CENTRAL ASIAN JOURNAL OF MEDICAL AND NATURAL SCIENCES

https://cajmns.casjournal.org/index.php/CAJMNS
Volume: 07 Issue: 01 | January 2026 ISSN: 2660-4159

Article

Detection of Relationship of Histopathological and Clinical Features of Colon Cancer in patient's

Sabreen Saleem Abd Alkreem Alsaqi[1]

1. Department of Anatomy, Collage of Medicine, University of Babylon, Iraq
* Correspondence: med.sabreen.saleem@uobabylon.edu.iq,

Abstract: Colon cancer, also referred to as colorectal carcinoma when associated with rectal cancer, A heightened rate of colonic epithelial proliferation and an expansion of the cryptal proliferative zone are likely indicators of enhanced susceptibility to colonic cancer. Colonic cancer is not a singular disease; instead, it comprises a heterogeneous array of diseases characterized by distinct genetic and epigenetic alterations. The aim of this study was to identify correlations between histopathological examination and clinical symptoms**.** A total of 50 tissue blocks embedded in paraffin wax were utilized. A total of 50 patients with colon cancer (17 females and 33 males) underwent histopathological examination using a staining technique involving hematoxylin and eosin. Our study found that the average age of the patients was 52.2 years, with a male-to-female ratio of 2.13:1. The study shows no correlation between age (male and female) and the stage and grade of the tumor. Improvements in molecular biology, targeted therapy, and immunotherapy have changed how we manage thing

Keywords: Colon cancer, histopathological, and risk factors.

1. Introduction

Colon cancer is among the most prevalent cancers globally., as well as one of the leading causes of death caused by cancer. Colorectal cancer is the third most frequently diagnosed cancer in both men and women worldwide [1]. Australia and New Zealand, Europe and North America and Africa and South-Central Asia have the highest and the lowest demonstrate rates of CRC respectively. Carcinoma of colon (also known as colorectal carcinoma) attacks the inner layer of the colon, but other forms of cancer may attack the colon, such as sarcoma, melanoma and lymphoma [2].

Colon cancer is an ailment in which the proliferation of colon epithelial cells is unregulated. The multistep carcinogenesis idea posits that colon cells undergo a sequence of molecular changes, finally transforming into completely malignant cells [3].

It is usually caused by a multistep process, which is Adenoma-carcinoma sequence that involves genetic mutations in essential oncogenes and tumor suppressor genes such as KRAS, p53and APC, Risk factors include age above 50, high intakes of red and processed meats, obesity, physical inactivity, tobacco use, alcohol consumption, and chronic inflammatory bowel disease [4].

The majority of CRC is sporadic and only a quarter of the patients possess a familial predisposition to the disease, showing that both genetic and environmental factors are essential. Environmental factors that raise the rates of this cancer could possibly be the indicators of risks [5].

Citation: Alsaqi, S. S. A. A. Detection of Relationship of Histopathological and Clinical features of colon cancer in patient's. Central Asian Journal of Medical and Natural Science 2026, 7(1), 248-253.

Received: 10th Aug 2025
Revised: 16th Sep 2025
Accepted: 24th Oct 2025
Published: 30th Nov 2025

The store of faeces in a short term and the movement of faeces are the main role of the colon. The colon absorbs about 1 litre of water daily so as to thicken the stools.

Besides that, it can absorb sodium, potassium and chloride and releases potassium to the lumen itself. It executes some important roles like digestion of indigestible food materials such as cellulose, synthesis of intestinal peristalsis ,vitamin K stimulation and strengthening the immune system by MALT [6].

Colorectal tumours present an extensive a spectrum of atypical tissue proliferations (malignant tumors) situated between benign tumors and invasive cancer, primarily consisting of tumors originating from epithelial cells (specifically, adenoma or adenocarcinoma) . Pathologists point out three types of lesions, namely non-neoplastic polyps (benign soft tissue tumours), neoplastic polyps (adenomatous polyps, adenomas), and cancers [7].

2. Methodology

The work described was executed in laboratories at Hila Teaching Hospital in Babylon, Iraq, from November 2024 to April 2025. We got 50 samples from 50 patients (33 men and 17 women) who were experiencing. These samples were 50 tissue blocks embedded in paraffin wax. We also got pathologic and clinical information about colon cancer, such as the type of surgery, the person's sex, age, family history, smoking, and diabetes.

