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Immunohistochemical Expression of E-Cadherin and Vimentin in Colorectal Carcinoma: A Cross-Sectional Study of Epithelial–Mesenchymal Transition-Related Alterations.

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Abstract

Background:
Colorectal carcinoma (CRC) is a major global health burden and a leading cause of cancer-related mortality worldwide. Tumor progression, invasion, and metastasis are closely associated with epithelial–mesenchymal transition (EMT), a biological process characterized by loss of epithelial markers and acquisition of mesenchymal features. E-cadherin and vimentin are key immunohistochemical markers representing epithelial and mesenchymal phenotypes, respectively. This study aimed to evaluate the expression of these markers in colorectal carcinoma and to examine their distribution across clinicopathological parameters.

Methodology:
This cross-sectional observational study included 72 histopathologically confirmed cases of colorectal carcinoma. Formalin-fixed paraffin-embedded tissue sections were subjected to immunohistochemical analysis for E-cadherin and vimentin using standardized protocols. Clinicopathological variables including age, sex, tumor size, grade, depth of invasion, lymph node involvement, lymphovascular space invasion, and pathological stage (AJCC 8th edition) were recorded. Data were analyzed using descriptive statistics, with categorical variables expressed as frequencies and percentages. No inferential statistical testing was performed.

Results:
E-cadherin expression was retained in the majority of cases, with higher positivity observed in smaller tumors, early depth of invasion (T1–T2), and well-differentiated histology. Reduced expression was more frequently observed in tumors with higher grade, greater depth of invasion (T3–T4), advanced pathological stage (AJCC Stage III–IV), lymph node involvement, and lymphovascular space invasion. Vimentin expression was observed in a subset of cases and showed higher frequency in tumors with larger size, deeper invasion, higher grade, advanced stage, nodal involvement, and lymphovascular space invasion. These findings demonstrate distribution patterns of EMT-related immunophenotypic alterations across adverse clinicopathological features.

Conclusion:
Altered expression of E-cadherin and vimentin is observed across clinicopathological characteristics associated with aggressive tumor behavior in colorectal carcinoma. However, due to the cross-sectional design and absence of survival data, no prognostic significance can be established. Further longitudinal studies are required to determine the clinical and prognostic relevance of these markers.

Keywords:
Colorectal Carcinoma, Epithelial–Mesenchymal Transition, E-Cadherin, Vimentin, Immunohistochemistry, Clinicopathological Parameters

Introduction

Colorectal carcinoma (CRC) is a major global health concern and remains one of the leading causes of cancer-related morbidity and mortality worldwide. According to global cancer statistics, colorectal cancer ranked as the third most commonly diagnosed malignancy and the second leading cause of cancer-related deaths in 2020 [1]. The burden of disease continues to rise in both developed and developing countries, with significant variation in incidence and distribution across populations. In India, colorectal cancer ranks among the top malignancies, being the fourth most common cancer in males and the fifth in females [2].

Despite advances in diagnosis and treatment, tumor recurrence, metastasis, and resistance to therapy remain significant challenges contributing to poor clinical outcomes. Increasing attention has been directed toward understanding the molecular mechanisms underlying tumor progression, particularly those involved in invasion and metastasis. Among these, epithelial–mesenchymal transition (EMT) has emerged as a critical biological process in cancer progression. EMT is characterized by the transformation of epithelial cells into mesenchymal-like cells, resulting in loss of cell polarity and intercellular adhesion, along with acquisition of migratory and invasive properties [3].

At the molecular level, EMT involves downregulation of epithelial markers and upregulation of mesenchymal markers. E-cadherin, a key transmembrane glycoprotein responsible for cell-to-cell adhesion in epithelial tissues, plays a central role in maintaining tissue architecture. Loss or reduction of E-cadherin expression is considered a hallmark of EMT and is associated with increased tumor invasiveness [3]. Conversely, vimentin, an intermediate filament protein, is a well-established mesenchymal marker whose increased expression is linked to enhanced tumor progression, invasion, and metastatic potential [4].

