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# ASSOCIATION OF CDH1 -160 C/A (rs16260) POLYMORPHISM WITH METASTASIS IN BREAST CANCER

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## ABSTRACT

**Background:** Metastasis is the primary cause of mortality in breast cancer, and the loss of E-cadherin function (encoded by the CDH1 gene) is a key molecular event in this process. The -160 C/A (rs16260) single nucleotide polymorphism (SNP) in the CDH1 gene promoter has been hypothesized to affect gene expression and potentially increase the risk of metastasis. This study analyzes the association between the CDH1 -160 C/A polymorphism and metastasis in patients at Prof. dr. I.G.N.G. Ngoerah General Hospital.

**Methods:** This study utilized an analytical-observational cross-sectional design, involving 22 breast cancer patients at Prof. dr. I.G.N.G. Ngoerah General Hospital. Stored genomic DNA samples were isolated from venous blood. The rs16260 polymorphism genotype was determined using the Polymerase Chain Reaction (PCR) method followed by sequencing. Clinical data, including metastasis status (M0 vs. M1), were obtained from medical records. The association between genotype (wild type [CC] vs. mutant [CA/AA]) and metastasis status was analyzed using Fisher's Exact Test, with Odds Ratios (OR) and 95% Confidence Intervals (95% CI) calculated to estimate the magnitude of the association.

**Results:** The prevalence of mutant genotypes (CA/AA) in this study population was high (86.4%). Of the 22 subjects, 4 (18.2%) had metastasis (M1). Statistical analysis showed no significant association between the CDH1 -160 C/A polymorphism genotype and metastasis status (Fisher's Exact Test, p-value = 0.073).

**Conclusion:** Based on the statistical significance test, no significant association was found between the CDH1 -160 C/A (rs16260) polymorphism and metastasis in breast cancer patients at Prof. dr. I.G.N.G. Ngoerah General Hospital. Further research with a much larger sample size is required to validate these findings and clarify the true role of this polymorphism in breast cancer progression.

**Keywords:** Breast Cancer, Metastasis, CDH1, Polymorphism, rs16260.

## INTRODUCTION

Breast cancer remains a formidable global health challenge, representing a biological and epidemiological crisis.[1,2] According to the International Agency for Research on Cancer (IARC) GLOBOCAN 2022 data, breast cancer accounted for approximately 2.3 million new diagnoses (2,296,840 new case) and 666,103 deaths worldwide, cementing its status as the most prevalent malignancy and the leading cause of cancer-related mortality among women.[2] In Indonesia, the trajectory of breast cancer mirrors these alarming global trends but is compounded by unique geographic and systemic challenges.[1] In 2022 alone, Indonesia recorded over 66,271 new cases, constituting approximately 30.1% of all new cancer diagnoses in the country.[1] The mortality burden is equally staggering, with over ±22,000 lives lost annually.[1] Factors such as genetics, family history, and age are non-modifiable risks, while lifestyle choices and hormonal exposure serve as modifiable contributors.[3–6]

At the cellular level, the maintenance of tissue architecture is governed by cell-cell adhesion complexes.[7] Central to this machinery is E-cadherin (Epithelial-cadherin), a transmembrane glycoprotein that functions as the core component of adherens junctions.[7,8] The intracellular domain of E-cadherin interacts with

the catenin family of proteins, specifically p120-catenin and β-catenin.[7] β-catenin, in turn, binds to α -catenin, which links the entire adhesion complex to the actin cytoskeleton.[7] This structural continuum between the extracellular environment and the intracellular cytoskeleton is vital for maintaining cell polarity and tissue integrity.[7]

The loss of E-cadherin expression is a hallmark event in the malignant progression of epithelial tumors.[9] This downregulation disrupts adherens junctions, leading to the detachment of tumor cells from the primary mass.[8–10] This event is the initiating step in the Epithelial-to-Mesenchymal Transition (EMT), a complex developmental program hijacked by cancer cells.[7] During EMT, stationary epithelial cells repress adhesion molecules (like E-cadherin) and upregulate mesenchymal markers (such as N-cadherin), acquiring a motile, invasive phenotype that facilitates intravasation into the vasculature.[7,8,11,12]

