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A Family History of Polyps in Patients with Colorectal Serrated Polyps Augments the Risk of Colon Cancer in Jordan

Faten Mohamed Mohamed Elnozhá[1], Ahmed Abu Ryash[2], Lashin Saad Ali[3], Walid Elsayed Hussein Ahmed Arafat[4], Rasha M. Attia[5], Mohamed Ali Elsayed[6], Amina Mohamed Thabet Abdelhafez[7], Tarek H. Mahmoud[*8], and Samiha Mohamed Ibrahim Abdelkader[9]

[1]Associate Professor of Physical Therapy for Internal Medicine, Chest, Cardiology and Geriatrics. Faculty of Allied Medical Science, Department of Physical Therapy, Al Aqaba University of Technology, Jordan.
[2]Lecture of Allied medical sciences of Isra University, Jordan.
[3]Depatment of basic medical science-faculty of dentistry, Al-Ahliyya Amman university- Amman-Jordan.
[4]Assistant professor, Department of Physical Therapy for cardiopulmonary and geriatrics, Faculty of Allied Medical Sciences, Isra university, Jordan.
[5]Assistant Professor of physical therapy for basic science, Faculty of Allied Medical Science, Philadelphia University, Jordan.
[6]Assistant professor of physical therapy for pediatrics, Faculty of Allied Medical Science, Philadelphia University, Jordan.
[7] Professor of Pediatric Nursing, Faculty of Nursing, Isra University.
[*8]Lecturer of Physical Therapy, Department of Physical Therapy for Internal Medicine, Chest and Cardiology, Faculty of Physical Therapy, Deraya University, Minia, Egypt.
[9]King Saud university, College of Applied Medical Sciences, Department of Re habilitation Health Sciences.

Abstract

Background: Colorectal cancer stands as the third most prevalent form of cancer globally. Among its variants, advanced adenomas, a subtype of colorectal polyps, present an escalated risk of colorectal neoplasia. Consequently, current professional guidelines advocate for earlier and more comprehensive screening for First-Degree Relatives. Consequently, current professional guidelines advocate for earlier and more comprehensive screening of individuals with advanced adenomas, akin to those with colorectal cancer. However, there is a lack of understanding regarding the familial risk associated with polyps, especially advanced serrated polyps, thereby creating a significant gap in screening strategies for these high-risk individuals.

Aims: This research seeks to delve into the intricate relationship between colorectal cancer and serrated polyps, while also examining the predictive role of family history in patients with adenomatous polyps.

Material and methods: Samples and data were meticulously collected from 61 patients diagnosed with colorectal cancer. Utilizing SPSS, data analysis conducted with the Functional Assessment of Cancer Therapy Colorectal scale serving as the primary assessment tool.

Results: The regression analysis findings suggest that family history does not serve as a reliable predictor of colorectal polyps. Conversely, colorectal polyps emerge as a significant predictor of colorectal cancer. Furthermore, t-test analysis reveals notable associations between individual differences, such as age, gender, and lifestyle factors, and colorectal polyps.

Conclusion: A comprehensive understanding of the relationship between colorectal cancer and serrated polyps, alongside predictive role of family history, holds paramount importance in enhancing screening efforts for high-risk groups, by bolstering screening strategies.

Keywords: Family history, polyps, colorectal serrated polyps, colorectal carcinoma.

Abbreviations:
CRC: Colorectal Cancer
FDRs: First-Degree Relatives
AAs: Advanced Adenomas

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Introduction:

Colorectal Cancer (CRC) is a malignant tumor in the colon and rectum. In 2018, notably in the United States, it was the second most frequent cancer death cause and the third most frequent carcinoma globally[1]. Age over 50, dietary factors (high meat and fat intake and low dietary fiber intake), alcohol use, obesity, and a family history of CRC are risk factors for the disease. Genetic and environmental factors influence CRC. Increased CRC screening use has significantly declined CRC incidence and mortality in the United States over the past 20 years[2]. Because the screening programs cannot reach all eligible people, the burden of CRC disease remains substantial. Most national efforts focus on screening people with average risk, but they frequently miss essential population subgroups at elevated risk. First-degree relatives (FDRs) of people with CRC and advanced adenomas (AAs) are one such high-risk group[3]. Despite the rising prevalence, CRC mortality has decreased in affluent countries with recent improvements in early detection screens and treatment choices. Those with a hereditary propensity for the tumor can take preventive actions thanks to genetic testing and better family history documentation. In the meantime, the general populace can lower their risk by consuming less red meat, alcohol, and tobacco while increasing their fiber intake, healthy foods, and particular vitamins and minerals. These hyper-proliferative cells give rise to benign adenomas that can over time, develop into carcinoma and metastasis[4]. Cancer develops when particular epithelial cells pick up several selectively advantageous genetic or epigenetic alterations[5]. The colon's essential ingredients are water, leftover minerals, and nutrients in the chyme. The progenitor and stem cells of the colon are near the bottom of the crypt. Self-renewal is a function of these pluripotent cells[6]. The progenitor cells move out of the crypt and up the villus as they develop into specialized epithelial cells. Enterocytes, Paneth, goblet, enteroendocrine cells, and enteroendocrine cells are examples of differentiated epithelial cells. At around 14 days, these cells undergo apoptosis once they reach the apex of the villus[7]. A gradient of signaling proteins, the most prevalent of which are Want, BMP, and TGF-B[8,9], strongly regulates this process. FDRs of CRC patients are more than two times more likely to get CRC. The risk rises if the patient's age falls and there are two or more impacted FDRs. As a result, screening is advised by age 40 or 10 years before the relative with the CRC diagnosis. The risk of FDRs in AA patients raised, and early screening advised[12]. Tubular adenoma less than 1 cm, or histology, defines As, a separate group of colorectal polyps (any adenoma with villous histology or high-grade dysplasia). They are more likely to develop neoplasia and its FDRs; thus, earlier and more frequent screening is necessary[13]. Many serrated polyps in the colon are a symptom of the uncommon genetic condition known as serrated polyposis syndrome (SPS). Increased colorectal cancer (CRC) risk linked to serrated polyps[14]. Due to the potential for an increased risk of colorectal colon cancer, patients with SPS and their first-degree relatives (FDR) should be under rigorous observation[15]. In these patients, conventional adenomas frequently seen alongside serrated polyps and can make up to 50% of the overall polyp count[16]. As CRC is the most common cancer, easily diagnosed during a routine colonoscopy and first arises from lesions, it may be avoided[17,18]. A serrated polyp is a lesion that appears to have saw-toothed or serrated colonic crypts under a microscope. Examples of these lesions include the sigmoid colon and the rectal hyperplastic polyp, which account for 20% of all cases of serrated polyps. Serrated polyps are typically sessile or flat lesions affecting the right colon[19,20]. The current study emphasizes the connection between colorectal serrated polyps and cancer development, particularly in patients with a family history of adenomatous polyps.

