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Prevalence of Colorectal Dysplasia in Patient with Ulcerative Colitis

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Abstract

Background: Ulcerative colitis (UC) is a chronic inflammatory condition of the colon that significantly increases the risk of developing colorectal cancer (CRC) over time. Dysplasia serves as a key marker for CRC risk in UC patients, influenced by factors such as disease duration, extension of disease, severity of disease, age at diagnosis, family history, and ongoing colonic inflammation. This study aims to assess the prevalence of colorectal dysplasia in UC patients and identify the high-risk factors associated with its development.

Methods: This cross-sectional analytical study was conducted at the department of Gastroenterology, BMU, Dhaka, for a period of 12-months following ethical approval. A total of 68 patients with ulcerative colitis 8 years after disease onset were included after getting informed written consent. Data were collected in separated case-record form and analyzed by SPSS-26.

Results: The mean age of the patients was 36.97±11 years, with 57.4% being male and 42.6% female. Histopathological findings showed only inflammation in 61.8% of patients, pseudopolyp in 16.2%, and normal findings in 22%. No dysplasia was observed. Most patients had a disease duration between 8 to 10 years (67.6%) and left side colitis (52.9%). Increased disease duration and left side colitis were significantly associated with inflammatory and pseudopolyp lesion.

Conclusion: This study found no dysplasia in ulcerative colitis patients but identified a significant association between increased disease duration, extension of disease, and the presence of inflammatory conditions and pseudopolyp, highlighting the need for ongoing surveillance and early intervention.

Keywords: Ulcerative colitis; Colorectal dysplasia; Colorectal cancer; Colonoscopy; Histopathology

Introduction

Ulcerative colitis is an idiopathic, chronic inflammatory disorder of the colonic mucosa, which starts in the rectum and generally extends proximally in a continuous manner through part of, or the entire, colon; however, some patients with proctitis or left-sided colitis might have a caecal patch of inflammation [1]. The precise cause of inflammatory bowel disease is unknown; however, genetically susceptible individuals seem to have a dysregulated mucosal immune response to commensal gut flora, which results in bowel inflammation [2]. When the disease is active, the lamina propria of the mucosa becomes heavily infiltrated with a mixture of acute and chronic inflammatory cells. There is a predominant increase in mucosal IgG production, evidence of complement activation, and activation of macrophages and T cells. This immunological activity is associated with the release of a vast array of cytokines, kinas, leukotrienes, platelet activating factors (PAF) and reactive oxygen metabolites. These mediators not only serve to amplify the immune and inflammatory response,

