The Significance of Endothelin-Converting Enzyme-1 and Mannan-Binding Lectin as a Predictive Biomarkers in Breast Cancer Malignancy

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ABSTRACT

Breast cancer, particularly Invasive Ductal Carcinoma (IDC), is the most prevalent malignancy among women worldwide. Early detection remains challenging, especially in younger women. Endothelin-Converting Enzyme-1 (ECE-1) and Mannan-Binding Lectin (MBL) have emerged as potential biomarkers due to their roles in tumor angiogenesis, immune modulation, and inflammatory pathways. This study evaluated serum ECE-1 and MBL levels in distinguishing malignant from benign breast disease. A cross-sectional study was conducted on 160 females (20–40 years) divided into three groups: IDC (n=40), benign braeast disease (BBD, n=40), and healthy controls (n=80). Serum ECE-1 and MBL were quantified using sandwich ELISA. Data were analyzed with one-way ANOVA, Tukey post-hoc tests, Pearson correlation, and ROC curve analysis, with $p<0.05$ considered significant. IDC patients showed significantly elevated serum ECE-1 (62.57 ± 8.34 U/L) and MBL (221.03 ± 23.31 ng/mL) compared to BBD (28.12 ± 3.51 U/L; 160.6 ± 21.86 ng/mL) and controls (8.54 ± 1.56 U/L; 81.82 ± 6.41 ng/mL) ($p<0.001$). Both biomarkers correlated positively within patient groups (IDC: r=0.373, p=0.018; BBD: r=0.495, p=0.001). ROC analysis revealed higher diagnostic accuracy for ECE-1 (AUC=0.773; sensitivity 72.8%, specificity 93.1%) than MBL (AUC=0.717; sensitivity 67.2%, specificity 89.7%. Elevated serum ECE-1 and MBL are significantly associated with IDC and may serve as useful non-invasive biomarkers. ECE-1 demonstrated superior diagnostic performance, suggesting its potential utility—alone or within a biomarker panel—for early breast cancer detection and differentiation from benign disease.

Keywords: Endothelin-Converting Enzyme-1 (ECE-1), Invasive Ductal Carcinoma (IDC), Mannan-Binding Lectin, Tumor Angiogenesis, Inflammatory Mediators in Cancer.

INTRODUCTION

Breast cancer is the most frequently diagnosed cancer among women worldwide and a leading cause of cancer-related mortality. Invasive Ductal Carcinoma (IDC) is the most common histological subtype, accounting for nearly 70–80% of all breast malignancies[1]. Despite advances in imaging and molecular diagnostics, a significant number of cases are still diagnosed at late stages, particularly in younger women whose dense breast tissue complicates early radiological detection[2]. This underscores the need for reliable, non-invasive biomarkers that can aid in the early diagnosis, monitoring, and potential stratification of disease aggressiveness[3].

Recent research in cancer biology highlights the critical role of the tumor microenvironment and associated inflammatory pathways in driving carcinogenesis and metastasis. Among the mediators implicated in this process is Endothelin-1 (ET-1)[4], a potent vasoconstrictive and mitogenic peptide. ET-1 exerts its biological effects primarily through the activation of endothelin receptors (ETA and ETB), contributing to angiogenesis, epithelial-to-mesenchymal transition (EMT), and resistance to apoptosis—all of which are pivotal in tumor development and invasion[5].

The production of ET-1 from its inactive precursor, big endothelin-1, is catalyzed by Endothelin-Converting Enzyme-1 (ECE-1), a zinc-dependent metallo-protease expressed in various tissues including vascular endothelium and tumor cells. Overexpression of ECE-1 has been identified in several solid tumors[6], including breast cancer, where it plays a role in tumor progression, local invasion, and neovascularization. However, while tissue expression of ECE-1 has been extensively studied, limited data are available regarding its circulating levels and potential use as a serum biomarker for early breast cancer detection[7].

