

# A Histopathological Study of Mean Eosinophil Count in Adult Gastrointestinal Pathologies: A Cross-sectional Study

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## ABSTRACT

**Introduction:** Elevated eosinophil counts are associated with various Gastrointestinal Tract (GIT) conditions, including autoimmune gastritis, gastroesophageal reflux disease, drug reactions, infections, radiation enteritis, collagen vascular disease, and inflammatory bowel disease. Assessing these eosinophils is essential for interpreting endoscopic biopsies, as they are frequently found alongside varying quantities of neutrophils and lymphocytes. Despite the growing prevalence of eosinophil-associated diseases, there is limited information regarding abnormal increases, distribution patterns across different diagnosis, and normal eosinophil levels.

**Aim:** To determine the eosinophil count and its distribution in various gastrointestinal lesions and to examine their association with final diagnosis.

**Materials and Methods:** A cross-sectional observational study was conducted in the Department of Pathology, Ramaiah Medical College, Bengaluru, India on a total of 503 biopsy samples from various sites of the GIT from 299 cases over a period of three months from January 2023 to March 2023. Haematoxylin and eosin-stained slides obtained from formalin-fixed paraffin-embedded blocks of all study patients were reviewed. Eosinophil counts were performed on performed on Haematoxylin and Eosin (H&E) stained slides. Areas with maximal eosinophilic counts were visually identified, and the mean mucosal eosinophilic count

was obtained from five different hotspot high-power fields. The average eosinophil count from these five fields was recorded, and the final report was expressed as 'eosinophils/hpf.' The average mean eosinophil count was tabulated, and the distribution in various gastrointestinal lesions and their association with the final diagnosis were studied. Statistical Package for Social Sciences (SPSS) version 22.0 software was used for data analysis after the data was entered into a Microsoft Excel spreadsheet.

**Results:** A total of 503 biopsy samples from various sites of the GIT from 299 cases were examined. Higher average mean eosinophil counts appeared to be significantly associated with adenomatous polyps of the stomach (p-value <0.001), transverse colon (p-value=0.001), descending colon and rectum (p-value=0.027), and tuberculosis of the terminal ileum (p-value=0.013).

**Conclusion:** The present study sheds light on the distribution and diagnostic significance of eosinophilic infiltration across various segments of the GIT in present population group. While eosinophils are usually detected in varying numbers in most mucosal biopsies of the GIT, present findings significantly contribute to defining the limits of eosinophil association in different conditions that can feature significant eosinophilia. Highlighting the causes of significant mucosal eosinophilia will also be of profound assistance in considering the differential diagnosis of eosinophilic gastroenteritis.

**Keywords:** Average mean eosinophil count, Autoimmune gastritis, Inflammatory bowel disease

## INTRODUCTION

Paul Ehrlich made the initial discovery of eosinophils, multifunctional granulocytes generated from pluripotent myeloid progenitor cells, in 1879 [1,2]. They are essential for aiding in tissue regeneration and shielding the body from external influences [3]. Within their distinct granules, these cells contain chemokines, regulatory cytokines, and readily released cytotoxic proteins [1]. An especially significant bodily interface between the environment and the body is the intestinal epithelium. While traditionally believed to be involved primarily in combating parasitic infections and allergic conditions, recent advancements have unveiled their essential role in the maintenance of the epithelial barrier, tissue remodeling, inflammation control, and bridging innate and adaptive immunity [1,3,4].

Eosinophils are primarily found in the lamina propria of the mucosa in the normal human GIT. Their presence increases distally (oesophagus < stomach < small intestine < colon), reaching maximum numbers in the caecum and ascending colon [1]. These cells cover a large portion of the GIT, constantly monitoring and regulating intricate innate reactions and tissue remodeling within the gut.

Elevated eosinophil counts are associated with various gastrointestinal conditions, such as autoimmune gastritis, gastroesophageal reflux

disease, drug reactions, infections, radiation enteritis, collagen vascular disease, and inflammatory bowel disease [3-5]. Assessing these eosinophils is essential for interpreting endoscopic biopsies, as they are frequently found alongside varying quantities of neutrophils and lymphocytes [3,4].

