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Original Article

Assessment of the immunohistochemical expression of E-cadherin in gastric carcinoma and its correlation with clinicopathological parameters

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ABSTRACT

Background: E-cadherin is a key calcium-dependent adhesion molecule involved in tissue integrity and cell differentiation. Epithelial-mesenchymal transition and cancer development in most malignancies, particularly gastric carcinoma, have been linked to loss of E-cadherin. This Study aimed to assess the immunohistochemical expression of E-cadherin in gastric carcinoma and its correlation with clinicopathological parameters. **Methods & Materials:** This cross-sectional study was performed at Shahabuddin Medical College, Dhaka, Bangladesh from October 2023 to October 2024 among 66 histologically verified cases of gastric carcinoma. Formalin-fixed, paraffin-embedded gastrectomy and endoscopic biopsy specimens were examined. Immunohistochemical staining for E-cadherin was carried out with a monoclonal antibody following heat-induced antigen retrieval. E-cadherin expression was semiquantitatively scored and divided into aberrant (0–2+) or preserved (3+). Statistical analysis was performed in SPSS version 26, including the Chi-square test, Welch's t-test, Spearman's correlation, and multivariable logistic regression. **Results:** Aberrant E-cadherin expression was observed in 87.9% of patients and was maintained in 12.1% of patients. Tumour grade and E-cadherin expression were strongly inversely correlated ($\rho = -0.97$, $p < 0.001$), and well-differentiated tumours showed increased expression. The diffuse Lauren type showed significantly higher frequencies of aberrant expression (94.7%) compared to the intestinal type (78.6%, $p = 0.048$). Multivariable logistic regression identified histopathological grade as an independent predictor of aberrant E-cadherin expression (OR = 7.81, $p < 0.001$). **Conclusion:** Down-regulation of E-cadherin is significantly correlated with poor tumour differentiation and diffuse histological type in gastric carcinoma, suggesting its potential use as a prognostic marker and therapeutic target.

Keywords: *E-cadherin, Gastric Carcinoma, Immunohistochemistry, Biomarker*

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INTRODUCTION

Stomach cancer remains one of the leading causes of cancer-related deaths among the entire population, with over a million new cases diagnosed annually and roughly 783,000 deaths worldwide [1]. Despite advances in diagnosis and therapy, the overall five-year survival rate remains unfavorable at around 20–30% due mainly to late diagnosis and extensively advanced disease at presentation [2]. The molecular heterogeneity of gastric cancer, for the purpose of histology, molecular characteristics, and clinical behaviour, demands extensive prognostic stratification to guide individualised treatment modalities and improve patient outcomes. Cell adhesion molecules, and cadherins in particular, are critical in maintaining tissue architecture, cell differentiation, and the prevention of tumor formation. E-cadherin is a calcium-dependent transmembrane glycoprotein of epithelial origin encoded at 16q22.1 and is a critical cell-cell adhesion mediator as well as an epithelial phenotype marker [3]. Loss of E-cadherin expression has been extensively documented in several carcinomas and is strongly correlated with epithelial-mesenchymal transition (EMT), a disease process where epithelial cells are depolarised and develop migratory and invasive capabilities [4]. The Lauren classification system, dividing gastric adenocarcinomas into intestinal and diffuse types based on morphologic characteristics, provides useful prognostic information on tumour behaviour and treatment response [5]. Intestinal-type gastric cancer is composed of well-differentiated glandular structures of varying grade, whereas diffuse-type tumors are composed of poorly cohesive cells without glandular differentiation but may have signet-ring cell variants [6]. There is growing evidence that improperly expressed E-cadherin is particularly frequent in diffuse-type gastric carcinomas and with increased metastatic ability and poor prognosis [7]. A few previous reports have demonstrated that E-cadherin downregulation is associated with aggressive tumour behaviour, advanced stage of disease, lymph node metastasis, and decreased survival [8]. Further, the E-cadherin-catenin complex, the lock to cell adhesion and epithelial integrity, is altered very early in gastric tumorigenesis, particularly in diffuse-type and poorly differentiated cancers [9]. However, the particular relationship between the pattern of E-cadherin expression and some clinicopathological characteristics in gastric carcinoma remains incompletely understood. Immunohistochemistry provides a cost-effective, clinically applicable method of assessing E-cadherin expression in formalin-fixed, paraffin-embedded tissue archives, which makes it suitable for large-scale prognostic and therapeutic studies. Clarification of the patterns of expression and clinicopathologic correlations of E-cadherin in gastric carcinoma has the potential to enhance risk stratification, guide therapy decision-making, and potentially identify candidates to receive targeted therapeutic interventions. Therefore, this study was designed to systematically evaluate E-cadherin expression by immunohistochemistry in a consecutive series of gastric carcinoma cases and determine its correlations with established clinicopathological variables.