Mannan-binding lectin (MBL), also known as mannose-binding lectin, is a soluble pattern recognition molecule that plays a crucial role in the innate immune system. MBL recognizes carbohydrate patterns on the surfaces of a wide range of pathogens and altered self-cells, activating the lectin pathway of the complement system through MBL-associated serine proteases (MASPs)[8]. MBL contributes to immune surveillance by recognizing abnormal glycosylation patterns on transformed or tumor cells, potentially targeting them for destruction through complement activation or opsonophagocytosis[9].

Given the increasing burden of breast cancer in younger populations and the limitations of current diagnostic approaches, investigating novel biomarkers such as ECE-1 and MBL are of growing importance. This study focuses specifically on the significance of serum ECE-1 and MBL levels in distinguishing malignant from benign breast conditions and explores its potential utility as a biomarker in the diagnosis and characterization of breast cancer malignancy.

MATERIALS AND METHODS

Study Design and Participants: A cross-sectional, was undertaken from 25 December 2024 to 15 May 2025. One-hundred-sixty females

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(20-40 y) attending Al-Imamien Al-Kadhimein Medical City and Al-Amal Hospital, Baghdad, divided into the following three groups:

Group 1 – Invasive Ductal Carcinoma (IDC) Group. Composed of 40 women with a histopathological confirmed diagnosis of IDC.

Group 2 – Benign Breast Disease (BBD) Group. Comprised 40 women diagnosed with benign breast conditions.

Group 3 – Healthy Control Group. Included 80 healthy women with no personal or family history of breast disease.

Inclusion Criteria
- Females aged 20–40 years.
- Confirmed IDC or benign breast disease by histopathology.
- Healthy controls with no breast abnormalities.
- No use of anti-inflammatory or lipid-altering medications.
- Provided informed consent.

Exclusion Criteria
- Pregnant or lactating.
- Smokers or recent ex-smokers.
- Chronic diseases (liver, kidney, diabetes, autoimmune).
- Recent hormonal therapy use.
- Family or personal history of breast/ovarian cancer.
- Unverified or incomplete tumor diagnosis.

Sample Collection and Biochemical Assays: Ten mL of fasting venous blood were collected from each participant using sterile, plain Vacutainers tubes. Blood samples were allowed to clot at room temperature and centrifuged at 4,000 rpm for 10 minutes at 22 °C. The serum was then separated, aliquoted, and stored at –80 °C until analysis. Quantification of Endothelin-Converting Enzyme-1 (ECE-1) and Mannan-binding lectin (MBL) were performed using a human-specific sandwich ELISA kit (Elabscience®, PRC), following the manufacturer's standard protocol.

Consent of Ethics: In the study, venous blood was withdrawn. Written consent was sought and secured from all study members before their samples were collected. The Ethical and Scientific Committee of the College of Medicine - Al-Nahrain University reviewed and approved the study proposal (Ref. No. 20241149 on 7/1/2025).

Statistical Analysis: SPSS-25 was utilized. Data are mean±SD. Inter-group differences were examined with one-way ANOVA and Tukey post-hoc tests. Pearson correlation gauged relationships among biomarkers. Diagnostic performance was assessed via ROC analysis. Significance was accepted at $p < 0.05$ (two-tailed)[10,11].

RESULTS

Out of 160 females, 40 females were with malignant breast tumor, 40 females with benign breast tumor and 80 healthy control females (Figure 1).

The findings of this study revealed significant differences in age and inflammatory markers between the groups and the results of age, CRP, and lipid profile appear in Table 1 and Figures 2 and Figure 3. Specifically, females with malignant breast tumors exhibited a significantly higher mean age (38.95 ± 0.47 years) compared to those with benign tumors (35.38 ± 0.96 years) and the control group (34.64 ± 0.75 years), with a highly significant p-value of 0.001. Additionally, the inflammatory marker C-reactive protein (CRP) was markedly elevated