Patients and Methods:

Tools, resources, and needs

The subject of CRC risk and screening communication has not received as much attention in research as genetic test results in families with a known inherited propensity. Thus, there is an urgent need for patient and provider education and good communication. The tools to raise awareness of the early screening recommendations for FDRs of patients with ACP among gastroenterologists and primary care physicians have recently created[21]. According to various polyp subtypes, the National Colorectal Cancer Roundtable offers free, downloadable template letters explaining the colonoscopy and pathology findings, the risks involved, and treatment suggestions for patients and FDRs[22]. Reaching more people who are eligible for early screening requires a concentrated effort to enhance communication of the ACP results and particular recommendations for family members. Further research needed to determine if and how patients with AA convey danger to unaffected family members and whether these people are aware that they are at risk. Finding the best and most practical ways to communicate with FDRs will be essential. It is time to improve our knowledge of the most effective ways to involve patients and their families in light of the numerous technological advancements available for communicating with and educating patients.

Patients' information:

A total of 61 patients with colorectal cancer were recruited, and data was collected from the Hospital of Al Basher Government of Jordanian Ministry of Health. A purposive sampling method was employed, targeting individuals who met the inclusion criteria—specifically, those with colorectal cancer and a history of adenomatous polyps—to explore relevant associations. Patients with other types of cancer were excluded to maintain the study's focus and reduce confounding variables. Ethical considerations were strictly followed, including obtaining informed consent, ensuring patient confidentiality, and securing approval from the relevant hospital departments to conduct the research.

The sample size selected through the guidance of literature:

Inclusion criteria: Patients with colorectal cancer and a history of adenomatous polyps were included.

Exclusion criteria: Patients suffering from other types of cancer excluded from this research. The study conducted following the proper ethical guidelines with consideration of confidentiality, informed consent and clarification of the questions. Respective departments granted permission to run this study.

Ethical Approval: The Ethical Board of Jordanian Ministry of Health approved the study, and informed consent acquired from all participants. Informed written consent had taken from each participant in the study. This work carried out following. The Code of Ethics of the World Medical Association (Declaration of Helsinki) for studies involving humans.

Survey methodology: A general survey implemented where questions were asked to the patients, and the researcher filled out the questionnaires himself. Each questionnaire took around 15 minutes, while data collection done within two months.

Screening for colorectal cancer (CRC): Patients' history of colorectal tumors or inflammatory bowel disease and a family history of CRC were included in screening for CRC. All patients with a family history of polyposis syndrome from 25 years old were included.

Socio-demographic questionnaire: The questionnaire (Cohort description) comprised gender, age, marriage status, colorectal screening, and history of colorectal cancer or adenomatous polyps pertaining to the patients in the family.

It has five subscales:

1= Physical well-being, 2= Social/Family well-being, 3= Emotional Well-being, 4= Functional Well-being and 5= Colorectal Cancer Subscale.

Diagnosis of SPS:

World Health Organization, in 2018, enumerated some clinical criteria, which our study used as our guidelines, which revealed[23]: - ≥5 serrated lesions proximal to the sigmoid colon after histological diagnosis, 2 of which should be ≥10 mm in diameter. - - In a patient with FDR and SPS, Serrated polyps lie proximal to the sigmoid colon. >20 serrated polyps diagnosed throughout the colon.

Data analysis:

The data are expressed as Mean ± SD/SEM, and the significant values were considered at $p<0.05$. One-way analysis of variance (ANOVA) by Duncan's test evaluated the difference between the mean values of the results. The investigation occurred for three measurements using SPSS software version 16. Data are also filtered via Q-Q plots, in addition to regression analysis, which is also used to measure the impact of variables[23].

Results:

Descriptive statistics related to gender, age, marital status and screening for colorectal cancer (CRC) using a socio-demographic questionnaire.