The interplay between loss of epithelial characteristics and gain of mesenchymal features contributes to tumor aggressiveness and dissemination. Numerous studies have reported altered expression of E-cadherin and vimentin in colorectal carcinoma, suggesting their involvement in EMT-related tumor behavior. However, variability exists in reported findings, particularly in relation to clinicopathological parameters such as tumor grade, stage, and lymph node involvement.

In this context, the present study was undertaken to evaluate the immunohistochemical expression of E-cadherin and vimentin in colorectal carcinoma and to analyze their distribution across clinicopathological parameters. The study specifically aims to characterize EMT-related immunophenotypic alterations within the tumor microenvironment. Given the cross-sectional design and absence of survival follow-up, the study does not attempt to establish prognostic significance but rather provides a descriptive assessment of marker expression patterns in relation to tumor characteristics.

Methodology

Study Design

This study was designed as a cross-sectional observational analysis aimed at evaluating the immunohistochemical expression of epithelial–mesenchymal transition (EMT)-related markers in colorectal carcinoma. The study focused on descriptive characterization of immunophenotypic patterns without establishing causal or prognostic relationships.

Ethics

Ethical approval was obtained from the institutional ethics committee prior to commencement, and all procedures were conducted in accordance with standard ethical guidelines for biomedical research involving human tissue samples. Patient confidentiality and data anonymity were strictly maintained throughout the study period.

Setting

The study was conducted in the Department of Pathology at R.G. Kar Medical College and Hospital, Kolkata, over a period of eighteen months from February 2021 to August 2022. Histopathological specimens were obtained from patients undergoing surgical resection in the Department of General Surgery within the same institution, ensuring consistency in clinical and laboratory workflow.

Participants

The study included patients with histopathologically confirmed primary colorectal carcinoma who underwent surgical resection. Inclusion criteria comprised newly diagnosed cases of colorectal carcinoma, including patients who had received neoadjuvant therapy prior to surgery. Exclusion criteria included recurrent colorectal carcinoma, non-adenocarcinoma lesions, and metastatic tumors involving the colon or rectum with a known primary elsewhere. A total of 72 resected specimens fulfilling eligibility criteria were included. The inclusion of patients receiving neoadjuvant therapy was acknowledged as a potential confounding factor; however, subgroup stratification was not feasible due to limited sample size.

Variables

The primary variables assessed included immunohistochemical expression of E-cadherin (epithelial marker) and vimentin (mesenchymal marker). Secondary variables included clinicopathological parameters such as age, sex, tumor size, anatomical site, histological grade, depth of invasion (pT stage), lymph node involvement, lymphovascular space invasion (LVSI), and pathological stage based on the American Joint Committee on Cancer (AJCC) 8th edition classification. These variables were selected to evaluate the distribution of EMT-related markers across different pathological characteristics.

Data Sources / Measurement

Data were obtained from histopathology records and examination of formalin-fixed paraffin-embedded (FFPE) tissue sections. Tissue sections of 3–4 µm thickness were prepared using a microtome and mounted on poly-L-lysine-coated slides. Standard immunohistochemistry protocols were followed, including deparaffinization, rehydration, and heat-induced epitope retrieval using Tris buffer (pH 9.0). Endogenous peroxidase activity was blocked prior to antibody application. Primary antibodies used were E-cadherin (rabbit monoclonal, Clone EP6; Thermo Fisher Scientific, USA) and vimentin (rabbit monoclonal, Clone EP21; Thermo Fisher Scientific, USA), both applied at a dilution of 1:100 and incubated at 37°C for 60 minutes.

Detection was carried out using a horseradish peroxidase (HRP)-based system with diaminobenzidine (DAB) chromogen, followed by hematoxylin counterstaining. Positive controls (normal colorectal mucosa for E-cadherin and tonsillar tissue for vimentin) and negative controls (omission of primary antibody) were included in each run. All procedures were conducted under standardized laboratory conditions to ensure consistency and reproducibility.