Despite the growing prevalence of eosinophil-associated diseases, there is limited information regarding abnormal increases, distribution patterns across different diagnosis, and normal eosinophil levels [4]. Hence, the present study aimed to determine the eosinophil count and distribution in different upper and lower gastrointestinal biopsy and resection specimens and to study their association with the final diagnosis.

## MATERIALS AND METHODS

A cross-sectional observational study was conducted on all biopsy and resected specimens of the upper and lower GIT received between January 2023 and March 2023 at the Department of Pathology, Ramaiah Medical College, Bengaluru, India. A waiver from the Institutional Ethical Committee was obtained as the study was observational in nature and was performed on archived histopathological slides, with no impact on the patients' final

diagnosis, report, or outcome. All biopsies of the upper and lower GIT, irrespective of the clinical diagnosis, were included in the study. A total of 503 biopsy samples from various sites of the GIT from 299 cases were received during this period. Multiple biopsies from different sites of the GIT were taken from some cases, depending on the presenting signs and symptoms of the patients.

**Inclusion criteria:** All reported cases of upper GI endoscopic biopsies, lower GI endoscopic biopsies, and resected specimens of the upper and lower GIT of patients above the age of 18 years.

**Exclusion criteria:** Cases with inadequate biopsies and crush artefacts.

Study Procedure

Haematoxylin and eosin-stained slides obtained from formalin-fixed paraffin-embedded blocks of all study patients were reviewed. Eosinophils were studied for their qualitative and quantitative properties. Areas with maximal eosinophilic counts were visually identified. A mean mucosal eosinophilic count was obtained from five different hot spot high-power fields (40X/0.65 lens). An average eosinophil count from the five fields was taken, and the final report was expressed as ‘eosinophils/hpf’ {eos/high power field (hpf)}. The final histopathological diagnosis, along with associated histological features, was recorded for each case. The cases were tabulated according to the gastrointestinal site: oesophageal, duodenal, ileal, caecal and ascending colon, transverse colon, descending colon, and rectal.

STATISTICAL ANALYSIS

Data was entered into a Microsoft Excel spreadsheet and was analysed with SPSS version 22.0 software. Frequencies and proportions were used to illustrate categorical data, while the mean and standard deviation were used to depict continuous data. The independent t-test was employed as a significance test to determine the mean difference between two quantitative variables. To assess the mean difference between more than two quantitative variables, an Analysis of Variance (ANOVA) test of significance was used. A p-value of less than 0.05 was accepted as statistically significant, provided that all statistical test assumptions were met.

RESULTS

A total of 503 biopsy samples from various sites of the GIT were examined from 299 cases. A significant number of subjects underwent biopsies at multiple anatomical sites [Table/Fig-1]. The cases were then categorised into different diagnostic groups based on the site of the biopsy and the histopathological findings [Table/Fig-2].

Anatomical site	Number of cases	Percentage
Oesophagus	18	3.5
Stomach	185	36.7
Duodenum	94	18.6
Terminal Ileum	41	8.1
Caecum and ascending colon	63	12.5
Transverse colon	28	5.5
Descending colon and rectum	74	14.7
Total	503	100

[Table/Fig-1]: Number of biopsies from various anatomical sites of GIT (N=503).