As illustrated in Figure 1A, the influence of age on colorectal polyps The group of patients having colorectal polyps shows that 40 participants' responses fall into the category of age range between 25 and 64 years old with 80.3% and 21 participants are all in the 65+ age range with 19.7%. As illustrated in Figure 1B, the effect of gender on colorectal polyps' shows that female patients have a lower suspected incidence of colorectal polyps than male patients, with 14 and 47 percentile probabilities, respectively. As illustrated in Figure 1C, the group of patients having colorectal polyps shows that seven patients fall into the category of marriage status: divorced (11.5%), 50 participants are married with a good percentage of 85.2%, and four are widowed (3.3%). Colorectal polyp patients vary in gender, as there are 14 female participants at 23% and 47 males at 77%. As illustrated in Figure 1D, the patients who responded "no" were 46 participants (75.4%) to the questions about colorectal polyp scanning, and those who answered yes were 15 (24.6%).

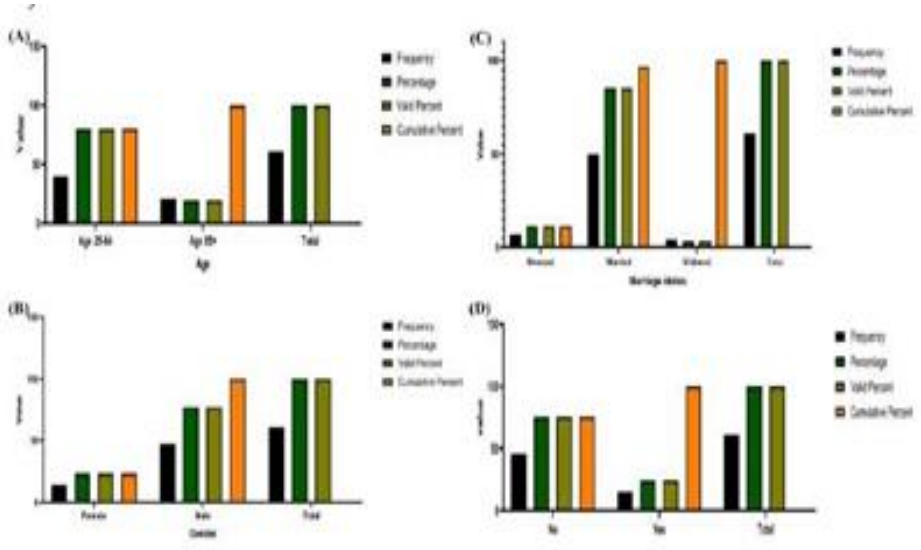
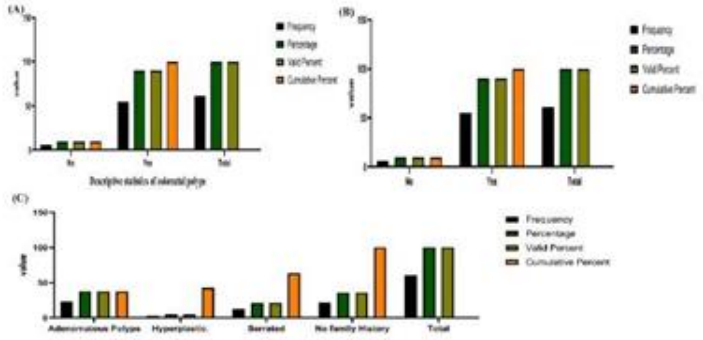


Figure 1: (A) The influence of age on colorectal polyps (B) The effect of gender on colorectal polyps (C) The influence of Marriage status on colorectal polyps (D)The impact of CP screening on colorectal polyps. Descriptive statistics of colorectal polyps, serrated polyps and family polyps



Figures 2: (A) and (C) showed descriptive statistics of colorectal polyps.

Most patients have serrated polyps found in 55 people (90.2%), and only six do not. Figure 2B shows the effect of family history on colorectal polyps. For the question: What kind of colorectal polyps do you have in your family? Most patients stated that they have adenomatous polyps, which is 37.7%. Some people do not have colorectal polyps in their family history, which is 36.1%.

Individual differences in colorectal polyps are likely to predict correlated colorectal cancer scale Results of the well-being effect on colorectal polyps were performed using regression (DV= fact c) and demographic variable (IV= demographic variables) analysis. Overall, the model was significant ($F(8, 52) = 2.59$, $p<0.05$), with an R-value of 0.534 and an R^2 value of 0.285. The value of R2 represents that the model accounted for 28% variance, where colorectal polyp screening ($\beta = -8.46$, $p<0.005$) and serrated polyps ($\beta = 8.25$, $p<0.05$) turned out to be significant predictors of FACT-C. It shows that the patients with colorectal polyp cancer significantly predicted by colorectal polyp screening and serrated polyps; this indicated by a positive correlation, as shown in Table (1).

Table 1: Predictors of FACT-C scale for colorectal polyps.

Variable	Category	Number of Participants	Percentage
Age	25 - 64 years old	40	80.3%
	65+ years old	21	19.7%
Gender	Female	14	23%
	Male	47	77%
Marital Status	Divorced	7	11.5%
	Married	50	85.2%
	Widowed	4	3.3%
Screening for CRC	No	46	75.4%
	Yes	15	24.6%

Table 2: Summary of Well-being Variables

Variable	Minimum Range	Maximum Range	Mean
P. Well-being	16.00	31.00	22.27
S. Well-being	14.40	32.40	26.28
E. Well-being	7.20	30.00	17.28
F. Well-being	14.40	39.00	28.40
Colorectal Well-being	18.00	36.00	24.75

There is likely to be a correlation between individual differences and patients having colorectal polyps.
As shown in Tables (3-4), a correlation analysis found that gender significantly positively correlated with the patient's marital status ($r = 0.4$, $p < 0.001$). Serrated polyps were also positively correlated with colorectal polyp types $r = 0.316$, $p<0.013$). Regression analysis used to test if patients with adenomatous polyps in their family history significantly increased the risk of colorectal polyps. The regression analysis results indicated that overall, the model was insignificant ($F (1, 59) = 1.69$, $p = 198$).