Bias

Potential sources of bias included selection bias due to single-center recruitment and inclusion of only surgically resected cases, which may not represent early or inoperable disease. Measurement bias was minimized through standardized immunohistochemical protocols and use of control tissues. However, absence of external quality assurance may limit generalizability. Confounding due to neoadjuvant therapy was acknowledged, as its potential influence on marker expression could not be separately analyzed due to sample size limitations.

Study Size

The study sample consisted of 72 cases of colorectal carcinoma collected over the defined study period. The sample size was determined based on the availability of eligible cases during the study duration rather than formal statistical calculation, consistent with the descriptive nature of the study.

Statistical Methods

Data were entered into Microsoft Excel and analyzed using SPSS version 27.0 (IBM Corp., Chicago, IL, USA). As the study was designed as a descriptive cross-sectional analysis, only descriptive statistics were applied. Categorical variables were summarized using frequencies and percentages. No inferential statistical testing, correlation analysis, or hypothesis testing was performed, and results were interpreted strictly in terms of distribution patterns without implying statistical significance or causal relationships.

Results

Participants

A total of 72 patients with histopathologically confirmed colorectal carcinoma were included in this cross-sectional study. All cases represented surgically resected specimens meeting the predefined inclusion and exclusion criteria. The cohort comprised both male and female patients across a wide age range, with all samples subjected to uniform histopathological and immunohistochemical evaluation. No cases were excluded after initial selection, and complete clinicopathological and immunohistochemical data were available for all included participants.

Descriptive data

The demographic and clinicopathological characteristics of the study population are summarized in Tables 1–8. The study cohort showed a male predominance, with 59.7% males (n = 43) and 40.3% females (n = 29), as presented in Table 1. Age distribution revealed that the majority of patients were diagnosed after the fourth decade of life, with the highest frequency observed in the 51–60-year age group (29.2%), followed by 61–70 years (20.8%) (Table 2).

Regarding tumor location, the ascending colon was the most commonly involved site (36.1%), followed by the sigmoid colon (19.4%) and rectum (15.3%), while other anatomical sites such as caecum, descending colon, and transverse colon were less frequently involved (Table 3). Tumor size distribution showed that more than half of the lesions (54.16%) measured between 5–10 cm, whereas 41.67% were <5 cm and only 4.17% exceeded 10 cm (Table 4).

Histopathological grading demonstrated that the majority of tumors were moderately differentiated (Grade 2, 58.3%), followed by well-differentiated (Grade 1, 27.8%) and poorly differentiated tumors (Grade 3, 13.9%) (Table 5). Depth of invasion analysis indicated that most tumors were classified as pT3 (58.3%), followed by pT2 (23.6%), pT4 (12.5%), and pT1 (5.6%) (Table 6). Lymphovascular space invasion (LVSI) was absent in the majority of cases (81.9%), while 18.1% showed LVSI positivity (Table 7). Similarly, nodal involvement was absent in most patients (N0, 68.1%), whereas varying degrees of nodal metastasis (N1 and N2 categories) were observed in a subset of cases (Table 8). These findings collectively describe the distribution of clinicopathological characteristics within the study population.

Outcome data

The immunohistochemical expression patterns of E-cadherin and vimentin across clinicopathological parameters are presented in Tables 9 and 10, with representative photomicrographs shown in Figure 1 and overall expression patterns illustrated in Figure 2. E-cadherin expression was retained in the majority of tumors, with higher positivity observed in smaller tumors (<5 cm), early depth of invasion (T1–T2), and well-differentiated histology (Grade 1), as detailed in Table 9.

In contrast, reduced or absent E-cadherin expression was more frequently observed in tumors with greater depth of invasion (T3–T4), higher histological grade (Grade 3), higher pathological stage (AJCC Stage III–IV), presence of lymph node involvement, and lymphovascular space invasion (Table 9).