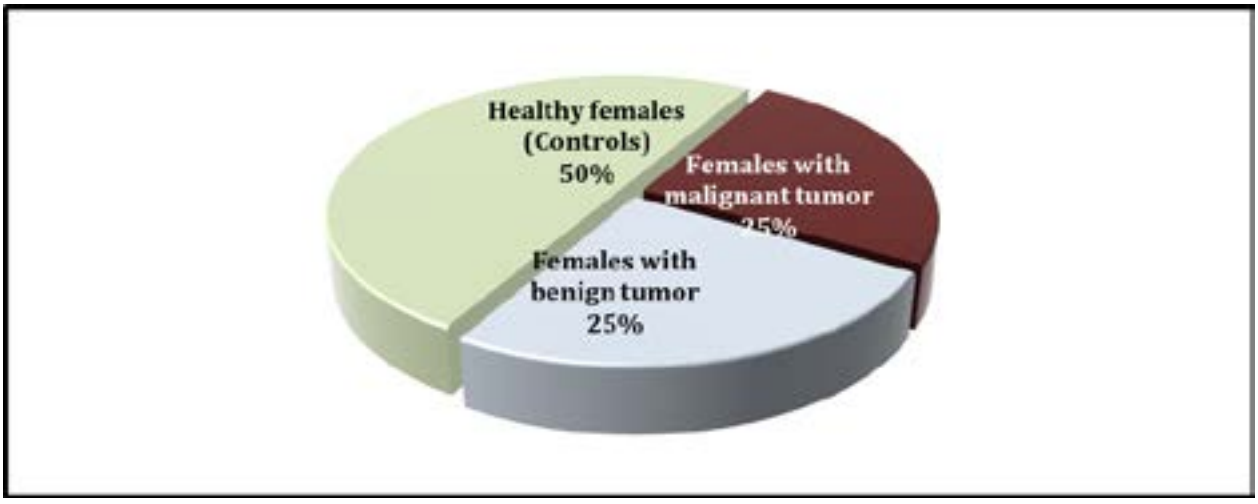

Figure 1. Classification of females enrolled in the present study

Table 1. Comparison of female's age, CRP and lipid profile between the studied groups

Parameters	Malignant tumors group N.=40	Benign tumors group N.=40	Control Group N.=80	p value
Age (years)	38.95 ± 0.47 ac, ab	35.38 ± 0.96 cc	34.64 ± 0.75 c	0.001 V S
CRP (mg/l)	43.91 ± 3.06 ac, ab	6.28 ± 0.55 cc	5.19 ± 0.31 c	< 0.001 V S
Serum cholesterol (mg/dl)	187.8 ± 7.97 cc	194.8 ± 5.23 cc	186.7 ± 2.46 c	0.444 V NS
Serum triglycerides (mg/dl)	164.7 ± 9.62 cc	165.3 ± 8.84 cc	167.5 ± 2.73 c	0.938 V NS
HDL (mg/dl)	40.60 ± 1.39 cc	39.83 ± 0.92 cc	40.79 ± 0.69 c	0.765 V NS
VLDL (mg/dl)	32..93 ± 1.92 cc	33.05 ± 1.77 cc	33.24 ± 0.55 c	0.938 V NS
LDL (mg/dl)	114.2 ± 7.48 cc	121.9 ± 4.59 cc	112.4 ± 2.66 c	0.311 V NS

V: Analysis of variance; S: Significant ($p \leq 0.05$); NS: Not significant ($p > 0.05$); a: Malignant tumors group; b: Benign tumors group; c: Control group