The E-cadherin protein is encoded by the CDH1 gene (Cadherin-1), located on chromosome 16q22.1.[13] The gene spans approximately 100 kb (98,250) and contains 16 exons.[13] Of particular interest is the single nucleotide polymorphism (SNP) located at position -160 in the promoter region of the CDH1 gene, relative to the transcriptional start site.[14,15] Designated as rs16260, this polymorphism involves a C to A substitution.[14,15] Meta-analyses suggest that the 'A' allele confers increased susceptibility to breast cancer.[16,17]

The relationship between CDH1 polymorphisms and cancer prognosis involves two pivotal mechanisms within the metastatic process.[9,18,19] While the downregulation of E-cadherin and the subsequent EMT are essential for the initial steps of metastasis, invasion and dissemination, the formation of clinically detectable metastases requires the reverse process: Mesenchymal-to-Epithelial Transition (MET).[7,9,18,20,21]

The vast majority of studies on the CDH1 -160 C/A polymorphism have been conducted in Kurdish, Swiss, Czech, Italy, China, and India populations.[15–17] Data regarding the Indonesian population, and specifically the genetically distinct Balinese ethnic group, are virtually non-existent. Furthermore, most studies focus on cancer risk (susceptibility) rather than progression (metastasis).

Therefore, this study aims to bridge this critical gap by investigating the association between the CDH1 -160 C/A (rs16260) polymorphism and the incidence of distant metastasis in breast cancer patients treated at Prof. dr. I.G.N.G. Ngoerah General Hospital. We hypothesize that the distribution of genotypes in this population may differ from global norms and that the polymorphism may exert a distinct influence on metastatic progression, potentially illuminating the complex interplay between genetic predisposition and the EMT-MET plasticity axis.

## METHODS

This research employed an observational analytic design with a cross-sectional approach to evaluate the association between the CDH1 -160 C/A genotype and metastatic status. The study was conducted at the Integrated Biomedical Laboratory and the Department of Biochemistry, Faculty of Medicine, Udayana University, utilizing archived biological materials (genomic DNA) and matched medical records of female patients diagnosed with breast cancer at Prof. dr. I.G.N.G. Ngoerah General Hospital. Inclusion criteria mandated hat samples must have sufficient quality for PCR amplification and be accompanied by complete medical records documenting TNM staging, age, parity, and histological subtype. Samples showing degradation or equivocal staging data were excluded. A total of 22 samples met all eligibility criteria. Ethical approval was granted by the Research Ethics Committee of the Faculty of Medicine, Udayana University (Ethical Clearance No: 0458/UN14.2.2.VII.14/LT/2025).

Genotyping was performed using Polymerase Chain Reaction (PCR) followed by direct Sanger sequencing. Specific primers were synthesized to amplify a 469 bp fragment of the CDH1 promoter region encompassing the -160 locus, consisting of the forward primer 5'-GCCCCGACTTGTCTCTCTAC-3' and the reverse primer 5'-ATTGGCTGAGGGTTCACCTG-3'. The amplification was carried out in a 35 µL reaction volume comprising 17.5 µL of GoTaq® Green Master Mix (Promega, USA), 1.4 µL of the forward primer (10 µM), 1.4 µL of the reverse primer (10 µM), 1.4 µL of genomic DNA template, and 13.3 µL of nuclease-free water.

Thermal cycling was performed in a calibrated thermocycler with an initial denaturation step at 95°C for 5 minutes to activate the polymerase. This was followed by 40 amplification cycles consisting of denaturation at 94°C for 40 seconds, annealing at 58°C for 30 seconds, and extension at 72°C for 50 seconds. A final extension step was conducted at 72°C for 10 minutes to ensure full-length amplicon synthesis. Subsequently, PCR products were resolved on a 1% agarose gel in TBE buffer, stained with ethidium bromide, and visualized under a UV transilluminator to verify the specific amplification of the expected 469 bp fragment prior to sequencing. Sequence analysis was performed using Chromas software, and validation was conducted using the NCBI Basic Local Alignment Search Tool (BLAST).