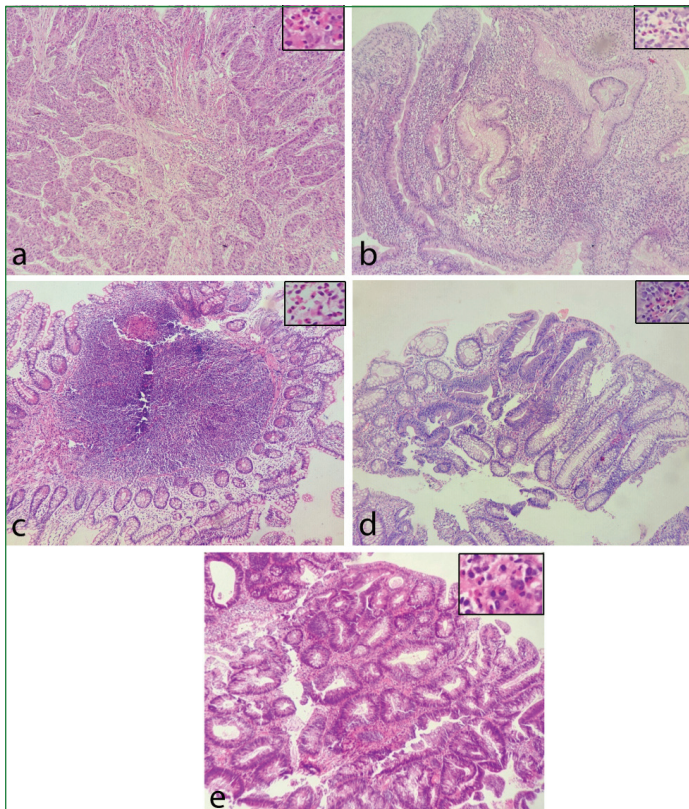
Histopathological diagnosis	Number of cases	Percentage (%)	Mean eosinophil count (eos/hpf)	p-value
Oesophagus (n=18)				
Non specific inflammation	05	27.7	4.6	0.03
Low-grade dysplasia	01	5.50	3.0	
Neoplasia	12	66.6	18.5	

Stomach (n=185)				
Acute <i>H.pylori</i> associated gastritis	25	13.3	5.6	<0.001
Chronic <i>H.pylori</i> associated gastritis	77	41.1	3.2	
Acute non specific inflammation	06	3.20	5.3	
Chronic non specific inflammation	58	31.0	4.3	
Inflammatory polyp	05	2.60	4.0	
Adenomatous polyp	03	1.60	22.3	
Neoplasia	11	5.80	14.4	
Duodenum (n=94)				
Chronic <i>H.pylori</i> associated inflammation	07	7.40	6.5	0.081
Acute non specific inflammation	07	7.40	14.9	
Chronic non specific inflammation	79	84.0	8.4	
Hyperplastic polyp	01	1.06	2.0	
Terminal ileum (n=41)				
Acute non specific inflammation	7	17	19.14	0.013
Chronic non specific inflammation	31	75.6	12.45	
Tuberculosis	3	7.3	34	
Caecum and ascending colon (n=63)				
Acute non specific inflammation	7	11.1	10.71	0.073
Chronic non specific inflammation	40	63.4	10.77	
Tuberculosis	3	4.7	12.33	
Inflammatory bowel disease	1	1.5	8	
Inflammatory polyp	1	1.5	20	
Adenomatous polyp	4	6.3	22.25	
Neoplasia	7	11.1	12.57	
Transverse colon (n=28)				
Acute non specific inflammation	1	3.5	1	0.001
Chronic non specific inflammation	16	57.1	10.75	
Tuberculosis	1	3.5	6	
Inflammatory bowel disease	1	3.5	12	
Inflammatory polyp	2	7.14	16	
Adenomatous polyp	4	14.2	40	
Neoplasia	3	10.7	15	
Descending colon and rectum (n=74)				
Chronic non specific inflammation	48	65.7	9.59	0.027
Inflammatory bowel disease	1	1.36	2	
Inflammatory polyp	7	9.58	5.69	
Adenomatous polyp	14	19.1	15.41	
Neoplasia	4	5.47	13	

[Table/Fig-2]: The average mean eosinophil counts from various anatomical sites of GIT and their association with specific histologic diagnosis.

The majority of the biopsies were from the stomach 185 (36.7%), followed by the duodenum 94 (18.6%), descending colon and rectum 74 (14.7%), and caecum and ascending colon 63 (12.5%). Biopsies from the terminal ileum, transverse colon, and oesophagus accounted for 41 (8.1%), 28 (5.5%), and 18 (3.5%) of the cases, respectively.

The mean eosinophil count and the association of the mean eosinophil count with different histopathological diagnosis at various anatomical sites of the GIT are described below. The representative images have been captured as depicted in [Table/Fig-3a-e].



