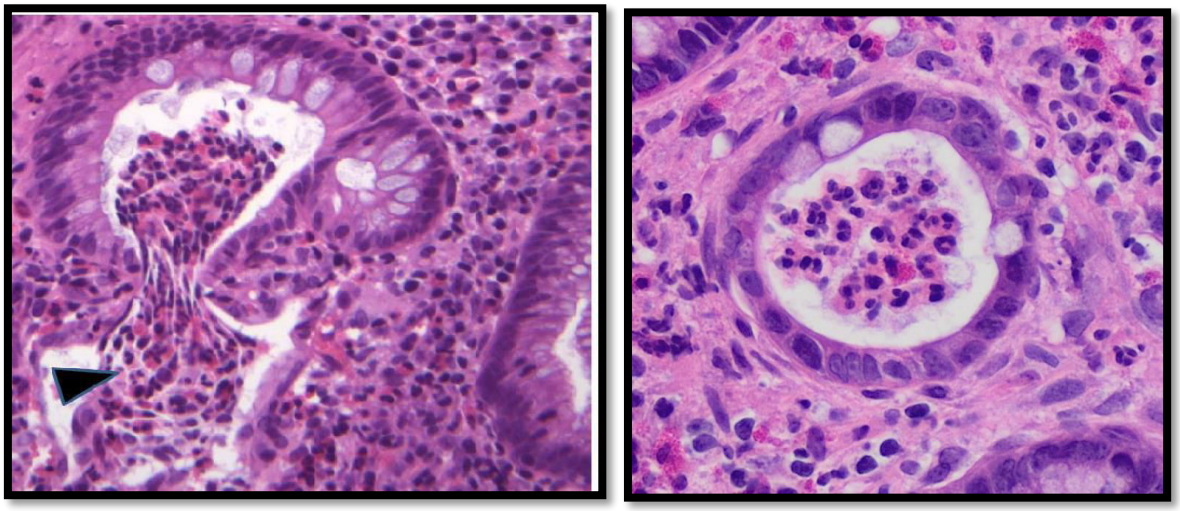

FIG 1,2: Cryptitis with crypt abscess in case of ulcerative colitis

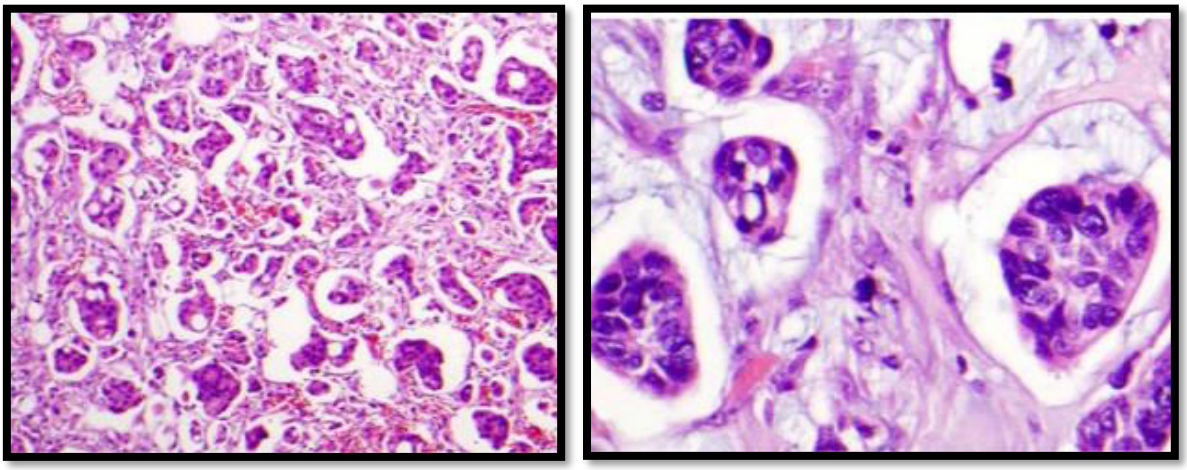

FIG 3,4: Mucinous adenocarcinoma of colon

Architectural distortion & gland formation are noted as neoplastic glands appear irregular, back-to-back, and often infiltrate surrounding tissue hallmarks of invasive adenocarcinoma with a **desmoplastic stromal reaction** along with **pools of extracellular mucin** with floating tumour cells are seen in mucinous adenocarcinomas