Table 3: Correlation between gender, marital status, and polyp types.

Variables	Correlation (r)	p-value
Gender & Marital Status	0.4	< 0.001
Serrated Polyps & CRC Types	0.316	< 0.013

The regression analysis results: Model significance: $F (1, 59) = 1.69$, $p = 198$
The difference in having serrated polyps in terms of "Yes" or "No" groups and all subcategories of the Fact-C scale.

Table 4: Serrated polyps and FACT-C scale comparison.

Variables	Mean (M)	Standard Deviation (SD)	t-value	p-value	Effect Size (d)
Positive Emotional Well Being	Without Serrated Polyps (Group 1)	25.67	11.99		
	With Serrated Polyps (Group 2)	23.08	4.83	1.036	< 0.01

The independent sample t-test results:
- t-value: 1.036
- p-value: < 0.01
- Effect size (Cohen's d): Large (d = 18.9)

Histopathological study of the colon:

Figure 3 represents an inflammatory hyperplastic colon and polyps. Figures 3 (A) and (B) showed that the "colon" is consistent with inflammatory polyps and adenomatous changes in individual glands. Figures 3 (C) and (D) showed the middle of the colon, serrated lesions, in line with the traditional serrated adenoma. "Ascending colon near the hepatic

flexure" hyperplastic polyps. Figures 3 (E) and (F) showed "rectal" tubular adenomas (low-grade intraepithelial neoplasia) and hyperplastic polyps. "Colon 35cm" villous adenoma (high-grade intraepithelial neoplasia) Figure 4 demonstrated colon and colorectal polyps. Figure 4 (A) showed an ascending colon and hyperplastic polyps. Figure 4 (B) showed an "ascending colon" villous-tubular adenoma (low-grade intraepithelial neoplasia). Figure 4 (C) showed hyperplastic polyps considered Peutz-Jeghers polyps. Figure 4 (D) showed "ascending colon" tubular adenoma (low grade intraepithelial neoplasia), "ascending colon" tubular adenoma (low-grade intraepithelial neoplasia), and Figure 4 (E) showed descending colon" villous tubular adenoma (low-grade intraepithelial neoplasia).

Table 5: Description of Histopathological Findings

Figure	Location/Description	Histological Classification
3 (A)	Inflammatory polyps and adenomatous changes	
3 (B)	Inflammatory polyps and adenomatous changes	
3 (C)	Serrated lesions in the middle of the colon	Traditional serrated adenoma
3 (D)	Serrated lesions in the middle of the colon	Traditional serrated adenoma
3 (E)	Hyperplastic polyps near hepatic flexure	
3 (F)	Hyperplastic polyps near hepatic flexure	
4 (A)	Hyperplastic polyps in ascending colon	
4 (B)	Villous-tubular adenoma in ascending colon	Low-grade intraepithelial neoplasia
4 (C)	Peutz-Jeghers polyps	Hyperplastic
4 (D)	Tubular adenoma in ascending colon	Low-grade intraepithelial neoplasia
4 (E)	Villous-tubular adenoma in descending colon	Low-grade intraepithelial neoplasia

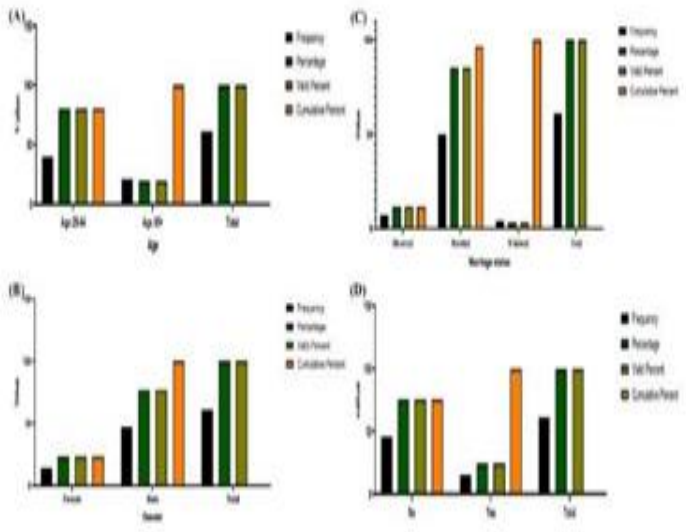


Figure 3: Influence of age, gender, marital status, and screening on colorectal polyps.
(A)The influence of age on colorectal polyps (B) The effect of gender on colorectal polyps (C) The influence of Marriage status on colorectal polyps (D)The impact of CP screening on colorectal polyps.

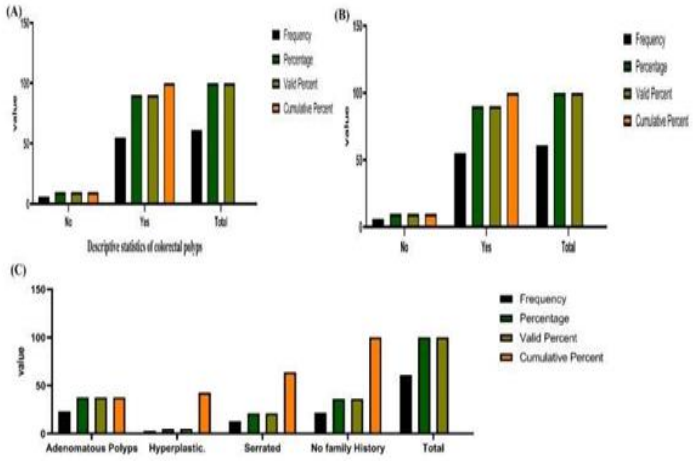


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Figure 4: Descriptive statistics and family history effect on colorectal polyps.