Vimentin expression was observed in a subset of cases and demonstrated a distinct distribution pattern across clinicopathological features (Table 10). Lower expression was noted in smaller tumors, early-stage disease, and well-differentiated tumors, whereas higher expression was more frequently observed in tumors with larger size, deeper invasion (particularly T4), higher grade, and advanced pathological stage (AJCC Stage III–IV). Additionally, vimentin positivity was more frequently observed in cases with nodal involvement and LVSI (Table 10). The distribution of these immunohistochemical markers across tumor characteristics is visually supported by Figure 2.

Main results

The overall findings indicate that altered expression patterns of E-cadherin and vimentin are distributed across adverse clinicopathological features in colorectal carcinoma. Specifically, reduced E-cadherin expression and increased vimentin expression were more frequently observed in tumors with higher grade, greater depth of invasion, advanced pathological stage (AJCC Stage III–IV), lymph node involvement, and lymphovascular space invasion, as demonstrated in Tables 9 and 10 and illustrated in Figures 1 and 2. These observations reflect the presence of epithelial–mesenchymal transition (EMT)-related immunophenotypic alterations within the study cohort.

As this study was designed as a descriptive cross-sectional analysis, the results are presented in terms of distribution patterns without inference of statistical significance or causality. Furthermore, no subgroup analysis was performed based on neoadjuvant therapy status; therefore, its potential influence on marker expression could not be independently evaluated within this dataset.

Table 1: Baseline and Clinicopathological Characteristics of the Study Population (n = 72).

Variables		n (%)
Gender	Male	43 (59.7)
	Female	29 (40.3)
Age (years)	< 30	5 (6.9)
	31–40	13 (18.1)
	41–50	13 (18.1)
	51–60	21 (29.2)
	61–70	15 (20.8)
	71–80	5 (6.9)
Tumour Site	Ascending colon	26 (36.1)
	Descending colon	6 (8.3)
	Sigmoid colon	14 (19.4)
	Caecum	7 (9.7)
	Transverse colon	4 (5.6)
	Rectum	11 (15.3)
	Caecum + Ascending colon	4 (5.6)
Tumour Size (cm)	< 5	30 (41.7)
	5–10	39 (54.2)
	> 10	3 (4.2)
Tumour Grade	Grade 1	20 (27.8)
	Grade 2	42 (58.3)
	Grade 3	10 (13.9)
Depth of Invasion (pT)	T1	4 (5.6)
	T2	17 (23.6)
	T3	42 (58.3)
	T4	9 (12.5)
Lymphovascular Space Invasion (LVSI)	Negative	59 (81.9)
	Positive	13 (18.1)
Lymph Node Status	N0	49 (68.1)
	N1a	10 (13.9)
	N1b	6 (8.3)
	N2a	3 (4.2)
	N2b	4 (5.5)