A histopathological examination study: We got sections that were 5 μm thick from paraffin-embedded tissues. We stained them with hematoxylin and eosin using a staining technique ,The tissue was preserved in 10% buffered formalin, routinely processed, and embedded in paraffin. The sections have been stained with eosin and hematoxylin 11.

Statistical Analysis

Statistical Package for the Social Sciences Version23 was expressed in the mean and standard deviation form (mean ± standard deviation andChi-square test, correlation and using odds ratio with $P < 0.05$.

3. Results

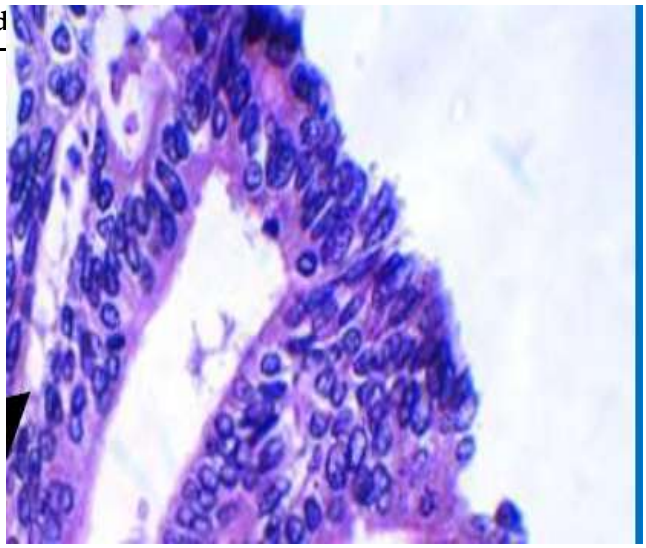
The results we got When we grouped patients by sex and age, we found that most of the men (24%) were between the ages of 51 and 60. Table 1 shows that the female patients also had a higher frequency in the age range of 51 to 60 years (14%). Our findings indicate that the patient cohort, comprising 17 males and 13 females, had a mean age of 52.2 ± 16.1 years and a median age of 54 years (range: 23–70 years) [8]. The male-to-female ratio was 2:1, and the distribution of patients based on tumor grade was as follows: Twenty-six patients (52%) exhibited Grade I, sixteen patients (32%) demonstrated Grade II, and eight patients (16%) presented Grade III show in Figures 1, 2, 3,4). The patients were distributed as follows based on the stage of colon cancer: Ten patients (20%) had Stage I disease, thirty-two patients (64%) had Stage II disease, and eight patients (16%) had Stage III disease (Table 2 and Additionally, 30% of patients were smokers, 35% had a family history, and 32% had diabetes.

Table 1. The distribution of patients according to Age

Age	Male	N(%)	female	N(%)
10-20	0	(0%)	0	(0%)
21-30	5	(10%)	1	(2%)
31-40	1	(2%)	2	(4%)
41-50	7	(14%)	4	(8%)
51-60	12	(24%)	7	(14%)
61-70	4	(8%)	2	(4%)
71-80	3	(6%)	1	(2%)
total	**33**	**100%**	**17**	**100%**

Table 2. The distribution of patients according Clinical feature

Colon carcinoma	Mean - SD
50	52.2-16.1
Male	33 (66%)
female	17 (34%)
grade	N(%)
grade I	26 (52%)
gradeII	16 (32%)
grade III	8 (16%)
stag	N(%)
Stag1	10 (20%)
Stag2	32 (64%)
Stag3	8 (16%)
smoking	30 (60%)
Family history	35 (70%)
d	