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but they also have direct effects on epithelial function, on endothelial function (which may increase permeability and lead to ischemia), and on repair mechanisms, thus increasing collagen synthesis [3]. Annual incidence of approximately 10 to 20 per 100,000 people [4]. Incidence of ulcerative colitis is higher in developed countries than in developing countries, and in urban versus rural areas. Several environmental factors act as triggers or protective factors for ulcerative colitis, with cigarette smoking being the most consistent. A meta-analysis showed that smoking is protective against ulcerative colitis compared with non-smoking [5]. Patients with ulcerative colitis who smoke tend to have a milder disease course than non-smokers do, and disease activity is often increased in those who stop smoking [6]. Episodes of previous gastrointestinal infection double the risk of subsequent development of ulcerative colitis, which suggests that acute intestinal infection might lead to changes in gut flora, hence triggering the start of a chronic inflammatory process in genetically predisposed individuals [7,8]. Normally, the intestinal immune system maintains equilibrium between tolerance to commensal flora and dietary antigens, and adequate responsiveness to enteric pathogens. Evidence from genetically engineered animal models, which develop chronic intestinal inflammation after colonization with commensal gut bacteria, but remain disease free in bacteria-free conditions, suggests a primary role of non-pathogenic enteric bacteria in the pathogenesis of ulcerative colitis [4,2]. Ulcerative colitis (UC) is a risk factor for the development of inflammation-associated dysplasia or colitis-associated neoplasia (CAN). This transformation results from chronic inflammation, which induces changes in epithelial proliferation, survival, and migration via the induction of chemokines and cytokines. There are notable differences in genetic mutation profiles between CAN in UC patients and sporadic colorectal cancer in the general population. Colonoscopy is the cornerstone for surveillance and management of dysplasia in these patients. There are several modalities to augment the quality of endoscopy for the better detection of dysplastic or neoplastic lesions, including the use of high-definition white-light endoscopy and image-enhanced colonoscopy [9,10]. Colitis-associated neoplasia is thought to be the result of chronic inflammation, which induces changes in epithelial proliferation, survival, and migration via the effect of various chemokines and cytokines. While sporadic CRC develops from one or two foci of dysplasia or adenoma, CAN is believed to develop from multiple dysplastic foci, by which chronically inflamed mucosa produces a field change of molecular alterations and, eventually, histologic changes [11]. Abnormal or disordered growth of cells is termed dysplasia and signifies a precancerous change of the mucosa. Dysplasia is subdivided into low-grade dysplasia (LGD) and high-grade dysplasia (HGD). LGD is characterized by cytological changes, such as nuclear atypia, and carries a relatively low risk for malignant transformation. In contrast, HGD demonstrates cytologic and architectural changes, such as loss of polarity or cribriform gland formation, and is a higher risk for malignant transformation. Occasionally, biopsies will return as indefinite for dysplasia if acute inflammation is present, which can mask or mimic dysplastic changes. Given the high risk of CAN arising from dysplasia in patients with UC, endoscopic screening is recommended for all patients with left-sided (approximately one-third of colon involved) or pan-colonic disease, starting 8 years after symptom onset [9,10]. Therefore, aim of this study is to evaluate the prevalence of colorectal dysplasia detected by colonoscopy biopsy and histopathology in patients with UC.

Methods

A Cross-sectional study Department of Gastroenterology, Bangladesh Medical University, Dhaka from July, 2023 to June 2024, sample size was 68. It is one of the largest tertiary care hospitals in Bangladesh. Patients with ulcerative colitis attended in Department of Gastroenterology, BMU was selected after fulfilling the selection criteria.

Inclusion criteria

- Diagnosed case of ulcerative colitis after 8 years of disease onset.
- Remission of Ulcerative Colitis.
- Patients given informed written consent.

Exclusion criteria

- Previous history of colorectal adenoma or carcinoma.
- Colonoscopy not reaching at least caecum.
- Pregnant woman.

Study Procedure

Patient variables such as age, gender, smoking status, age at UC onset, disease duration at the last visit and at the time of lesion diagnosis, and family history of colorectal cancer (CRC) and inflammatory bowel disease (IBD) were noted. Disease characteristics, including colonic extension, maintenance treatment drugs, presence of pseudo polyps, and diagnosis of dysplasia, were recorded. The extent of disease was classified according to the Montreal classification: E1 (proctitis), E2 (left-sided colitis), and E3 (extensive colitis). UC-associated dysplasia (high-grade dysplasia [HGD] and low-grade dysplasia [LGD]) was diagnosed histopathologically according to the Vienna classification. All patients with colorectal dysplasia in their colonic biopsy samples were reviewed and extracted from the pathology department.

Data Collection

A checklist was prepared for each patient. Informed

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written consent form in Bangla. Informed written consent form in English. Tools for physical examination. Laboratory investigation reports. semi-structured questionnaire.

Data processing and analysis

After collecting all the required data, these were checked, verified for consistency and tabulated using the SPSS version 26 (IBM Corp., Armonk, NY). Frequencies and percentages were calculated as summary measures for the qualitative variables. Arithmetic mean and standard deviation were used to describe the quantitative variables. Chi-square test and Fisher's exact test were used to compare categorical variables. A p value <0.05 was considered as statistically significant. After completion, the data were presented in the form of tables, figures and graphs as necessary.

Ethical consideration

Researcher was careful about the ethical issues related to this study. Before starting this study, the research protocol was approved by the IRB (Institutional Review Board) of Bangladesh Medical University. In this study precaution were taken to protect confidentiality of the participants. Informed written consent were obtained from the study subject without any influences. The contents of the consent requirements were including explanation of the nature and purpose of the study, explanation of the procedure of the study, all information regarding benefits and hazard to all participants, explanation that they have the right to refuse and withdraw or accept to participate in the study & full understanding of the results of this research and their probable welfare implications.