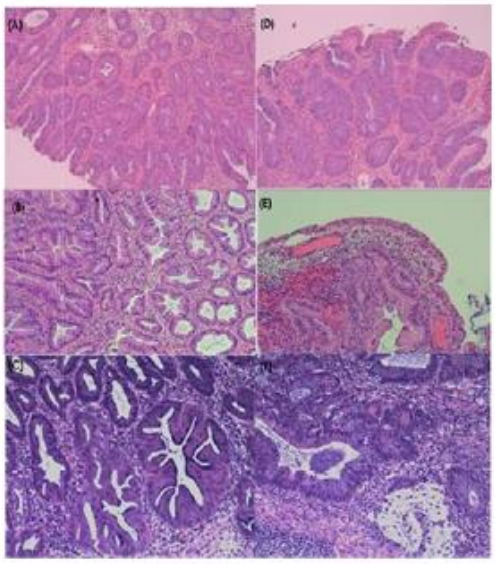

Figure 5: Colon and polyps, including serrated hyperplastic polyps, and adenomas.
Colon and polyps inflammatory and hyperplastic, (A) and (B) showed that the "Colon" is consistent with inflammatory polyps and adenomatous changes in individual glands, (C) and (D) middle of the colon serrated lesion in line with the traditional serrated adenoma, Ascending colon near the hepatic flexure" hyperplastic polyps, (E) and (F) Rectal tubular adenoma (low grade intraepithelial neoplasia) and hyperplastic polyp, Colon 35cm villous adenoma (high-grade intraepithelial neoplasia).

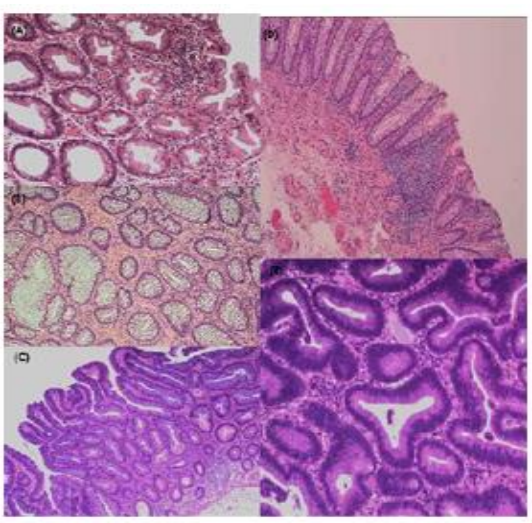

Figure 6: Various types of polyps in the colon, including hyperplastic, adenomas, and villous adenoma.

Colon and colorectal polyps (A): Ascending colon and hyperplastic polyps, (B): Ascending colon 1" villous-tubular adenoma (low-grade intraepithelial neoplasia),2. "Ascending colon 2" villous-tubular adenoma (low-grade intraepithelial neoplasia), (C): Hyperplastic polyps are considered as Peutz-Jeghers polyps, (D): "Ascending colon" tubular adenoma (low-grade intraepithelial neoplasia), tubular adenoma (low-grade intraepithelial neoplasia), (E): Descending colon" villous tubular adenoma (low-grade intraepithelial neoplasia).

Discussion:

This paper discusses the global health challenges posed by cancers of the colon and rectum, with both incidence and mortality rates expected to rise. Lifestyle changes, including avoiding risk factors such as physical inactivity and obesity, and promoting healthy habits like regular exercise and dietary fiber intake, have contributed to a decline in CRC incidence. Additionally, advancements in treatment and early detection have led to reduced mortality rates, especially in high-HDI nations [1]. Individuals with first-degree relatives (FDRs) who have succumbed to CRC face a significantly higher risk of developing the disease themselves. This risk remains consistent across various study designs and demographics, with affected FDRs having two to three times higher CRC risk compared to controls. The location of the index cancer (colon vs. rectum) influences the risk, with colon cancer relatives exhibiting a higher risk ratio compared to rectal cancer relatives. However, no significant difference was found in the colon cancer risk ratio based on the tumor site (right side vs. left side)[24].

CRC risk is significantly associated with age and family history at diagnosis, with younger individuals and those with affected first-degree relatives facing higher relative risks. Strong correlations exist between familial CRC risk, age at diagnosis, and the number of affected family members, highlighting the importance of considering these factors in assessing CRC risk[25,26]. Computer models aid in assessing CRC likelihood and genetic counseling, particularly for familial adenomatous polyposis (FAP) and Lynch syndrome, although some families with familial CRC may lack specific genetic associations[24-27]. Challenges persist in accurately assessing family history data due to potential unawareness or inaccuracies. Colonoscopy usage may increase detection of precancerous colon polyps while reducing CRCs, but awareness of polyp history is lower compared to cancer. Patient-reported family history of colon cancer is generally accurate, but medical records should confirm it, especially for reproductive tract malignancies relevant to assessing Lynch syndrome risk. Patients with newly discovered CRC suspected of having a genetic cancer syndrome can undergo evaluation techniques, with genetic tests available to corroborate suspicions following guidelines from the American College of Medical Genetics and Genomics[24,29]. The criteria aim to identify individuals necessitating genetic counseling referral, with specialized gene-directed testing valuable for diagnosis, especially in cases of multiple polyps (>20) or suspected Lynch syndrome. However, diagnosis can be challenging with ambiguous clinical presentations[14]. Despite advancements, CRC remains a significant health burden, with an estimated 147,950 new cases and 53,200 deaths expected in 2020 in the US, ranking as the second leading cause of cancer-related deaths[15]. Existing screening programs primarily target average-risk individuals, leaving high-risk populations like FDRs of CRC patients and individuals with AAs underserved[16]. FDRs face increased CRC risk, warranting early screening starting at age 40 or 10 years before the youngest affected relative's diagnosis. Similarly, individuals with AAs require early and regular