**[Table/Fig-3]:** a) Biopsy from oesophagus- case of squamous cell carcinoma with mean eosinophil count of 30eos/hpf (10X magnification, inset- 40X magnification) b) Biopsy from stomach- case of hyperplastic polyp of stomach with a mean eosinophil count of 42eos/hpf (10X magnification, inset- 40X magnification) c) Biopsy from terminal ileum- Case of tuberculosis of terminal ileum (10X magnification) with adjacent areas showing a mean eosinophil count of 34 eos/hpf (inset- 40X magnification) d) and e) Biopsy from colon and rectum - Case of adenomatous polyp of colon and rectum with mean eosinophil count of 45 and 44 eos/hpf, respectively (10X magnification, inset- 40X magnification).

**Biopsy from the oesophagus (n=18):** The average mean eosinophil count per high-power field (hpf) ranged from three in non specific inflammation to 18.5 in neoplasms of the oesophagus. As observed, the average mean eosinophil count in neoplasms of the oesophagus was higher compared to non neoplastic lesions, and this association was found to be statistically significant ( $p=0.03$ ).

**Biopsy from the stomach (n=185):** The average mean eosinophil count in various lesions of the stomach, as depicted in [Table/Fig-2], ranged from 4.0/hpf to 22.3/hpf, with adenomatous polyps showing the highest average mean eosinophil count. The presence of increased eosinophils in adenomatous polyps compared to other lesions of the stomach was found to be statistically significant ( $p<0.001$ ).

**Biopsy from the small intestine (duodenum- 94 cases; terminal ileum-41 cases):** Among the various lesions encountered in the duodenum, the average mean eosinophil count ranged from 2/hpf in hyperplastic polyps to 14.88/hpf in acute non specific inflammation. No statistically significant association was found between various lesions of the duodenum and the average mean eosinophil count. Among the 41 cases of the terminal ileum, tuberculosis showed the highest average mean eosinophil count (34/hpf). The association of increased eosinophils with tuberculosis was found to be statistically significant ( $p=0.013$ ).

**Biopsy from the large intestine (caecum and ascending colon- 63 cases, transverse colon-28 cases and descending colon with rectum-74 cases):** The average mean eosinophil count in lesions of the caecum and ascending colon ranged from 8/hpf

[Inflammatory Bowel Disease (IBD)] to 22.25/hpf (adenomatous polyp). This association was not found to be statistically significant ( $p=0.073$ ). Among the lesions of the transverse colon, adenomatous polyps showed an average mean eosinophil count of 40/hpf. This association was found to be statistically significant ( $p=0.001$ ) when compared with the average mean eosinophil count of other lesions of the transverse colon. Likewise, in lesions of the descending colon and rectum, adenomatous polyps showed an average mean eosinophil count of 15.41/hpf. This association was found to be statistically significant ( $p=0.027$ ).

## DISCUSSION

Eosinophils, initially recognised for their role in parasitic infections and allergic reactions, have emerged as key players in maintaining mucosal homeostasis and modulating immune responses within the GI tract [1]. The observed increase in eosinophil counts from the proximal to distal segments of the GI tract aligns with previous studies, underscoring the unique immunological milieu of each anatomical site [1]. Research indicates that the cecum and ascending colon have the highest eosinophil counts, which may indicate a regional difference in the distribution of eosinophils in the colon [1,6]. The association between elevated eosinophil counts and various GI pathologies underscores their potential role as biomarkers for disease activity and severity.

The present study observed an elevated eosinophil count in the esophagus, particularly in cases of neoplasia. This aligns with findings by Jamali E et al., who reported significant eosinophil counts in Iranian populations, underscoring regional and ethnic variability in eosinophil distribution [4]. Furthermore, studies by Ishibashi S et al., and Jacobse J et al., that explored the role of eosinophils in Oesophageal Squamous Cell Carcinoma (ESCC) reveal similar findings. Ishibashi S team found that higher eosinophil counts correlate with less aggressive tumour behavior and improved survival rates, suggesting a protective, antitumorigenic role [7,8]. Similarly, Jacobse J research indicates that eosinophils can influence the tumour microenvironment by suppressing cancer cell proliferation, promoting apoptosis, and enhancing the activation of immune cells like T cells and natural killer cells [8]. These findings challenge the traditional view of eosinophils as merely proinflammatory and highlight their potential therapeutic implications in cancer treatment. While neoplasia showed higher mean eosinophil counts, present research did not demonstrate elevated eosinophil counts in cases of oesophageal dysplasia. The known association between eosinophils and oesophageal carcinoma highlights the need for further investigation.