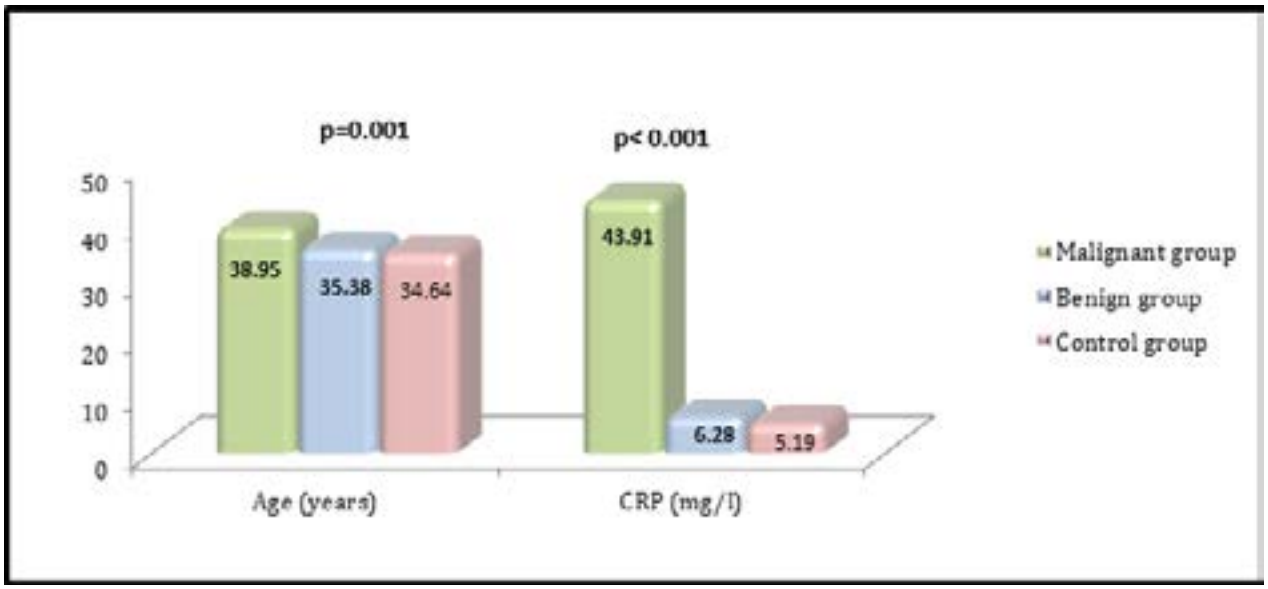

Figure 2. Comparison of mean age and CRP levels between the studied groups

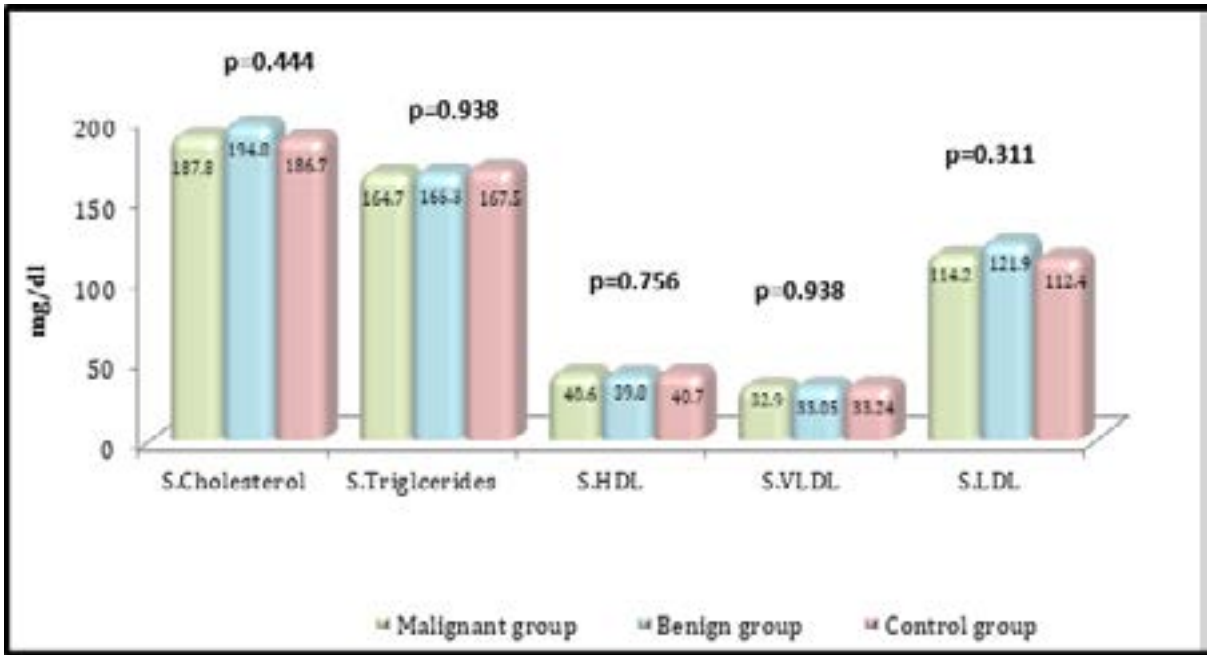

Figure 3. Comparison of lipids profile between the studied groups

in the malignant group (43.91 ± 3.06 mg/L) relative to both the benign group (6.28 ± 0.55 mg/L) and controls (5.19 ± 0.31 mg/L), indicating a strong systemic inflammatory response in malignant conditions ($p < 0.001$). These findings suggest that both increasing age and elevated CRP levels may be associated with breast malignancy. In contrast, the comparison of lipid profiles, including serum cholesterol, triglycerides, HDL, VLDL, and LDL, showed no statistically significant differences among the malignant, benign, and control groups ($p > 0.05$ for all parameters). This indicates that while lipid profile alterations are commonly studied in relation to cancer, they may not serve as reliable markers for distinguishing between malignant and benign breast tumors in this sample.

Biochemical marker analysis revealed significantly elevated levels of both mannan binding lectin (MBL) and endothelin converting enzyme (ECE) activity in the malignant group and the results were found in Table 2 and Figure 4. MBL levels in malignant patients (221.03 ± 23.31 ng/mL) were substantially higher compared to the benign group (160.6 ± 21.86 ng/mL) and controls (81.82 ± 6.41 ng/mL), with a p-value < 0.001. Similarly, ECE activity was highest in the malignant group (62.57 ± 8.34 U/L), followed by the benign group (28.12 ± 3.51 U/L), and was lowest in controls (8.54 ± 1.56 U/L), also with a p-value < 0.001. These results suggest that both MBL and ECE are strongly associated with malignancy and may play roles in tumor-related immune and vascular changes.