Results

Table 1: Demographic characteristics of the study subject (n=68).

Age Group (years)	Frequency (n)	Percentage (%)
<30	18	26.5
30 to 50	38	55.9
>50	12	17.6
Mean ±SD	36.97±11	
Gender		
Male		57.4
Female		42.6
Occupation		
Service Holder		20.6
Businessman		29.4
Housewife		36.8
Farmer		5.9
Worker		4.4
Other		2.9
Monthly Family Income		
<20,000		47.1
20,000 to 40,000		23.5
>40,000		29.4

Majority of the patients were aged between 30 to 50 years (55.9%) followed by under 30 years (26.5%), and above 50 years (17.6%). Mean age of the patients was 36.97±11 years. Among all the patients, 57.4% were male and 42.6% were female. Among all the patients, 20.6% were service holder, 29.4% were Businessman, 36.8% were housewife, 5.9% were farmer and 4.4% were worker. Besides, 2.9% of the participants had another type of job. Among all, 44.1% of the patients had monthly family income below 20000 takas followed by 29.4% had above 40000 taka and 23.5% had monthly income 20000 to 40000 taka.

Table 2: Distribution of patients according to co-morbidity (n=68).

Co-morbidity	Frequency (n)	Percentage (%)
Hypertension	7	10.3
Diabetes mellitus	15	22.1
No co-morbid illness	46	67.6

Among all, 10.3% of the patients had hypertension and 22.1% had diabetes mellitus whereas 67.6% had no co-morbid illness.

Table 3: Distribution of patients according to Drug history (n=68).

Drug history	Frequency (n)	Percentage (%)
No drug history	46	67.6
Amlodipine	2	2.9
Linagliptin	2	2.9
Metformin	13	19.1
Olmesartan	5	7.4

Majority of the patients had no drug history (67.6%) followed by 19.1% were taking Metformin, 7.4% were taking Olmesartan, 2.9% were taking Amlodipine and 2.9% were taking Linagliptin.

Table 4: Distribution of patients according to clinical history (n=68).

Clinical history	Frequency (n)	Percentage (%)
Duration of disease		
8 to10 years	46	67.6
10 to 15 years	14	20.6
>15 years	8	11.8
Mean ±SD	9.9±3.1	
Extension of ulcerative colitis		
Extensive colitis	15	22.1
Left sided colitis	36	52.9

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Proctitis	17	25
Extra-intestinal involvement		
Joint Pain	36	52.9
None	32	47.1
Treatment modalities		
5-Aminosalicylates	63	92.6
Immunomodulator	5	7.4
Take medication regularly	52	76.5

Mean duration of disease among the patients was 9.9±3.1 years with 67.6% had duration of disease between 8 to 10 years. Among all, 52.9% had left sided colitis, 22.1% had extensive colitis and 25% had proctitis with 52.9% had joint pain. Regarding treatment modalities, 92.6% were having 5-Aminosalicylates and 7.4% were having immunomodulator. Among all, 76.5% were taking medication regularly.

Table 5: Distribution of the patients according to albumin level (n=68).

Serum albumin (g/dl)	Frequency (n)	Percentage (%)
<35	16	23.5
≥35	52	76.5

Among all the patients, 76.5% had albumin level of 35 g/dl or above and 23.5% had below 35 g/dl.

Table 6: Distribution of the patients according to Colonoscopy and Histopathological findings (n=68).

Colonoscopy and Histopathological findings	Frequency (n)	Percentage (%)
Colonoscopy findings		
Normal	44	64.7
Mild UC	11	16.2
Pseudopolyp	13	19.1
Histopathological findings		
Normal	15	22
Inflammation	42	61.8
Pseudo polyp	11	16.2
Dysplasia	0	0

In colonoscopy, macroscopically 64.7% of the patients showed normal, 16.2% showed mild ulcerative colitis and 19.1% showed pseudo-polyp, in histopathology, microscopically 61.8% of the patients showed inflammation, 16.2% showed pseudo-polyp and 22% showed normal. Among all the patients no dysplasia was found in histopathology.