The mean eosinophil count varied across different diagnosis, with the highest counts observed in cases of adenomatous polyps and neoplasia, aligning with the findings of Reva I et al., [9]. In contrast, a study by Piazuelo MB et al., showed a strong correlation between the presence of intestinal metaplasia in chronic gastritis and increased eosinophilic infiltration in the stomach [10].

Similarly, research by Iwasaki K et al., highlights the prognostic significance of eosinophils in gastric cancer. Their study reveals that higher eosinophil levels in the tumour microenvironment correlate with better patient prognosis. This suggests that eosinophils may exert beneficial effects by modulating immune responses and influencing the tumour microenvironment, potentially suppressing tumour growth and promoting antitumour immune responses [11]. These findings propose eosinophils as a potential biomarker for predicting outcomes in gastric cancer patients and highlight their therapeutic potential. Understanding the mechanisms underlying eosinophil-mediated effects could lead to novel strategies to improve patient outcomes. Further research is essential to elucidate these mechanisms and explore their clinical implications in oncology.

Additionally, Moorchung N et al., found increased numbers of eosinophils in gastric biopsy specimens from patients with chronic gastritis, indicating their contribution to the inflammatory processes



of the condition [12]. McGovern TW et al., specifically examined *Helicobacter pylori*-associated chronic gastritis and observed significant eosinophil infiltration and degranulation, suggesting the active involvement of eosinophils in the inflammatory response and tissue damage [13]. Together, these findings emphasise the importance of eosinophil evaluation in gastric pathology. However, these findings contrast with the results of this study, where minimal involvement of eosinophils in chronic gastritis was observed, suggesting that alternative inflammatory pathways might be at play. The findings of present study regarding duodenal eosinophil counts are consistent with previous studies highlighting the variability in eosinophil distribution and its association with inflammatory conditions. However, no statistical significance was observed between mean eosinophil counts and histopathological diagnosis in this study. Genta RM et al., emphasised the importance of quantifying duodenal eosinophil content for diagnosing duodenal eosinophilia, supporting present approach [6].

It was observed that a significant connection exists between diagnosis in terminal ileum samples and eosinophil counts, particularly in cases of tuberculosis, non specific acute inflammation, and non specific chronic inflammation. This finding echoes previous research by Babayeva GH et al., and Filippone RT et al., which indicated that eosinophils contribute to the pathogenesis of terminal ileal inflammation, thereby supporting present findings regarding their potential role in intestinal pathology [14,15]. The significant association between eosinophilic infiltrates and specific diagnosis, such as tuberculosis, underscores the diagnostic relevance of eosinophil quantification in terminal ileum specimens.

The present study showed an average eosinophil count of 40 eos/hpf in adenomatous polyps of the transverse colon and 15 eos/hpf in adenomatous polyps of the descending colon. Comparing the findings of this study with previous research, Loktionov A reported similar eosinophil densities in colonic polyps, suggesting a potential role of eosinophils in preneoplastic colonic lesions [1]. Several studies support the role of eosinophils in colorectal pathology. Kurome M et al., and Bilinski C et al., suggest that eosinophilic infiltration is associated with neoplastic colonic polyps and may serve as a marker for disease progression towards high-grade dysplasia or carcinoma [16,17]. Similarly, Moezzi J et al., highlight the significance of stromal eosinophilia in colonic epithelial neoplasms, suggesting a complex role for eosinophils in the pathology of colonic polyps [18]. Dennis KL et al., implicate eosinophils in the inflammatory response within adenomatous polyps, driven by microbial factors and regulated by IL-10-producing T-cells [19]. In paediatric patients, Roma-Giannikou ES et al., also observed increased mucosal eosinophilia in juvenile polyps, suggesting a role for eosinophils in the pathogenesis or inflammatory response in these polyps [20]. Further research is needed to elucidate the mechanisms and clinical implications of eosinophilia in childhood colon polyps.