screening interventions due to their elevated CRC risk[17]. The global CRC prevalence is rising, especially in developing nations adopting a "western" lifestyle. However, mortality rates have declined in more developed countries, credited to advancements in early detection screening and treatment[14-17]. Individuals with a hereditary predisposition to CRC can benefit from preventive measures facilitated by genetic testing and improved documentation of family history. Concurrently, adopting healthier lifestyle habits such as reducing consumption of red meat, alcohol, and tobacco, while increasing intake of fiber and nutritious foods, can mitigate the risk of developing CRC[17]. CRC development involves specific epithelial cells acquiring genetic or epigenetic alterations, leading to cancer formation. Benign adenomas, originating from hyper proliferative cells, can progress into cancer over time due to abnormally high rates of replication and survival[18].Improved communication between patients, providers, and family members is crucial in identifying and screening high-risk individuals based on positive family history[1]. Studies emphasize the higher risk of colorectal cancer in individuals with a family history of colorectal adenomas, particularly among first-degree relatives (FDRs)[32]. Diagnostic criteria are crucial for identifying patients with adenomatous polyps in high-risk families, enabling timely screening and polyp removal to reduce colorectal cancer risk[23]. Research explores the association between colorectal cancer and serrated polyps, indicating that colorectal cancer strongly predicts neoplastic serrated polyps, and early screening can lower the risk of colorectal cancer in patients with serrated polyps[36,37]. There is a likely correlation between patient characteristics, such as having serrated polyps, and receiving early therapies for serrated polyps[38]. Early detection of colorectal polyps effectively reduces their incidence and the risk of colorectal cancer[39,40]. Family history may predict colorectal cancer among individuals with colorectal polyps or cancer, indicating an association between colorectal polyps and cancer[39,40]. Serrated polyps increase the risk of colorectal cancer, particularly in individuals with a history of serrated polyposis syndrome[32].Behavioral modifications, such as maintaining a healthy weight and engaging in physical activity, can significantly reduce CRC risk by up to 50%[4]. Dietary changes, including increased intake of antioxidants from fruits and coffee, as well as calcium and vitamin D from supplements or low-fat dairy products, can further lower CRC risk[4]. Adenocarcinomas, originating from colon mucosa epithelial cells, account for over 90% of colorectal cancers. Adenomas, which can progress to adenocarcinomas, are clonal lesions typically displaying enlarged nuclei and stratified arrangement along the basement membrane[40]. Serrated polyps encompass a diverse group of lesions and contribute to a better understanding of colorectal polyps and their association with colorectal cancer, emphasizing the importance of early detection and surveillance in high-risk individuals, histologically, hyperplastic polyps (HPs) characterized by elongated, straight crypts with luminal serration, particularly evident in the upper sections, resembling surface maturation. HPs classified into micro vesicular, goblet cell, and mucin-poor subtypes without significant clinical implications[40]. Peutz-Jeghers syndrome, marked by hamartomatous polyps in the GI tract, pigmented mucocutaneous lesions, and increased cancer risk, rarely shows dysplasia in these polyps[39]. Serrated polyposis syndrome, defined by multiple serrated polyps, carries increased cancer risk, especially in those with a first-degree relative with the syndrome[34], high-grade dysplasia, the direct precursor to invasive colorectal adenocarcinoma, is characterized by cytologic atypia, architectural complexity, crowded glands, cribriform growth patterns, spherical nuclei, coarse chromatin, and prominent nucleoli. Dysplastic glands may contain necrotic material[40].

Conclusion:

Our study investigated the connection between colorectal polyps and colorectal cancer and the significance of family histories. The results depict a significant link between colon polyps and colorectal cancer and the importance of early detection and screening in preventing colorectal cancer. Nevertheless, they have also suggested that family history has no significant role in lowering colorectal cancer risk. This study further emphasizes the significance of early colorectal polyp detection, also, it suggests that other factors contributing to colorectal cancer, such as food, living arrangements and psychological issues, should be considered in addition to family history. This research's conduct is fraught with many difficulties. Identifying particular cancer patients from just two hospitals was tough, and getting their consent to participate in the study was even more difficult because acquiring information from them while receiving treatment was challenging. It was difficult to discover that most persons had a family history of colorectal cancer. Future researchers can concentrate on qualitative research to understand their issues. It would be more appropriate to perform the study among relatives of patients with a family history of colorectal cancer and consider various cancer combinations. Future researchers would benefit from combining the prevalence of colorectal cancer with the population's demographics.

Declarations:

Ethics approval and consent to participate

Not applicable

Consent for publication

Not applicable

Availability of data and materials

All data and materials are fully presented in the manuscript

Competing interests
The authors declare that they have no conflict of interest.