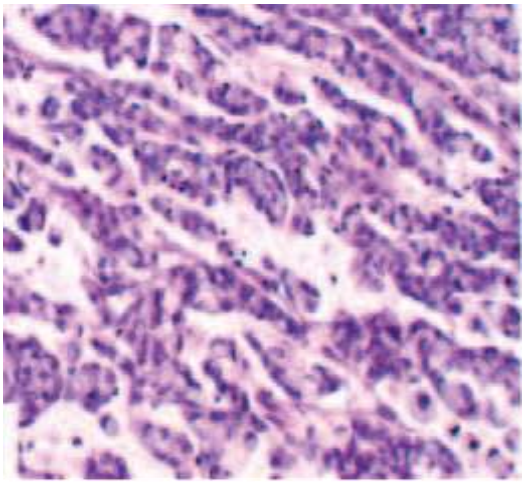


Figure 1. Histological section of colon cancer grade I

Figure 2. Histological section of colon cancer grade2

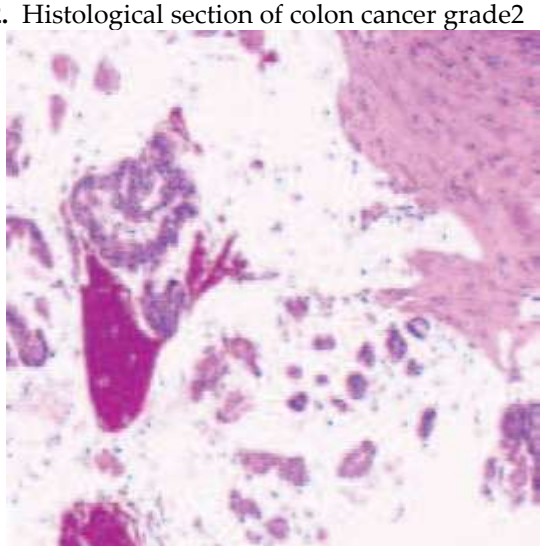


Figure 3. Histological section of colon extracellular mucin and free-floating carcinoma cells

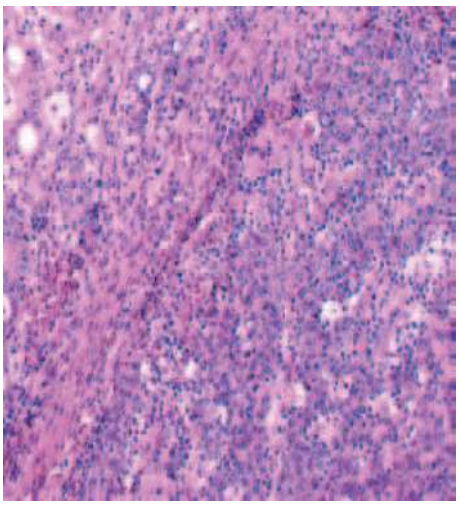


Figure 4. Histological section of colon cancer grade3

4. Discussion

The present study reveals that the average age of patients with colon cancer is 52.2 ± 16.1 years, with a median age of 53 years and an age range of 21 to 75 years. In contrast, the majority of patients with colorectal cancer were over 40 years old, accounting for 75%, whilst 25% of cases were under 40 years old [9]. The genesis of cancer is primarily attributed to two main causes: genetic germline mutations in genes that govern the cell cycle and proliferation, and environmental influences [10].

It can be inferred that mutations in genes causing malignancy in colonic mucosa are attributable to Environmental variables, particularly the interplay between digestive wastes and microorganisms in the colonic mucosa [11]. It takes a long time for many genes to change because of chemical carcinogens on the colonic mucosa before frank malignancy shows up. This is why most of the patients are older [12].

The male-to-female ratio in this study was 2.1:1. that women have much higher levels of estrogenic hormones than men do, Hartman and Gustafsson , Estrogens are essential in protecting against the onset and advancement of colorectal cancer, possibly mediated by

estrogen receptor β (ERβ). The current study indicated that the majority of participants (64%) had stage II disease [13]. early-stage cancers (carcinoma in situ and stage I) are frequently undetected because to the inadequacy of effective screening programs, such as colonoscopy and imaging techniques, for high-risk populations. It is necessary to use screening programs in Iraq to find these common tumors early on because Treating patients with advanced-stage malignancies is complex [14], [15].

The current study demonstrated that the majority of colorectal carcinoma (52%) exhibited grade I histological pattern Grade well differentiated The histological pattern is distinguished by a glandular growth pattern featuring, Multilayering, loss of polarity, back-to-back configuration, and aberrant nuclear characteristics, including hyperchromatism, an elevated nuclear-to-cytoplasmic (N/C) ratio, and discernible nucleoli. despite their shared risk factors, including obesity and a sedentary lifestyle [16], [17]. Some research indicates that the association might be more pronounced in men compared to women [18], [19].

The correlation seems to be more pronounced in rectal cancer compared to colon cancer and specific molecular subtypes of both colon and rectal cancer [20]. Early studies concur with our findings, suggesting they may have overlooked this association due to the notably prolonged latency period between tobacco exposure and colon cancer diagnosis, spanning at least three to four decades [21].