Data management and analysis were conducted using IBM SPSS Statistics version 27.0. Descriptive statistics were expressed as Mean ± Standard Deviation (SD) and frequencies. The association between CDH1 genotypes (dichotomized as Wild Type [CC] vs. Mutant [CA/AA]) and metastasis status (M0 vs. M1) was evaluated using Fisher's Exact Test due to the limited sample size. The Odds Ratio (OR) with 95% Confidence Intervals (CI) was calculated to quantify the strength of the association, with a p-value $< 0.05$ considered statistically significant.

## RESULT

The study analyzed a cross-sectional of 22 female breast cancer patients. The mean age of the participants at the time of diagnosis was 56.45 ± 13.06 years. Reproductive history analysis showed a mean parity of 2.50 ±1.41. Histopathological profiling revealed that the hormone receptor-positive subtypes were dominant: Luminal B accounted for 36.4% of cases, followed by Luminal A at 22.7%.

A critical observation from the clinical data is the advanced stage at presentation. A substantial proportion of patients (36.4%) were diagnosed at Stage IIIB, and 18.2% presented with Stage IV disease. Regarding the primary outcome, 4 patients (18.2%) were classified as having distant metastasis (M1), while 18 (81.8%) were non-metastatic (M0).

**Table 1.** Demographic and Clinical Characteristics of Breast Cancer Patients (N=22)

| Characteristic | Category | Frequency (n) | Percentage (%) |
|---|---|---|---|
| **Age (Years)** | Mean ± SD | 56,45 ± 13,059 | - |
| | Median (Min-Maks) | 54 (39-86) | - |
| **Parity** | mean ± SD | 2,50 ± 1,406 | - |
| | Median (Min-Max) | 2,50 (0-6) | - |
| **Histopato-logical Subtype** | Luminal A | 5 | 22,7 |
| | Luminal B | 8 | 36,4 |
| | HER2 | 3 | 13,6 |
| | TNBC | 4 | 18,2 |
| | Luminal-HER2 | 2 | 9,1 |
| **Tumor Size (T)** | T1 | 3 | 13,6 |
| | T2 | 2 | 9,1 |
| | T3 | 6 | 27,3 |
| | T4 | 11 | 50,0 |
| **Nodal Status (N)** | N0 | 9 | 40,9 |
| | N1 | 10 | 45,5 |
| | N2 | 3 | 13,6 |
| **Metastasis (M)** | M0 | 18 | 81,8 |
| | M1 | 4 | 18,2 |
| **Clinical Stage** | IA | 3 | 13,6 |
| | IIA | 1 | 4,5 |
| | IIB | 3 | 13,6 |
| | IIIA | 3 | 13,6 |
| | IIIB | 8 | 36,4 |
| | IV | 4 | 18,2 |

PCR amplification of the CDH1 promoter region successfully yielded a specific 469 bp amplicon in all samples. BLAST analysis of the sequenced products confirmed 98-99% identity with the human CDH1 gene reference sequence, validating the specificity of the primer set.

The distribution of genotypes for the rs16260 polymorphism was markedly skewed in this Prof. dr. I.G.N.G. Ngoerah General Hospital patients. The Mutant genotype (defined as the presence of the 'A' allele, either heterozygous CA or homozygous AA) was the predominant form, identified in 86.4% (19/22) of patients. Conversely, the Wild Type (CC) genotype was observed in only 13.6% (3/22) of the participants.

**Table 2.** Genotype Frequencies of CDH1 -160 C/A (rs16260)

| Variabel | Category | Frequency (n) | Percentage (%) |
|---|---|---|---|
| **Genotype** | CC | 3 | 13,6 |
| | CA | 10 | 45,5 |
| | AA | 9 | 40,9 |
| | Wild Type (CC) | 3 | 13,6 |
| | Mutant (CA/AA) | 19 | 86,4 |
| | **Total** | **22** | **100,0** |

To determine the prognostic implication of the rs16260 polymorphism, we analyzed the association between the genotype (dichotomized as Wild Type [CC] vs. Mutant [CA/AA]) and the presence of distant metastasis (M1).