METHODS & MATERIALS

This cross-sectional study was conducted at Shahabuddin Medical College, Dhaka, Bangladesh from October 2023 to October 2024. A total of 66 histologically confirmed cases of gastric carcinoma were included in the study. Formalin-fixed, paraffin-embedded tissue samples from gastrectomy and endoscopic biopsy specimens were analyzed. Patients with inadequate tissue, poor fixation, or prior chemotherapy or radiotherapy were excluded. All cases were reviewed by two pathologists following the

WHO classification [10] and Lauren typing (intestinal or diffuse) [11]. Tissues were fixed in 10% neutral buffered formalin, processed, and stained with hematoxylin and eosin to assess tumor morphology and grade. Immunohistochemical staining for E-cadherin was performed using a monoclonal antibody (clone NCH-38, Dako, Denmark) after heat-induced antigen retrieval in citrate buffer (pH 6.0). The DAB chromogen method was used for visualization, and hematoxylin served as a counterstain. Positive and negative controls were included for quality assurance. E-cadherin expression was scored semi quantitatively as 0 (negative), 1+ (weak), 2+ (moderate), or 3+ (strong); scores 0–2+ were considered aberrant, and 3+ as preserved. Staining was independently evaluated by two observers, and discrepancies were resolved by consensus. Data were analyzed using IBM SPSS version 26.0. Descriptive statistics summarized baseline features, while the Chi-square assessed associations between E-cadherin status and clinicopathological variables. Welch's t-test compared continuous variables, and Spearman's correlation examined relationships between tumor grade and E-cadherin intensity. Multivariable logistic regression identified independent predictors of aberrant expression, with $p < 0.05$ considered statistically significant.

RESULTS

Table I represents an overview of the clinicopathological and demographic characteristics of the study population. The group included 37 men (56.1%) and 29 women (43.9%) aged 60.5 ± 6.8 years, a relatively even distribution by sex. The tumor site distribution was the following: the pylorus site was the most common site (51.5%, n=34), followed by the body (33.3%, n=22) and cardia (15.2%, n=10). The surgical specimen nature was heterogeneous, consisting of 36.4% from total gastrectomy, 42.4% from partial gastrectomy, and 21.2% from endoscopic biopsy specimens. According to the Lauren classification system, diffuse-type gastric carcinomas (57.6%, n=38) were found to occur more than intestinal-type tumors (42.4%, n=28), reflecting the aggressive nature of diffuse morphology. Histopathological grading revealed mostly poorly differentiated tumors (42.4%, n=28), followed by moderately differentiated (30.3%, n=20) and well-differentiated (27.3%, n=18) carcinomas.

Table I
Baseline Characteristics of Patients.