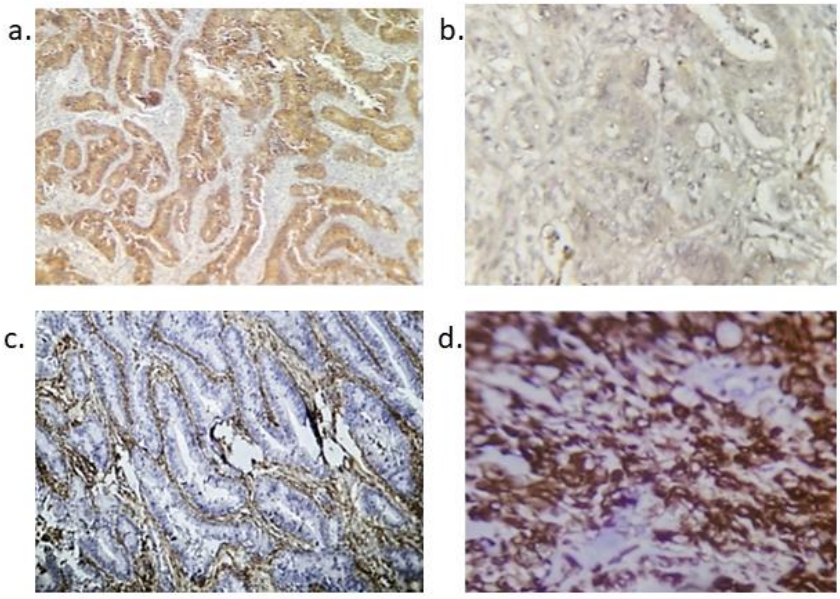

Figure 1: Representative photomicrographs of colorectal carcinoma. (A) H&E-stained section showing moderately differentiated adenocarcinoma (400×). (B) Immunohistochemical staining showing preserved membranous E-cadherin expression (400×). (C) Loss of membranous E-cadherin expression (400×). (D) Cytoplasmic positivity for vimentin in tumor cells (400×).

Table 2: Association of E-cadherin and Vimentin Expression with Clinicopathological Parameters (n = 72).

Variables		E-cadherin		p-value	Vimentin		p-value
		Positive n (%)	Negative n (%)		Positive n (%)	Negative n (%)	
Age	≤ 51	25 (75.8)	8 (24.2)	0.364	12 (36.4)	21 (63.6)	0.338
	> 51	33 (84.6)	6 (15.4)		10 (25.6)	29 (74.4)	
Sex	Male	34 (79.1)	9 (20.9)	0.708	15 (34.9)	28 (65.1)	0.332
	Female	24 (82.8)	5 (17.2)		7 (24.1)	22 (75.9)	
Tumour size (cm)	< 5	27 (90.0)	3 (10.0)		4 (13.3)	26 (86.7)	
	5–10	29 (74.4)	10 (25.6)	0.214	17 (43.6)	22 (56.4)	0.028
	> 10	2 (66.7)	1 (33.3)		1 (33.3)	2 (66.7)	
Depth of invasion	T1	4 (100.0)	0 (0.0)		0 (0.0)	4 (100.0)	
	T2	16 (94.1)	1 (5.9)	0.001*	2 (11.8)	15 (88.2)	<0.001*
	T3	35 (83.3)	7 (16.7)		13 (31.0)	29 (69.0)	
	T4	3 (33.3)	6 (66.7)		7 (77.8)	2 (22.2)	
Tumour grade	Grade 1	19 (95.0)	1 (5.0)	0.006*	2 (10.0)	18 (90.0)	0.011*
	Grade 2	34 (81.0)	8 (19.0)		15 (35.7)	27 (64.3)	
	Grade 3	5 (50.0)	5 (50.0)		5 (50.0)	5 (50.0)	
Tumour stage	Stage I	19 (95.0)	1 (5.0)	0.002*	2 (10.0)	18 (90.0)	0.004*
	Stage II	26 (89.7)	3 (10.3)		7 (24.1)	22 (75.9)	
	Stage III–IV	13 (56.5)	10 (43.5)		13 (56.5)	10 (43.5)	
Lymph node involvement	Positive	13 (56.5)	10 (43.5)	<0.001*	13 (56.5)	10 (43.5)	<0.001*
	Negative	45 (91.8)	4 (8.2)		9 (18.4)	40 (81.6)	
LVSI	Positive	11 (61.1)	7 (38.9)	0.018*	10 (55.6)	8 (44.4)	0.012*
	Negative	47 (87.0)	7 (13.0)		12 (28.6)	42 (71.4)	

Discussion

Epithelial–mesenchymal transition (EMT) is a fundamental biological process observed across various physiological and pathological conditions, including embryonic development, tissue remodeling, and tumor progression [3]. During EMT, epithelial cells lose their polarity and intercellular adhesion while acquiring mesenchymal characteristics that enhance migratory and invasive potential [5–8]. In the context of malignancy, this transition contributes to tumor aggressiveness, invasion, and metastasis, thereby playing a central role in cancer progression [9,10].

E-cadherin, a transmembrane glycoprotein encoded by the CDH1 gene, is essential for maintaining epithelial cell adhesion and tissue integrity. Loss or reduction of E-cadherin expression is widely recognized as a hallmark of EMT and is associated with increased tumor invasiveness and dedifferentiation [6]. In the present study, E-cadherin expression was observed in the majority of cases, while a subset demonstrated reduced or absent expression. These findings are consistent with previous studies, including those by Mitselou et al. [11] and Rajkumar SD et al. [12], which reported high rates of E-cadherin positivity in colorectal carcinoma.