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The study plan and experiment design were done by all authors, who read and approved the final version of the manuscript

References:

(1) Siegel RL, Miller KD, Jemal A. Cancer Statistics. CA Cancer J Clin. 2019; 69:7–34. doi: 10.3322/caac.21551.
(2) Kolb JM, Molmenti CL, Patel SG, Lieberman DA, Ahnen DJ. Increased Risk of Colorectal Cancer Tied to Advanced Colorectal Polyps: An Untapped Opportunity to Screen First-Degree Relatives and Decrease Cancer Burden. Am J Gastroenterol. 2020 Jul;115(7):980 988. doi: 10.14309/ajg.00000000000000639.
(3) National Colorectal Cancer Roundtable. 80% by 2018 (http://nccrt.org/what-we-do/80 percent-by-2018/). Accessed November 1, 2019.doi.org/10.1016/j.cgh.2020.06.053
(4) Rawla P, Sunkara T, Barsouk A. Epidemiology of colorectal cancer: incidence, mortality, survival, and risk factors. Prz Gastroenterol. 2019;14(2):89-103. doi: 10.5114/pg.2018.81072.
(5) Ewing I, Hurley JJ, Josephides E, Millar A. The molecular genetics of colorectal cancer. Frontline Gastroenterol. 2014;5:26–30.doi.org/10.1136/flgastro-2013-100329
(6) Peifer M. Developmental biology: colon construction. Nature. 2002;420:274–5. doi.org/10.1038/420274a
(7) Kosinski C, Li VS, Chan AS, et al. Gene expression patterns of human colon tops, basal crypts, and BMP antagonists as intestinal stem cell niche factors. Proc Natl Acad Sci USA. 2007;104(39):15418–23.doi: 10.1073/pnas.0707210104
(8) Medema JP, Vermeulen L. Microenvironmental regulation of stem cells in intestinal homeostasis and cancer. Nature. 2011;474:318–26. doi.org/10.1038/nature10212
(9) Sideris, M., & Papagrigoriadis, S. (2014). Molecular Biomarkers and Classification Models in the Evaluation of the Prognosis of Colorectal Cancer. Anticancer Research, 34(5), 2061-2068. http://ar.iiarjournals.org/content/34/5/2061.abstract
(10) Kekelidze M, D'Errico L, Pansini M, et al. Colorectal cancer: current imaging methods and future perspectives for the diagnosis, staging and therapeutic response evaluation. World J Gastroenterol. 2013;19:8502–14. doi: 10.3748/wjg.v19.i46.8502
(11) Colussi D, Brandi G, Bazzoli F, Ricciardiello L. Molecular pathways involved in colorectal cancer: implications for disease behavior and prevention. Int J Mol Sci. 2013; 14: 16365–85. doi.org/10.3390/ijms140816365
(12) Baglietto L, Jenkins MA, Severi G, et al. Measures of familial aggregation depend on definition of family history: Meta-analysis for colorectal cancer. J Clin Epidemiol 2006;59:114 24.doi.org/10.1016/j.jclinepi.2005.07.018
(13) Rex DK, Boland CR, Dominitz JA, et al. Colorectal cancer screening: Recommendations for doctors and patients from the U.S. Multi-Society Task Force on Colorectal Cancer. Gastroenterology 2017;153:307–23.doi.org/10.1053/j.gastro.2017.05.013
(14) Carballal, S., et al., Colorectal cancer risk factors in patients with serrated polyposis syndrome: a large multicentre study. Gut, 2016; 65(11): 1829-1837.doi.org/10.1136/gutjnl-2015 309647
(15) Deen, K.I., et al., Colorectal cancer in the young, many questions, few answers. World J Gastrointest Oncol, 2016; 8(6): 481-8. doi: 10.4251/wjgo.v8.i6.481
(16) Muller, C., et al., Risk of Colorectal Cancer in Serrated Polyposis Syndrome: A Systematic Review and Meta-analysis. Clin Gastroenterol Hepatol, 2022; 20(3): 622-630 e7.doi.org/10.1016/j.cgh.2021.05.057
(17) Hiraoka, S., et al., The presence of large serrated polyps increases risk for colorectal cancer. Gastroenterology, 2010; 139(5): 1503-10, 1510 e1 3.doi.org/10.1053/j.gastro.2010.07.011
(18) Aran, V., et al., Colorectal Cancer: Epidemiology, Disease Mechanisms and Interventions to Reduce Onset and Mortality. Clin Colorectal Cancer, 2016; 15(3): 195 203.doi.org/10.1016/j.clcc.2016.02.008
(19) Bateman, A.C., The spectrum of serrated colorectal lesions-new entities and unanswered questions. Histopathology, 2021. 78(6): 780-790.doi.org/10.1111/his.14305
(20) JEG, IJ, et al., Detection rate of serrated polyps and serrated polyposis syndrome in colorectal cancer screening cohorts: a European overview. Gut, 2017; 66(7): 1225-1232 doi.org/10.1136/gutjnl-2015-310784
(21) Molmenti CL, Kolb JM, Karlitz JJ. Advanced colorectal polyps on colonoscopy: A Trigger for earlier screening of family members. Am J Gastroenterol 2020;115:311 4.DOI: 10.14309/ajg.0000000000000467