Variable	Category	n	%
Age (years)	Mean ± SD	60.5 ± 6.8	-
Sex	Male	37	56.1
	Female	29	43.9
Tumor site	Pylorus	34	51.5
	Body	22	33.3
	Cardiac	10	15.2
Type of sample	Total gastrectomy	24	36.4
	Partial gastrectomy	28	42.4
	Endoscopic biopsy	14	21.2
Lauren type	Diffuse	38	57.6
	Intestinal	28	42.4
Histopathological grade	Well differentiated	18	27.3
	Moderately differentiated	20	30.3
	Poorly differentiated	28	42.4

Table II depicts the immunohistochemical staining patterns of E-cadherin in the entire study population. The intensity of E-cadherin staining revealed that the majority of cases (42.4%, n=28) presented with medium (2+) staining intensity, followed by negative (24.2%, n=16), weak (21.2%, n=14), and strong (12.1%, n=8) staining intensity. Based on functional expression status, 87.9% of the tumours (n=58) expressed aberrant E-cadherin (scores 0–2+), where the immunostaining was decreased, weak, or lost, and only 12.1% (n=8) had retained strong (3+) expression.

Table II
Distribution of E-cadherin Staining Intensity and Expression Status.

Variable	Category	n	%
E-cadherin intensity	0 (Negative)	16	24.2
	1+ (Weak)	14	21.2
	2+ (Moderate)	28	42.4
	3+ (Strong)	8	12.1
E-cadherin status	Aberrant (0–2+)	58	87.9
	Preserved (3+)	8	12.1

Table III demonstrates the correlations between E-cadherin expression patterns and a number of clinicopathological variables by means of Chi-square analysis. No significant differences were noted between E-cadherin expression status and patient gender (p=0.465), tumour site location (p=0.105), or surgical sample type (p=0.215), implying that loss of E-cadherin is not dependent on these variables. Of special interest, Lauren type showed a statistically significant association with E-cadherin expression (p=0.048), with diffuse-type carcinomas having a higher proportion of aberrant expression (94.7%) compared to intestinal-type tumours (78.6%). Most significantly, histopathological grade correlated most strongly with E-cadherin status (p<0.001), such that all moderately differentiated (100%) and poorly differentiated (100%) carcinomas exhibited aberrant expression, whereas well-differentiated carcinomas exhibited preserved expression in 44.4% of cases.

Table III
Association Between E-cadherin Expression and Clinicopathological Variables (n = 66).

Variable	Category	Aberrant (0–2+) n (%)	Preserved (3+) n (%)	p-value
Sex	Male	33 (86.8)	4 (13.2)	0.465
	Female	25 (86.2)	4 (13.8)	
Tumor site	Pylorus	31 (91.2)	3 (8.8)	0.105
	Body / Cardiac	27 (84.4)	5 (15.6)	
Type of Sample	Total gastrectomy	21 (87.5)	3 (12.5)	0.215
	Partial gastrectomy	25 (89.3)	3 (10.7)	
	Endoscopic biopsy	12 (85.7)	2 (14.3)	
Lauren Type	Diffuse	36 (94.7)	2 (5.3)	0.048
	Intestinal	22 (78.6)	6 (21.4)	
Histopathological Grade	Well differentiated	10 (55.6)	8 (44.4)	< 0.001
	Moderately differentiated	20 (100.0)	0 (0.0)	
	Poorly differentiated	28 (100.0)	0 (0.0)	