The Pearson's correlation analysis was done for two patients group among study variables and list in Table 3 and Table 4. Correlation analyses within the malignant and benign groups showed no significant associations between MBL or ECE and age, CRP, or lipid parameters, indicating that the elevation of these biomarkers is likely independent of these variables. However, a significant positive correlation was found between MBL and ECE levels within both groups ($r = 0.373$, $p = 0.018$ in malignant; $r = 0.495$, $p = 0.001$ in benign), suggesting a potential mechanistic or regulatory link between the two biomarkers.

Receiver operating characteristic (ROC) curve analysis was used to evaluate the diagnostic performance of MBL and ECE in distinguishing malignant tumors. ECE demonstrated a higher area under the curve (AUC = 0.773) compared to MBL (AUC = 0.717), indicating better overall diagnostic accuracy. ECE also showed superior specificity (93.1%) and sensitivity (72.8%) compared to MBL (specificity 89.7%, sensitivity 67.2%). These results support the potential clinical utility of ECE, and to a lesser extent MBL, as non-invasive serum biomarkers for identifying malignant breast tumors. The results of ROC curve and AUC showed in Table 5 and Figure 5.

Table 2. Comparison of biochemical markers between the studied groups

Parameters	Malignant tumor group N.=40	Benign tumors group N.=40	Control Group N.=80	p value
Mannan-Binding Lectin	221.03 ± 23.31 ac	160.6 ± 21.86 bc	81.82 ± 6.41 c	< 0.001 V S
Endothelin converting enzyme activity	62.57 ± 8.34 ac, ab	28.12 ± 3.51 bc	8.54 ± 1.56 c	< 0.001 V S

V: Analysis of variance; S: Significant (p ≤ 0.05); NS: Not significant (p > 0.05); a: Malignant tumors group; b: Benign tumors group; c: Control group