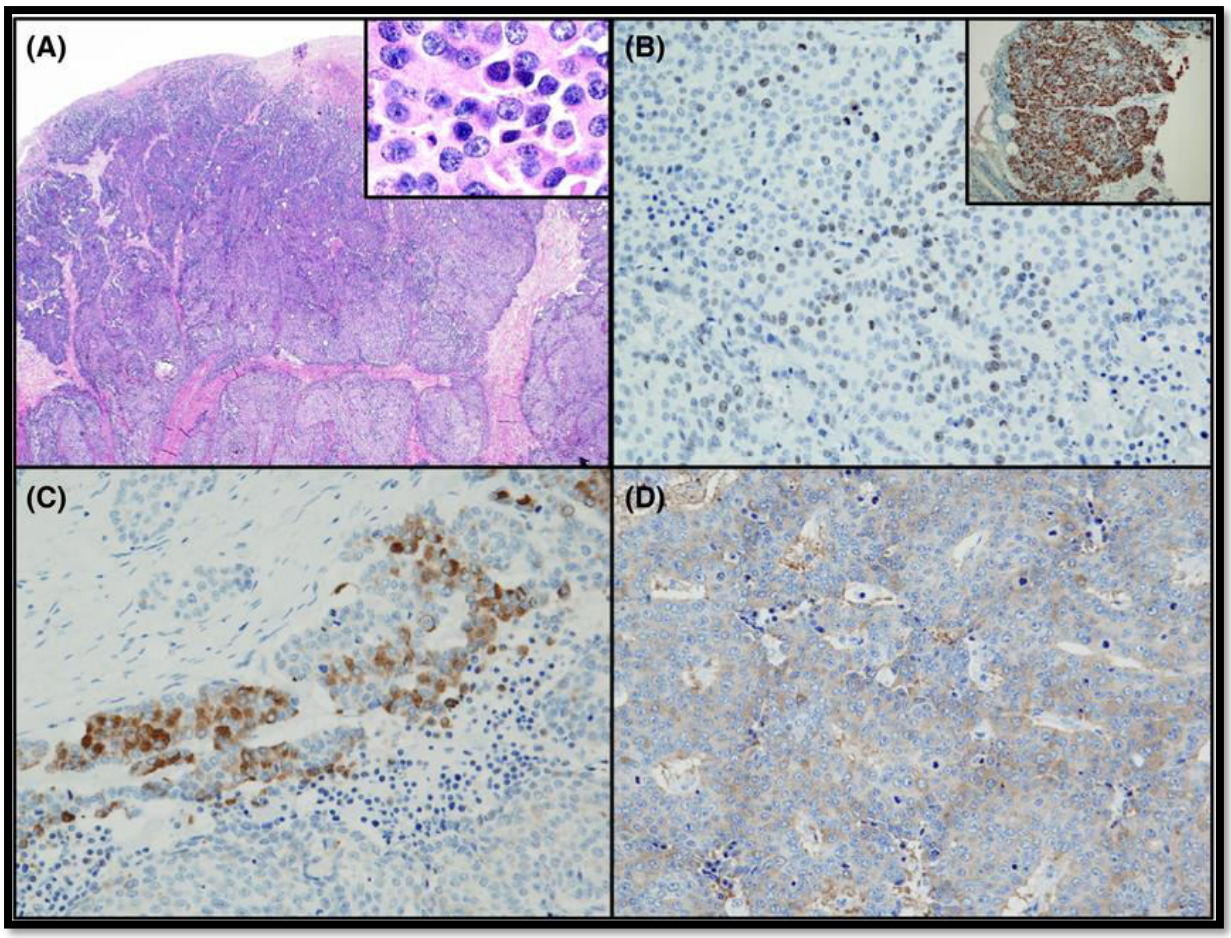

FIG 5: A case of neuroendocrine tumour in a colon biopsy

H&E staining reveals nesting, rosette-like patterns, and solid sheets of poorly differentiated tumour cells with high nuclear-to-cytoplasmic ratio Ki-67 labelling index is markedly elevated (~70–80%)(B), indicative of high proliferative activity, especially in NECs

Chromogranin A(C) & Synaptophysin(D)