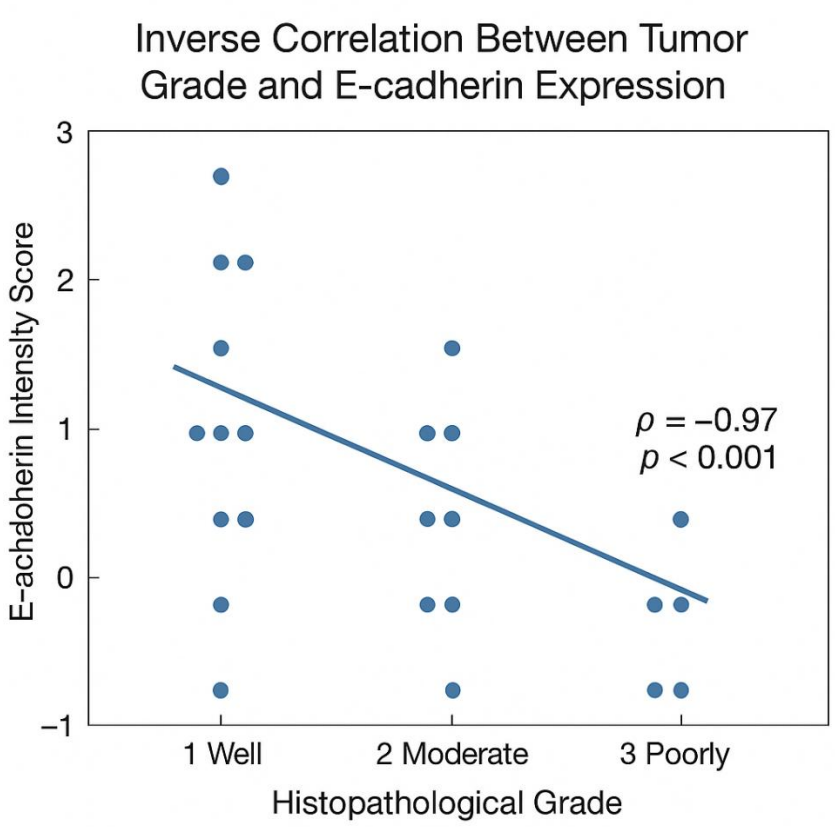

Figure 1 Inverse Correlation Between Tumor Grade and E-cadherin Expression.

This scatter-with-trend plot demonstrates a strong negative correlation between histopathological grade and E-cadherin staining intensity in gastric carcinoma. Well-differentiated tumors show the highest E-cadherin intensity scores, whereas poorly differentiated tumors exhibit markedly reduced expression. The Spearman correlation coefficient ($\rho = -0.97$, $p < 0.001$) confirms a highly significant inverse trend, indicating that loss of E-cadherin expression is strongly associated with increasing tumor dedifferentiation and hence greater biological aggressiveness (*Figure 1*).

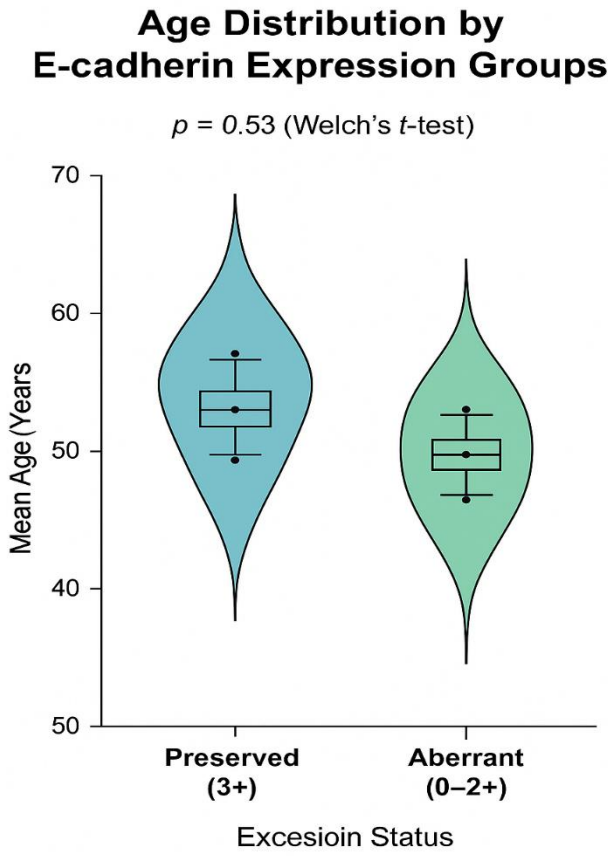

Figure 2 Age Distribution by E-cadherin Expression Groups

This violin-plus-box plot compares the age distribution between patients with Preserved (3+) and Aberrant (0–2+) E-cadherin expression. Both groups show nearly identical median and mean ages (≈ 61.3 ± 7.2 years vs 60.4 ± 6.7 years), and the distributions overlap substantially. The Welch's *t*-test ($p = 0.53$) confirms there is no statistically significant age difference between the two expression categories, indicating that E-cadherin loss is independent of patient age in this cohort (*Figure 2*).