This highlights the role of eosinophils in mediating or exacerbating the inflammatory environment associated with polyp development. Kızıltas Ş et al., found that tissue eosinophilia decreases as colonic neoplasms progress from tubular adenoma to adenocarcinoma, indicating that a robust eosinophilic presence in less advanced lesions might serve as a host defense mechanism [21]. This pattern suggests that eosinophil density could help predict the malignancy potential of colonic neoplasms. The present study demonstrates similarly concordant findings of relatively lower eosinophilic counts in adenocarcinomas of the transverse and descending colon. Saraiva AL and Carneiro F further elaborate on the complex role of eosinophils in colorectal cancer, indicating that eosinophils can either support tumour progression by contributing to tissue remodeling and creating favorable conditions for cancer growth or exert antitumour effects by releasing substances that inhibit tumour cell proliferation and enhance immune responses against cancer cells [22]. This dual functionality highlights the intricate nature of

eosinophils' involvement in colorectal cancer and emphasises the need for further research to understand their mechanisms and potential therapeutic applications.

The present study does not include any cases of mucosal, mural, or serosal eosinophilic gastroenteritis. Specific histopathological findings, such as patchy involvement, abnormal clustering, and degranulation of eosinophils, were not noted in any of the cases. Collectively, these studies underscore the multifaceted role of eosinophils in gastrointestinal pathology. Eosinophils appear to be involved in both protective and pathogenic processes within the GIT, influencing neoplastic progression and inflammatory responses, with potential therapeutic outcomes. Understanding their dual roles could pave the way for novel diagnostic and therapeutic strategies in colorectal and other gastrointestinal diseases.

### Limitation(s)

It was retrospective and single-centred study with a relatively small sample size and a narrow pathological spectrum. Therefore, the applicability of the results on a larger scale may be limited. Moreover, the reliance on histopathological assessment alone may overlook the dynamic nature of eosinophilic infiltration in response to various stimuli.

### CONCLUSION(S)

The present study sheds light on the distribution and diagnostic significance of eosinophilic infiltration across various segments of the GIT in present population group. While eosinophils are usually detected in varying numbers in most mucosal biopsies of the GIT, the findings of the present study significantly define the limits of eosinophil association with different conditions that can feature significant eosinophilia. Despite the mentioned limitations, present study underscores the clinical relevance of eosinophils in gastrointestinal pathology and highlights the need for further prospective, multicenter studies to validate present findings and understand how eosinophils contribute to gastrointestinal diseases.