In this study cohort, E-cadherin expression did not show a consistent variation with respect to age, sex, or tumor size, which is in agreement with observations reported by Rajkumar SD et al. [12] and Choi et al. [13]. However, reduced E-cadherin expression was more frequently observed in tumors with greater depth of invasion, higher histological grade, advanced pathological stage (AJCC Stage III–IV), lymph node involvement, and lymphovascular space invasion. Similar distribution patterns have been described in earlier studies by Choi et al. [13], Lugli et al. [14], Rashed et al. [15], Kwak et al. [16], Seo et al. [17], and Kim A Sun et al. [18], supporting the association of reduced epithelial marker expression with adverse clinicopathological features.

Vimentin, an intermediate filament protein, serves as a key marker of mesenchymal differentiation and is widely implicated in EMT-related tumor progression. Its increased expression has been associated with enhanced tumor invasiveness, metastatic potential, and resistance to therapy through activation of multiple signaling pathways [19,20]. Vimentin expression has been reported across a wide range of malignancies, including colorectal carcinoma, where it reflects mesenchymal transformation of tumor cells [21-28].

In the present study, vimentin expression was observed in a subset of cases, with variable expression rates comparable to those reported in the literature, including studies by Maghrabi et al. [29], Choi et al. [13], Rashed et al. [15], and Wang et al. [30]. This variability may be attributed to differences in study populations, tumor characteristics, antibody clones, and scoring criteria. Similar to previous reports, vimentin expression in this study did not demonstrate a consistent association with age or sex [13,29-31].

However, increased vimentin expression was more frequently observed in tumors with larger size, greater depth of invasion, higher histological grade, advanced pathological stage (AJCC Stage III–IV), lymph node involvement, and lymphovascular space invasion. These findings are consistent with studies by Toiyama et al. [31], Dai et al. [32], Niknami et al. [33], Wang et al. [30], and Choi et al. [13], which have reported similar distribution patterns of mesenchymal marker expression in relation to adverse tumor characteristics.

Taken together, the findings of this study demonstrate the distribution of EMT-related immunophenotypic alterations across clinicopathological parameters in colorectal carcinoma. The observed patterns of reduced E-cadherin expression and increased vimentin expression in tumors with adverse features reflect the biological process of EMT within the tumor microenvironment. However, as this study is cross-sectional in design, these observations represent descriptive associations rather than causal or prognostic relationships.

Limitations

This study has several limitations that should be considered when interpreting the findings. First, the sample size was relatively small and derived from a single institution, which may limit the generalizability of the results to broader populations. Second, the cross-sectional design precludes assessment of temporal relationships, disease progression, or survival outcomes, thereby limiting the ability to establish prognostic significance. Another important limitation is the inclusion of patients who received neoadjuvant therapy prior to surgical resection. Neoadjuvant treatment may influence immunohistochemical expression of E-cadherin and vimentin; however, due to limited sample size, subgroup analysis or adjustment for treatment status was not feasible. Additionally, external quality assurance for immunohistochemical procedures was not performed, which may affect reproducibility across different laboratory settings. Future studies incorporating larger, multicentric cohorts with longitudinal follow-up and standardized protocols are required to validate these findings and to determine the clinical and prognostic relevance of EMT-related markers in colorectal carcinoma.

Conclusion

The present study demonstrates that reduced E-cadherin expression and increased vimentin expression are distributed across clinicopathological features associated with aggressive tumor characteristics in colorectal carcinoma, including higher histological grade, greater depth of invasion, advanced pathological stage (AJCC Stage III–IV), lymph node involvement, and lymphovascular space invasion. These findings support the presence of epithelial–mesenchymal transition (EMT)-related immunophenotypic alterations within the tumor microenvironment.

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