The contingency table analysis (Table 3) revealed a striking disparity. Among the small group of patients with the Wild Type

(CC) genotype (n=3), a majority (66.7%, n=2) presented with distant metastasis. In contrast, among the larger group of patients with the Mutant (CA/AA) genotype (n=19), only 10.5% (n=2) exhibited metastasis.

**Table 3.** Association between CDH1 -160 C/A Genotype and Metastasis Status

| CDH1 Genotype | M1 n (%) | M0 n (%) | Total | OR (95% CI) | p-value |
|---|---|---|---|---|---|
| **Mutant (CA/AA)** | 2 (10.5%) | 17 (89.5%) | 19 | **0.059** (0.004 – 0.979) | **0.073** |
| **Wild Type (CC)** | 2 (66.7%) | 1 (33.3%) | 3 | | |
| **Total** | 4 (18.2%) | 18 (81.8%) | 22 | | |

*Statistical test used: Fisher's Exact Test.*

Fisher's Exact Test, deemed more appropriate for small sample sizes, was employed for data interpretation. The test yielded a p-value of 0.073, which exceeds the significance level of $\alpha = 0.05$. Consequently, it can be concluded that there is no statistically significant association between the CDH1 -160 C/A (rs16260) polymorphism and metastasis status in this study sample. However, risk factor analysis indicated that individuals with the mutant genotype (CA or AA) exhibited significantly lower odds of metastasis compared to those with the wild-type (CC) genotype. The obtained Odds Ratio (OR) was 0.059. The potential significance of this finding was supported by the 95% Confidence Interval (CI), which ranged from 0.004 to 0.979. Indicating that the mutant genotype (CA/AA) may exert a protective effect against breast cancer metastasis. Nevertheless, based on the primary hypothesis testing using Fisher's Exact Test, the overall association remains statistically non-significant ($p = 0.073$).

## DISCUSSION

Genetic polymorphism constitutes variations within DNA sequences, manifesting as genotypic or allelic differences within a population.[22] These variations occur as single nucleotide differences, known as single nucleotide polymorphisms (SNPs), arising from substitutions, deletions, and insertions.[22] A notable SNP located within CDH1, the gene responsible for regulating the E-cadherin protein, is -160 C/A (rs16260).[13,14] This study focuses on the relationship between the CDH1 -160 C/A (rs16260) polymorphism and breast cancer metastasis. Investigating the genetic factors underlying metastasis is crucial for the development of improved prognostic and therapeutic strategies.

The rs16260 polymorphism is situated in the promoter region of the CDH1 gene, located on chromosome 16q22.[13] The CDH1 gene encodes E-cadherin, a transmembrane glycoprotein functioning as a tumor suppressor protein.[7] Its primary function is serving as a core component of adherens junctions, mediating calcium-dependent cell-cell adhesion and maintaining the integrity and architecture of epithelial tissues.[7,8] In a pathological context, the loss or reduction of E-cadherin function is a key event in cancer progression.[16] Downregulation of E-cadherin expression disrupts cellular cohesion and acts as a primary trigger for the Epithelial-to-Mesenchymal Transition (EMT).[7,12] EMT is a transdifferentiation process wherein stationary, polarized epithelial cells lose their characteristics and transform into cells with a motile and invasive mesenchymal phenotype.[12,23] This transformation enables tumor cells to detach from the primary tumor mass, invade surrounding tissues, enter the circulation, and ultimately form metastatic colonies in distant organs.[7,9] Consequently, there is a strong biological basis implying that individuals with the mutant genotype, who exhibit lower E-cadherin expression, would mechanistically face a higher risk of metastasis.