### REFERENCES

- [1] Loktionov A. Eosinophils in the gastrointestinal tract and their role in the pathogenesis of major colorectal disorders. *World J Gastroenterol*. 2019;25(27):3503-26.
- [2] Travers J, Rothenberg M. Eosinophils in mucosal immune responses. *Mucosal Immunol*. 2015;8(3):464-75.
- [3] Kazeminezhad B, FalahatianMehrdadi H, Moradi A, Mollasharifi T. Quantity and distribution of eosinophils in the adult human gastric specimens. *Iran J Pathol*. 2022;17(2):166-73.
- [4] Jamali E, Kazeminezhad B, Ahadi M, Moradi A, Khabbazi H. Quantity and distribution of eosinophils in esophageal specimens of adults: An Iranian population-based study. *Iran J Pathol*. 2022;17(2):136-42.
- [5] Yantiss R. Eosinophils in the GI tract: How many is too many and what do they mean? *Mod Pathol*. 2015;28(Suppl 1):S7-S21.
- [6] Genta RM, Sonnenberg A, Turner K. Quantification of the duodenal eosinophil content in adults: a necessary step for an evidence-based diagnosis of duodenal eosinophilia. *Aliment Pharmacol Ther*. 2018;47(8):1143-50.
- [7] Ishibashi S, Ohashi Y, Suzuki T, Miyazaki S, Moriya T, Satomi S, et al. Tumour-associated tissue eosinophilia in human esophageal squamous cell carcinoma. *Anticancer Res*. 2006;26(2B):1419-24.
- [8] Jacobse J, Aziz Z, Sun L, Chaparro J, Pilat JM, Kwag A, et al. Eosinophils exert antitumorigenic effects in the development of esophageal squamous cell carcinoma. *Cell Mol Gastroenterol Hepatol*. 2023;16:961-83.
- [9] Reva I, Yamamoto T, Zvyagintsev D, Kalinin I. Analysis of tissue eosinophils in the structure of gastric polyps. *Archiv Euromedica*. 2021;2:4.
- [10] Piazuolo MB, Camargo MC, Mera RM, Delgado AG, Peek RM Jr, Correa H, et al. Eosinophils and mast cells in chronic gastritis: Possible implications in carcinogenesis. *Hum Pathol*. 2008;39(9):1360-69.
- [11] Iwasaki K, Torisu M, Fujimura T. Malignant tumour and eosinophils: I. Prognostic significance in gastric cancer. *Cancer*. 1986;58(6):1321-27.
- [12] Moorchung N, Srivastava AN, Gupta NK, Malaviya AK, Achyut BR, Mittal B. The role of mast cells and eosinophils in chronic gastritis. *Clin Exp Med*. 2006;6:107-14.
- [13] McGovern TW, Talley NJ, Kephart GM, Carpenter HA, Gleich GJ. Eosinophil infiltration and degranulation in *Helicobacter pylori*-associated chronic gastritis. *Digest Dis Sci*. 1991;36:435-40.
- [14] Babayeva GH, Ibrahimli HI, Guliyev FV, Asadova GV, Mahmudov UR, Hasanov RH, et al. The role of eosinophilic inflammation in inflammatory bowel diseases: Conductor or "first" violin? [Internet]. Eosinophils and their Role in Human Health and Disease [Working Title]. IntechOpen; 2024. Available from: <http://dx.doi.org/10.5772/intechopen.1005563>.

[15]

Filippone RT, Sahakian L, Apostolopoulos V, Nurgali K. Eosinophils in inflammatory bowel disease. *Inflamm Bowel Dis*. 2019;25(7):1140-51.

[16]

Kurome M, Kato J, Nawa T, Fujimoto T, Yamamoto H, Shiode J. Risk factors for high-grade dysplasia or carcinoma in colorectal adenoma cases treated with endoscopic polypectomy. *Eur J Gastroenterol Hepatol*. 2008;20(2):111-17.

[17]

Bilinski C, Burleson J, Forouhar F. Inflammation associated with neoplastic colonic polyps. *Ann Clin Lab Sci*. 2012;42:266-70.

[18]

Moezzi J, Gopalswamy N, Haas RJ Jr, Markert RJ, Suryaprasad S, Bhutani MS. Stromal eosinophilia in colonic epithelial neoplasms. *Am J Gastroenterol*. 2000;95(2):520-23.

[19]

Dennis KL, Wang Y, Blatner NR, Wang S, Saadalla A, Trudeau E. Adenomatous polyps are driven by microbe-instigated focal inflammation and are controlled by IL-10 producing T-cells. *Cancer Res*. 2013;73(19):5905-13.

[20]

Roma-Giannikou ES, Papazoglou TA, Panayiotou JV, van Vliet CP, Kitsiou S, Syriopoulou V, et al. Colon polyps in childhood: Increased mucosal eosinophilia in juvenile polyps. *Ann Gastroenterol*. 2008;21(4):229-32.

[21]

Kızıltas Ş, Sezgin Ramadan S, Topuzoğlu A, Küllü S. Does the severity of tissue eosinophilia of colonic neoplasms reflect their malignancy potential? *Turk J Gastroenterol*. 2008;19(4):239-44.

[22]

Saraiva AL, Carneiro F. New insights into the role of tissue eosinophils in the progression of colorectal cancer: A literature review. *Acta Med Port*. 2018;31(6):329-37.

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