The multivariable logistic regression analysis in *Table IV* identifies independent predictors of aberrant E-cadherin expression, adjusting for confounders. Histopathologic grade was the strongest independent predictor (OR=7.81, 95% CI: 2.45–24.85, $p<0.001$), showing that with every increase in tumor grade, the probability of aberrant E-cadherin expression significantly increases. Lauren type, intestinal type over diffuse type (OR=0.42, 95% CI: 0.18–0.98, $p=0.045$), was a protective factor against aberrant expression. Conversely, tumor site (pylorus vs. body/cardia), year of age, and sex (male vs. female) did not achieve statistical significance as independent predictors within the multivariable model, suggesting that their association with E-cadherin expression is confounded by other variables, in particular tumor grade.

Table IV Multivariable Logistic Regression for Aberrant E-cadherin Expression.

Predictor	OR	95 % CI (2.5–97.5 %)	p-value
Histopathological grade (per ↑)	7.81	2.45 – 24.85	< 0.001
Lauren type: Intestinal (vs Diffuse)	0.42	0.18 – 0.98	0.045
Site: Pylorus (vs Body/Cardia)	1.31	0.48 – 3.59	0.597
Age (per year)	1.02	0.94 – 1.10	0.624
Sex: Male (vs Female)	1.48	0.56 – 3.90	0.431