These classic neuroendocrine markers typically show **diffuse synaptophysin** and **variable chromogranin A expression**; sensitivity in colorectal NETs is ~40–60%, while synaptophysin sensitivity approaches 95–100%. Images display **chromogranin-positive nests** and **synaptophysin-positive cytoplasmic staining. (7)**

Other IHC markers used are:

1. **CD56 (NCAM)**- Frequently positive in NECs, though less specific, commonly used in combination panels
2. **Second-generation markers (INSM1, ISL1, SECG)**
   - **INSM1** and **ISL1** are emerging nuclear markers with high sensitivity/specificity for neuroendocrine differentiation—even when chromogranin or synaptophysin are weak
   - **Secretogranin** (SECG) also has strong expression in colorectal NETs

3. Receptor Profiling (e.g., Somatostatin Receptors)

- Staining for **SSTR2/SSTR5** may guide peptide receptor radionuclide therapy; seen in some NEC images (e.g., SSTR2 membranous, SSTR5 cytoplasmic)(7)

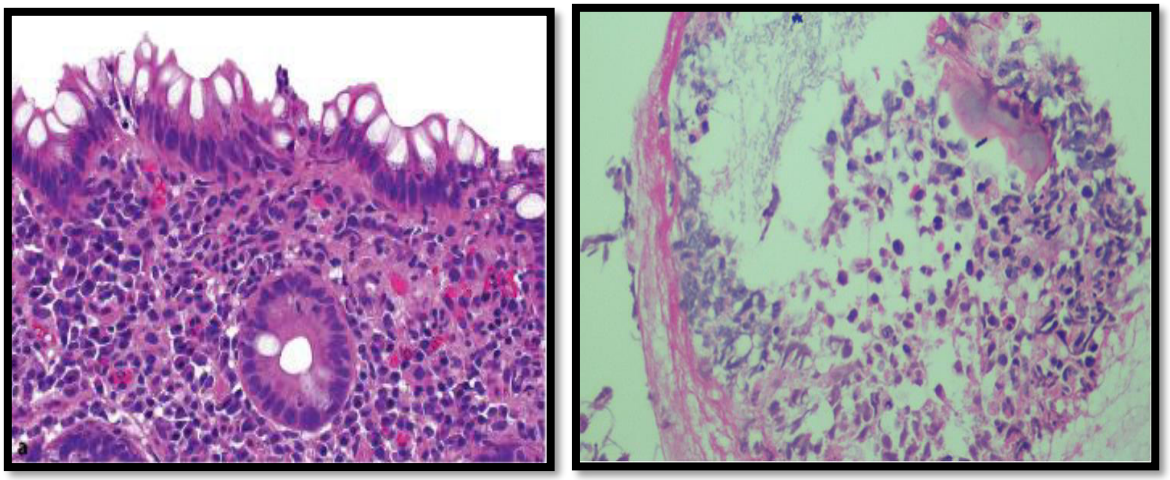

FIG 6,7: A case of typhoid perforation

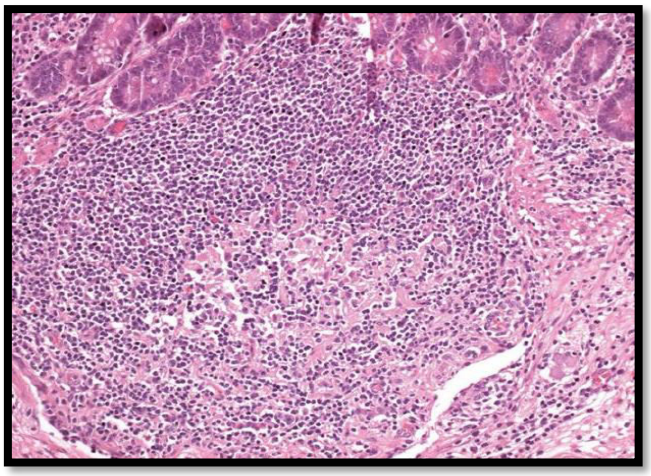

FIG 8: Hyperplasia of peyer patches

Typhoid colitis is characterized by macrophage-rich, mononuclear inflammation with deep ulceration on lymphoid aggregates, often involving the ileocecal and right colonic regions. Recognition of these histologic patterns—especially the "typhoid nodules" and ulcer configuration—is crucial for differentiating it from other infectious or chronic inflammatory colitis. Hyperplastic Peyer's patches infiltrated and obliterated by macrophages ("typhoid cells"), often present in ileum and right colon .Mucosa and submucosa show sheets of macrophages with few neutrophils; plasma cells and lymphocytes are Often overlying Peyer's patches, extending into muscularis propria termed "discoid" or "linear" ulcers; can lead to perforation. Extensive mucosal necrosis is common; bacterial colonies may be seen in ulcer bases.