5. Conclusion

In conclusion, the number of benign colonic polyps being surgically removed nationwide is declining. These results hold true for all age and sex groups and are in line with an increasing amount of research that supports endoscopic resection as a safer, more effective, and less morbid alternative management approach. However, there was a higher rate of colectomy among Black patients, indicating that there are still inequalities in access to and use of minimally invasive endoscopic proced

REFERENCES

[1] R. L. Siegel, K. D. Miller, H. E. Fuchs, and A. Jemal, "Cancer statistics, 2025," CA Cancer J. Clin., vol. 75, no. 1, pp. 5–29, 2025.

[2] World Health Organization, Colorectal Cancer Fact Sheet. Geneva, Switzerland: WHO, 2024.

[3] National Cancer Institute, "Colon and Rectal Cancer—Patient Version," 2024. Accessed: Nov. 2025. [Online]. Available: https://www.cancer.gov/types/colorectal

[4] American Cancer Society, Colorectal Cancer Statistics, 2024. [Online]. Available: https://www.cancer.org/

[5] E. Dekker, P. J. Tanis, J. L. A. Vleugels, P. M. Kasi, and M. B. Wallace, "Colorectal cancer," Lancet, vol. 394, no. 10207, pp. 1467–1480, 2019.

[6] J. Guinney et al., "The consensus molecular subtypes of colorectal cancer," Nat. Med., vol. 21, no. 11, pp. 1350–1356, 2015.

[7] M. J. Overman et al., "Nivolumab in patients with metastatic DNA mismatch repair-deficient colorectal cancer," J. Clin. Oncol., vol. 42, no. 3, pp. 210–220, 2024.

[8] R. Dienstmann, L. Vermeulen, J. Guinney, S. Kopetz, S. Tejpar, and J. Tabernero, "Consensus molecular subtypes and the evolution of precision medicine in colorectal cancer," Nat. Rev. Cancer, vol. 17, no. 2, pp. 79–92, 2017.

[9] H. Brenner, M. Kloor, and C. P. Pox, "Colorectal cancer," Lancet, vol. 383, no. 9927, pp. 1490–1502, 2014.

[10] E. J. Kuipers et al., "Colorectal cancer," Nat. Rev. Dis. Primers, vol. 1, p. 15065, 2015.

[11] L. Falkeholm, C. A. Grant, A. Magnusson, and E. Möller, "Xylene-free method for histological preparation: a multicentre evaluation," Lab. Invest., vol. 81, no. 9, pp. 1213–1221, 2001.

[12] E. Van Cutsem et al., "ESMO consensus guidelines for the management of patients with metastatic colorectal cancer," Ann. Oncol., vol. 27, no. 8, pp. 1386–1422, 2016.

[13] P. M. Kasi and A. Grothey, "Chemotherapy in advanced colorectal cancer: A review," JAMA Oncol., vol. 4, no. 6, pp. 847–856, 2018.

[14] J. Hartman and J.-Å. Gustafsson, "Estrogen receptors in colorectal cancer: goalkeepers, strikers, or bystanders?," Cancer Prev. Res., vol. 3, no. 8, pp. 897–899, 2010.

[15] M. Arnold, M. S. Sierra, M. Laversanne et al., "Global patterns and trends in colorectal cancer incidence and mortality," Gut, vol. 66, no. 4, pp. 683–691, 2017.
[16] B. Vogelstein, E. R. Fearon, S. R. Hamilton et al., "Genetic alterations during colorectal-tumor development," N. Engl. J. Med., vol. 319, no. 9, pp. 525–532, 1988.
[17] E. R. Fearon and B. Vogelstein, "A genetic model for colorectal tumorigenesis," Cell, vol. 61, no. 5, pp. 759–767, 1990.
[18] C. J. Rees and R. Bevan, "The future of colorectal cancer screening: Innovations and challenges," Nat. Rev. Gastroenterol. Hepatol., vol. 18, no. 10, pp. 605–617, 2021.
[19] S. C. Larsson, E. Giovannucci, and A. Wolk, "Diabetes and colorectal cancer incidence in the cohort of Swedish men," Diabetes Care, vol. 28, no. 7, pp. 1805–1807, 2005.
[20] W. Luo, Y. Cao, C. Liao, and F. Gao, "Diabetes mellitus and the incidence and mortality of colorectal cancer: a meta-analysis of 24 cohort studies," Colorectal Dis., vol. 14, no. 11, pp. 1307–1312, 2012.
[21] P. S. Liang, T. Y. Chen, and E. Giovannucci, "Cigarette smoking and colorectal cancer incidence and mortality: Systematic review and meta-analysis," Int. J. Cancer, vol. 124, no. 10, pp. 2406–2415, 2009.