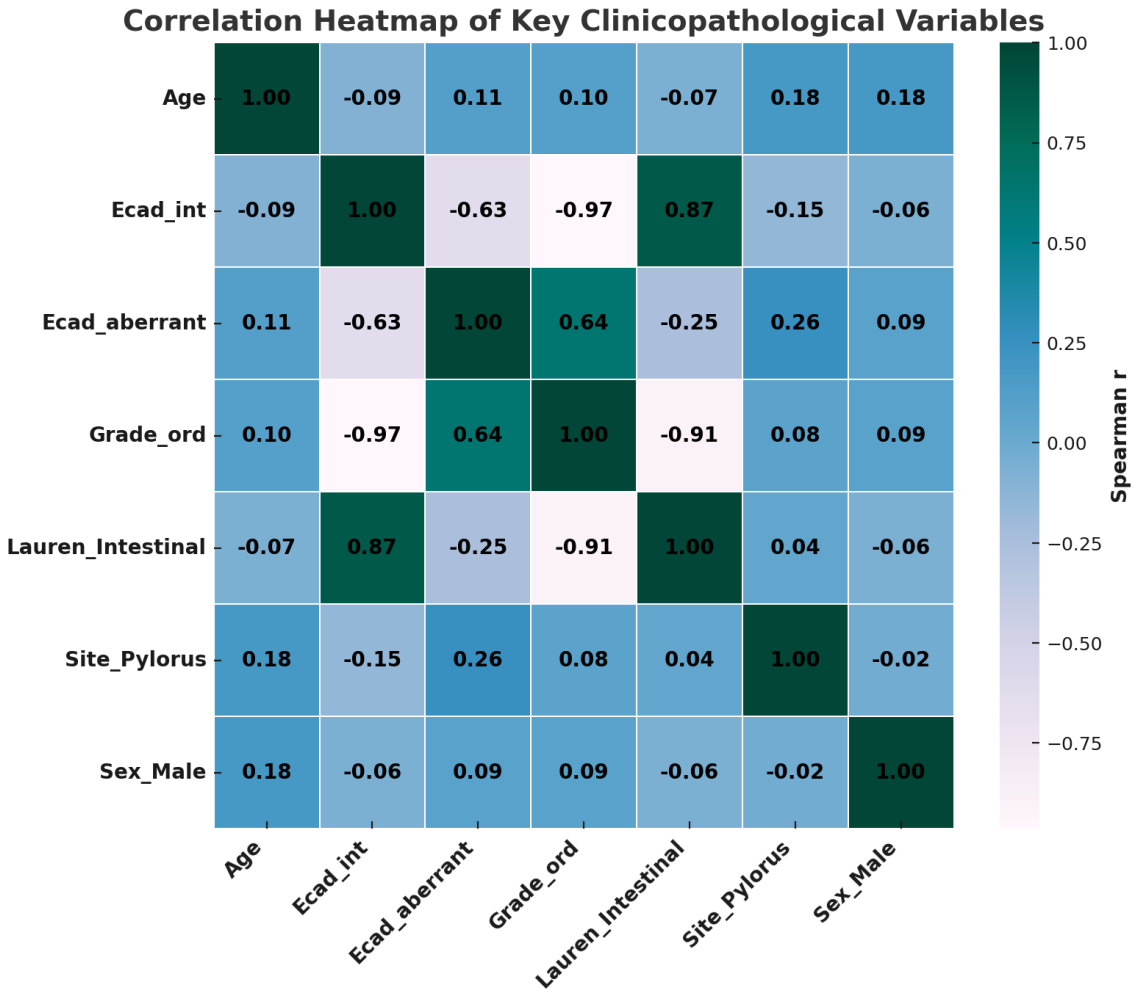

Figure 3 Correlation Heatmap of Key Clinicopathological Variables.

This heatmap illustrates the Spearman correlation (r) values among key clinicopathological variables, including age, tumor grade, Lauren type, sex, and E-cadherin indices (intensity and aberrant status). A strong negative correlation (r = –0.97) is observed between tumor grade and E-cadherin intensity, indicating that higher histopathological grade is strongly associated with lower E-cadherin expression. Other correlations remain weak (r close to 0), suggesting minimal association of age, sex, or site with E-cadherin loss in gastric carcinoma (*Figure 3*).

DISCUSSION

This study reveals that aberrant E-cadherin expression is frequent in gastric carcinoma and occurs in 87.9% of the cases studied, and it presents apparent clinicopathological correlations, particularly with tumor differentiation grade and Lauren histological type. These findings are consistent with Xing et al., wherein it has been noted that loss of regular E-cadherin expression is a sign of gastric cancer progression and is associated with increased metastatic potential and poorer clinical outcomes [12]. The predominance of abnormal E-cadherin expression in this study corroborates findings that E-cadherin down-regulation is an early process in gastric tumorigenesis and is the crucial step in the dedifferentiation process from well-differentiated to poorly differentiated phenotypes [13]. The inverse relationship between tumor grade and E-cadherin expression in our analysis (Spearman $\rho = -0.97$, $p < 0.001$) indicates that loss of E-cadherin is correlated with low cellular differentiation and increased biological aggressiveness. Notably, 44.4% of well-differentiated gastric carcinomas retained strong E-cadherin expression (3+), whereas all moderately and poorly differentiated carcinomas had abnormal expression patterns. Such a result is consonant with the hypothesis that diminished E-cadherin expression facilitates loss of cellular cohesion and dedifferentiation, a key feature of malignant transformation [14]. The persistence of E-cadherin expression in well-differentiated tumors suggests that epithelial phenotype is present in low-grade, early-stage carcinomas, whereas its gradual loss is associated with dedifferentiation of the carcinoma and acquisition of more virulent biological features. The high correlation between Lauren histology type and E-cadherin expression ($p = 0.048$) is particularly noteworthy since 94.7% of diffuse-type carcinomas revealed aberrant expression compared with 78.6% of intestinal-type tumors. This finding is consistent with Ohene et al., confirming that diffuse-type gastric cancers show higher degrees of E-cadherin loss and disruption of the E-cadherin-catenin complex [15]. The diffuse type of cancer comprises poorly cohesive cells that typically lack adhesion molecules, and our immunohistochemical observations essentially mirror this morphological phenotype. The molecular basis of increased loss of E-cadherin in diffuse-type tumours may be due to the common incidence of CDH1 gene mutations, epigenetic suppression by promoter methylation, or the induction of transcriptional repressors such as Snail and Slug, which prevent expression of E-cadherin [16]. In contrast, intestinal-type gastric cancers that arise characteristically through multistep intestinal metaplasia and are typically associated with *Helicobacter pylori* infection and environmental exposure maintain relatively better