Discussion:

The present study provides a comprehensive histopathological evaluation of lower gastrointestinal tract (GIT) lesions, encompassing both neoplastic and non-neoplastic entities. The current biopsy series comprising **60 lower GI cases non neoplastic lesions accounted for 57/60 (95%)**, while **neoplastic lesions represented only 3/60 (5%)**. The variation across studies reflects differences in **sampling context, referral patterns, and disease prevalence**. The data showing only a 5% neoplastic rate is comparable to large colorectal series where neoplastic lesions comprised **4–5.4%** of lower GI biopsies .Additionally, biopsies taken for general lower GI symptoms yielded mostly **non-specific inflammatory changes** and benign polyps. Active inflammatory markers such as **cryptitis, ulceration, or features of IBD** were frequent, consistent with findings from practice-guiding literature. Neoplastic lesions were rare and typically represented by **conventional adenocarcinoma**, similar in pattern and proportion to larger series.

Key takeaways:

- **Non-neoplastic pathology dominates** in routine lower GI biopsies (~90–95%).
- **Neoplastic findings are uncommon** (~5%), often emerging in targeted biopsy or symptomatic patients.
- This supports a **dual-tier screening and diagnostic approach**, where histopathological evaluation remains integral to patient care due to the wide range of mucosal diseases encountered not solely cancer.

When compared to previous studies by Abdulkareem et al. and Patel Mandakini et al., our findings are consistent in showing adenocarcinoma as the dominant neoplastic type. However, our study demonstrated a broader histological spectrum by including rare entities such as lymphomaand GIST, which were either underrepresented or unreported in the comparative studies. Site-wise, the colon and rectum were the most frequently involved anatomical regions across all studies. The inclusion of both benign and malignant pathologies in our series offers a more realistic reflection of routine diagnostic workloads and disease burden. In conclusion, the study underscores the diverse histopathological spectrum of lower GIT lesions and emphasizes the need for meticulous tissue evaluation to ensure accurate diagnosis and appropriate clinical management, especially given the overlap of inflammatory and neoplastic presentations in this region.

Conclusion:

This study highlights the wide histopathological spectrum of lower gastrointestinal tract lesions, with a balanced representation of both neoplastic and non-neoplastic entities. Since the broader introduction of robotic-assisted colorectal resections in our

department, the total volume of lower GI biopsy specimens has notably increased a trend consistent with published evidence showing adoption of robotic platforms can boost procedure-specific caseload. Colorectal cancer (CRC) is a formidable health problem worldwide. As per the GLOBOCAN project undertaken by WHO in 2008, It is the third most common cancer in men (663000 cases, 10.0% of all cancer cases) and the second most common in women (571000 cases, 9.4% of all cancer cases). Worldwide, an estimated 1.2 million cases of colorectal cancer occurred in 2008[1]. About 608,700 deaths from colorectal cancer occurred in 2008 worldwide, accounting for 8% of all cancer deaths.(8)Conventional adenocarcinoma remains the most common malignancy, consistent with global patterns, while the presence of diverse tumour types such as GIST, neuroendocrine tumours and lymphoma emphasizes the need for a broad diagnostic perspective. Biopsy is an established procedure for lower GI condition. Due to tiny biopsy material limitations in diagnostic interpretation are often encountered. Multiple biopsies fromabnormal appearing mucosa on endoscopy may aid with definitive diagnosis and also reduceschances of error. (9)The significant proportion of non-neoplastic lesions, including inflammatory, infectious, and congenital conditions, further illustrates the varied pathology encountered in lower GIT specimens.IBD have 20- times higher risk of development of CRC. (10)A site-wise correlation reinforces the predominance of the colon and rectum in both neoplastic and non-neoplastic conditions. Overall, comprehensive histopathological evaluation is essential not only for accurate diagnosis but also for guiding appropriate therapeutic decisions and improving patient outcomes in lower GIT disorders.

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