Discussion

Recurrent episodes of mucosal inflammation are the hallmark of ulcerative colitis, a chronic inflammatory bowel disease (IBD). There are various possible outcomes from this persistent mucosal inflammation. Chronic inflammation produces alterations in epithelial proliferation, survival, and migration through the production of chemokines and cytokines, which leads to this metamorphosis [10]. An increased chance of developing inflammation-related dysplasia relates to ulcerative colitis (UC). The aim of this current study was to assess the prevalence of colorectal dysplasia in patient with Ulcerative colitis. Among 68 ulcerative colitis patients, majority of the patients were aged between 30 to 50 years (55.9%) with mean age of the patients was 36.97±11 years. In a previous study, mean age of the patients with ulcerative colitis was 46.7±15.1 years [12]. Another study by Iram et al., age of patients ranged from 20 to 70 years with a mean age of 40.64 ± 13.9 years whereas most of the patients 22 (32%) belong to 21-30 years [13]. Ulcerative colitis may affect any age group. There are peaks at ages 15 to 30 years and then again at ages 50 to 70 years [14]. Among all, 57.4% of the patients were male which was consistent with the previous study suggested that number of male patients was slightly higher than female patients [12]. In the study of Quezada et al., also revealed that ulcerative colitis was more common among male participants [14]. It is possible that a combination of immune response variations, hormonal variances, and genetic predispositions contribute to the higher prevalence of ulcerative colitis (UC) in men. A role is also played by environmental and lifestyle factors, such as nutrition and smoking behaviors. This discrepancy may also be influenced by variations in the gut microbiomes of the sexes and the ways in which they seek medical attention [15]. Among all the patients, in colonoscopy 16.2% of the patients showed mild ulcerative colitis and 19.1% showed pseudo-polyp whereas in histopathology, 61.8% of the patients showed inflammatory condition and 16.2% showed pseudo-polyp. Among all the patients no dysplasia was found. In a previous study, among 801 patients with ulcerative colitis only 7 (0.85%) had dysplasia [12]. Another study also documented that patients with UC have lower incidences of adenomatous colon polyp, dysplasia and CRC [16]. Patients with UC are known to have a higher risk of colorectal cancer (CRC) than the general population [17]. According to a past meta-analysis released by Eaden et al., this risk is high [18]. However, the risk of colorectal cancer (CRC) in individuals with UC has significantly decreased over the past few decades, according to a recent meta-analysis of 58 publications from referral centers and population-based cohorts [19]. But previous study conducted in Pakistan

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revealed that out of 68 cases of ulcerative colitis, 38 (56%) cases were diagnosed as negative for dysplasia, 20 cases (29%) were diagnosed as positive for low grade dysplasia, and 10 (15%) cases were diagnosed as positive for high grade dysplasia [13]. The prevalence of colorectal dysplasia varies between countries due to genetic differences, environmental factors, healthcare access and quality, surveillance practices, and the overall prevalence of inflammatory bowel disease (IBD). Socioeconomic status and levels of awareness and education about IBD also play significant roles [11]. Mean duration of disease among the patients was 9.9±3.1 years with 67.6% had duration of disease between 8 to 10 years. Among all, 52.9% had left sided colitis, 22.1% had extensive colitis and 25% had proctitis with 52.9% had joint pain. Whereas increased duration of disease and left side colitis were found significantly associated with inflammatory lesion which was consistent with the previous studies [11,18]. Another study also suggested that colorectal dysplasia, a precancerous condition in the colon and rectum, is strongly associated with the duration and severity of inflammatory bowel diseases such as ulcerative colitis (UC) [12]. Another study demonstrated that the duration of UC and geographical location are independent risk factors for the transformation of UC into CRC [20].

Conclusion

This study concluded that in patients with ulcerative colitis, inflammatory conditions were the most common histopathological findings, with no cases of dysplasia identified. Increased disease duration and moderate to severe disease activity were significantly associated with the presence of inflammatory lesion. The absence of dysplasia suggests that, within this cohort, dysplasia may not yet have developed, though long-term monitoring remains essential due to the increased risk of colorectal cancer in ulcerative colitis patients.

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