(22) Molmenti CLSP Ahnen DJ, Karlitz J, et al.; National Colorectal Cancer Advanced Adenoma Working Group, American Cancer Society. Advanced Colorectal Polyp: GI Brief. American Cancer Society, 2019DOI: 10.14309/ajg.0000000000000639
(23) Jeon J, Schoen RE, Hoffmeister M, Newcomb PA, Berndt SI, et al. Determining Risk of Colorectal Cancer and Starting Age of Screening Based on Lifestyle, Environmental, and Genetic Factors. Gastroenterology. 2018 2164.e19.doi.org/10.1053/j.gastro.2018.02.021 Jun; 154(8): 2152
(24) PDQ Cancer Genetics Editorial Board. (2002, February 2). Genetics of Colorectal Cancer (PDQ®): Health Professional Version. In: PDQ Cancer Information Summaries [Internet]. Bethesda (MD): National Cancer Institute (US). PMID: 26389505. Bookshelf ID: NBK126744.
(25) Johns LE, Houlston RS: A systematic review and meta-analysis of familial colorectal cancer risk. Am J Gastroenterol 96 (10): 2992-3003, 2001.DOI: 10.1111/j.1572 0241.2001.04677.x
(26) Cottet V, Pariente A, Nalet B, et al.: Colonoscopic screening of first-degree relatives of patients with large adenomas: increased risk of colorectal tumors. Gastroenterology. 2007; 133(4): 1086-92.doi.org/10.1053/j.gastro.2007.07.023
(27) Win AK, Macinnis RJ, Hopper JL, et al.: Risk prediction models for colorectal cancer: a review. Cancer Epidemiol Biomarkers Prev. 2012; 21(3): 398-410. doi.org/10.1158/1055-9965.EPI-11-0771
(28) Sieber OM, Lamlum H, Crabtree MD, et al.: Whole-gene APC deletions cause classical familial adenomatous polyposis, but not attenuated polyposis or "multiple" colorectal adenomas. Proc Natl Acad Sci. 2002;99(5): 2954-8.doi.org/10.1073/pnas.042699199
(29) Hampel H, Bennett RL, Buchanan A, et al.: A practice guideline from the American College of Medical Genetics and Genomics and the National Society of Genetic Counselors: referral indications for cancer predisposition assessment. Genet Med. 2015; 17(1): 70-87. doi.org/10.1038/gim.2014.147
(30) JEG, IJ, et al., Detection rate of serrated polyps and serrated polyposis syndrome in colorectal cancer screening cohorts: a European overview. Gut, 2017. 66(7): p. 1225-1232. doi.org/10.1136/gutjnl-2015-310784
(31) O'Brien, M.J., Q. Zhao, and S. Yang, Colorectal serrated pathway cancers and precursors. Histopathology, 2015. 66(1): 49-65.doi.org/10.1111/his.12564
(32) Pai, R.K., et al., An update on the morphology and molecular pathology of serrated colorectal polyps and associated carcinomas. Mod Pathol, 2019; 32(10): 1390-1415. doi.org/10.1038/s41379-019-0280-2
(33) Egoavil, C., et al., Increased Risk of Colorectal Cancer in Patients With Multiple Serrated Polyps and Their First-Degree Relatives. Gastroenterology. 2017. 153(1): 106-112 e2 doi.org/10.1053/j.gastro.2017.04.003
(34) Okamoto, K., et al., Clinicopathological characteristics of serrated polyps as precursors to colorectal cancer: Current status and management. J Gastroenterol Hepatol. 2017; 32(2): 358 367. doi.org/10.1111/jgh.13482
(35) Yang Wu, A.M., and Alina Stoita, Clinical predictors for sessile serrated polyposis syndrome- A case control study. World J Gastrointest Endosc. 2017 Sep 16; 9(9): 464–470. doi: 10.4253/wjge.v9.i9.464doi: 10.4253/wjge.v9.i9.464
(36) Leonardo Zorron, C.T.P., Khizar Rana, and Rajvinder Singh, Different factors are associated with conventional adenoma and serrated colorectal neoplasia. Nagoya J Med Sci. 2020 May; 82(2): 335–343. doi: 10.18999/nagjms.82.2.335
(37) Aslam R Syed, P.T., and Shyam Thakkar, Old vs new- Risk factors predicting early onset colorectal cancer. World journal of gastrointestinal oncology. doi: 10.4251/wjgo.v11.i11.1011
(38) JEG, IJ, et al., Detection rate of serrated polyps and serrated polyposis syndrome in colorectal cancer screening cohorts: a European overview. Gut. 2017; 66(7): 1225-1232. doi.org/10.1136/gutjnl-2015-310784
(39) Snover DC, Ahnen DJ, Burt RW, et al. Serrated polyps of the colon and rectum and serrated polyposis. In: WHO Classification of Tumours of the Digestive System. Bosman FT, Carneiro F, Hruban RH, Theise ND, eds. Lyon: IARC Press. 2010; 160-5.ID: 20001204973. Data source type: CiNii articles. Retrieved from https://ci.nii.ac.jp/naid/1570009749402645760
(40) Snover DC, Jass JR, Fenoglio-Preiser C, et al. Serrated polyps of the large intestine: a morphologic and molecular review of an evolving concept. Am J Clin Pathol. 2005;124:380-91. doi.org/10.1309/v2ep-tplj-rb3f-ghjl
(41) Sacco M, De Palma FDE, Guadagno E, Giglio MC, Peltrini R, Marra E, et al. Serrated lesions of the colon and rectum: Emergent epidemiological data and molecular pathways. Open Med (Wars). 2020; 15(1): 1087–1095. doi: 10.1515/med-2020-0226