Research results on the CDH1 -160 C/A (rs16260) polymorphism in breast cancer patients at Prof. dr. I.G.N.G. Ngoerah General Hospital present intriguing preliminary findings regarding the prevalence of the mutant genotype (CA/AA), which was observed to be quite high at approximately 86.4%, whereas the wild-type genotype (CC) in the sample population was relatively small at approximately 13.6%. This figure stands in contrast to the study by Zarei et al.[17], where the wild-type genotype (CC) was approximately 47%.[17] The high prevalence of the mutant genotype raises the hypothesis that the frequency of CA/AA may be inherently higher in the specific ethnicity studied, a notion consistent with previous reports stating that the effects of this polymorphism may vary across ethnicities.[17] These findings provide valuable primary data and underscore the importance of genetic studies in local populations to map existing genetic diversity.

Although this study did not find a statistically significant association between the CDH1 -160 C/A polymorphism and breast cancer metastasis (OR = 0.059; p = 0.073), these findings provide crucial comparative data. In contrast, a study by Zarei et al.[17] on a Kurdish population with a larger sample size reported that the 'A' allele significantly increased the risk of metastasis.[17] These divergent results highlight the likelihood of complex gene-environment interactions or the influence of differing genetic backgrounds across populations. Therefore, the results of this study do not refute previous findings; rather, they enrich the scientific landscape by demonstrating that this association may not be uniform across all populations and requires further investigation to understand the modulating factors. Similarly, this contrasts with evidence from a meta-analysis by Ma et al.[16], which concluded that this polymorphism is a risk factor for breast cancer susceptibility.[16]

This suggests that the role of the CDH1 -160 C/A (rs16260) polymorphism in tumor initiation may differ from its role in metastatic progression, or that its influence is modified by other factors specific to this study. As previously explained, decreased E-cadherin expression is a primary trigger for EMT.[12] However, for tumor cells to develop into macroscopic metastatic masses in target organs, they require the ability to cease motility and resume proliferation, a process termed Mesenchymal-Epithelial Transition (MET).[18] MET is the expression of EMT reversibility; however, the exact mechanisms underlying MET, including where and how it occurs and how it facilitates metastasis formation, remain largely elusive.[18] Compared to EMT, the molecular mechanisms mediating MET are relatively less characterized.[18] This provides a potential explanation for the low incidence of metastasis in the population possessing the mutant genotype in this study; while decreased E-cadherin expression in these conditions promotes EMT, polymorphisms in the promoter region may conceivably impede the MET process, thereby preventing tumor cells from establishing colonies in distant organs or undergoing metastasis.

In the breast cancer patient population at Prof. dr. I.G.N.G. Ngoerah General Hospital, an Odds Ratio (OR) of 0.059 was observed in the metastasis analysis. Although this did not reach conclusive statistical significance ($p = 0.073$), it represents a preliminary statistical signal warranting further research. This observation, emerging from analysis within a smaller wild-type group, serves as a thought-provoking starting point for follow-up studies designed to explore these dynamics with greater statistical power.

Consequently, further research with specific sample characteristics, including defined sizes and clinical distributions, as well as larger sample sizes, is required to provide a more specific profile of the genetics within the local breast cancer patient population. In patient groups with specific characteristics, the mutant genotype (CA/AA) may be highly dominant, a phenomenon that merits deeper exploration.

## CONCLUSION AND SUGGESTIONS

Following the data analysis and discussion, it is concluded that the statistical evaluation has not established a significant relationship between the CDH1 -160 C/A (rs16260) polymorphism and metastatic events in breast cancer patients at Prof. dr. I.G.N.G. Ngoerah General Hospital.

In light of the conclusions and limitations of this study, several recommendations are proposed for future research. Building upon the foundation laid by this investigation, it is highly recommended that subsequent studies utilize larger sample sizes with more uniform subject characteristics. This approach would not only enhance statistical power and facilitate a broader generalization of findings but also serve to validate the intriguing preliminary observations reported here. Furthermore, to more definitively evaluate the causal relationship between CDH1 -160 C/A (rs16260) polymorphisms and metastatic risk, the adoption of a prospective cohort design is advised. Unlike the cross-sectional design employed in this current study, which effectively served for hypothesis generation, a longitudinal cohort approach would enable the rigorous analysis of cause-and-effect relationships by tracking disease progression over time, thereby providing evidence with a higher degree of certainty.

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