epithelial organization and greater degrees of preserved E-cadherin expression [17]. Multivariable logistic regression analysis revealed that histopathological grade was the strongest independent predictor of aberrant E-cadherin expression (OR = 7.81, $p < 0.001$) and that with every increase in tumor grade, it strongly enhanced the odds of loss of E-cadherin. Such a strong correlation demonstrates that E-cadherin expression status is a good biomarker for tumour differentiation and malignancy independent of other factors. The protective effect of intestinal Lauren type against aberrant expression (OR = 0.42, $p = 0.045$) in the multivariable analysis also suggests that histological subtype and differentiation status are strongly correlated, and that intestinal-type tumors preserve relatively well cellular differentiation and epithelial characteristics [18]. Notably, patient features including sex, age, and original tumour location did not show up in the final multivariable model as independent predictors of E-cadherin expression patterns, indicating that none of these influences significantly impact E-cadherin expression patterns after controlling for tumour grade. Saad et al. showcased the prognostic significance of E-cadherin loss, suggesting that its deficiency or reduced expression correlates with increased risk of lymph node metastasis, peripheral blood micro metastasis, and reduced overall and recurrence-free survival [19]. Furthermore, dysregulation of the entire E-cadherin-catenin complex, including α-, β-, and γ-catenins, has been implicated in early gastric carcinogenesis and more frequently encountered in poorly differentiated and diffuse-type pathways [20]. These alterations at the molecular level are potential therapeutic targets, and the latest evidence suggests that either resumption of E-cadherin expression or inhibition of EMT-inducing pathways can be used to inhibit tumor growth and metastasis. Future research should clarify the mechanistic link between loss of E-cadherin and metastatic capacity, determine its utility for prognostication and treatment selection in patients, and investigate ways to therapeutically modulate E-cadherin expression or related adhesion pathways.

LIMITATIONS

The study was also restricted by having a comparatively small sample size of only 66 cases, which may restrict the external validity of the results to more general and more diverse patient populations. A cross-sectional study design inherently precludes taking account of temporal associations or long-term prognostic correlations between E-cadherin expression and patient survival results.

CONCLUSION

This study demonstrates that aberrant expression of E-cadherin is prevalent in gastric carcinoma, with 87.9% of the cases, and is highly associated with poor tumor differentiation and diffuse type histology. The inverse relationship between E-cadherin expression and tumor grade ($\rho = -0.97$, $p < 0.001$) makes E-cadherin a probable prognostic biomarker for the identification of tumor aggressiveness and prediction of clinical outcome. Multivariable analysis confirmed histopathological grade as the strongest independent predictor of abnormal E-cadherin expression, confirming its employment in patient risk stratification. These findings suggest that E-cadherin determination by immunohistochemistry may contribute to the conventional prognostic factors and provide valuable information for therapy planning and surveillance maneuvers in gastric carcinoma patients.

RECOMMENDATIONS

Future studies should incorporate larger, multicenter cohorts with comprehensive follow-up data to establish the definitive prognostic value of E-cadherin expression and its relationship to overall and recurrence-free survival. Molecular characterization by combining immunohistochemistry with genetic sequencing and epigenetics would delineate the mechanisms of E-cadherin loss and offer active therapeutic targets.

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CONFLICT OF INTEREST

None declared.

ETHICAL APPROVAL

The study was approved by the Institutional Ethics Committee.

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