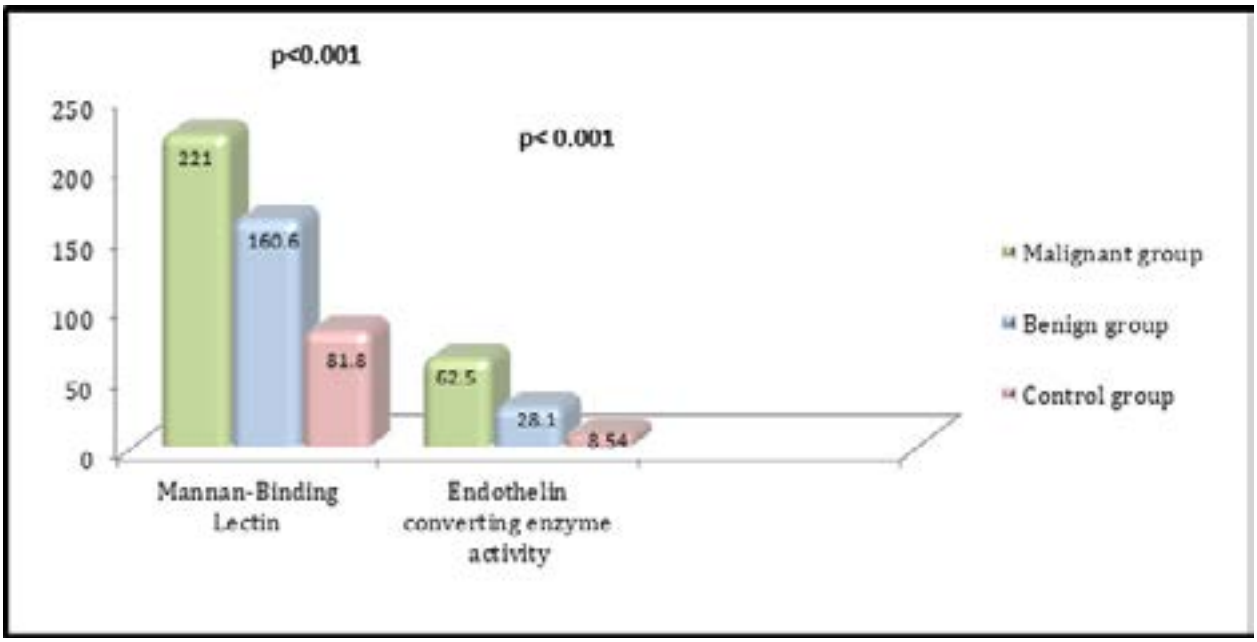

Figure 4. Comparison of biochemical markers between the studied groups

Table 3. Correlations between serum biomarkers with female's age, CRP and lipid profile among malignant group

Parameters	Statistics	Mannan- Binding lectin	Endothelin converting enzyme
Age	r	0.235	0.265
	p	0.144 NS	0.099 NS
CRP	r	0.165	-0.019
	p	0.308 NS	0.909 NS
Serum cholesterol	r	0.278	0.265
	p	0.116 NS	0.099 NS
Serum triglycerides	r	0.101	0.213
	p	0.301 NS	0.187 NS
Serum HDL	r	-0.282	-0.225
	p	0.078 NS	0.162 NS
Serum VLDL	r	- 0.096	0.213
	p	0.378 NS	0.187 NS
Serum LDL	r	- 0.096	0.269
	p	0.378 NS	0.093 NS
Mannan- Binding lectin	r	1.0	0.373*
	p	-	0.018 S
Endothelin converting enzyme	r	0.373*	1.0
	p	0.018 S	-

r: Pearson's correlation coefficient; NS: Not significant (p > 0.05)

Table 4. Correlations between serum biomarkers with female's age, CRP and lipid profile among benign group

Parameters	Statistics	Mannan binding lectin	Endothelin converting enzyme
Age	r	0.247	0.271
	p	0.125 NS	0.090 NS
CRP	r	0.132	-0.028
	p	0.417 NS	0.863 NS
Serum cholesterol	r	0.058	0.133
	p	0.723 NS	0.413 NS
Serum triglycerides	r	0.187	0.218
	p	0.247 NS	0.176 NS
Serum HDL	r	-0.159	-0.124
	p	0.327 NS	0.446 NS
Serum VLDL	r	0.187	0.218
	p	0.247 NS	0.176 NS
Serum LDL	r	0.026	0.092
	p	0.874 NS	0.571 NS
Mannan- Binding lectin	r	1.0	0.495**
	p	-	0.001 S
Endothelin converting enzyme	r	0.495**	1.0
	p	0.001 S	-

r: Pearson's correlation coefficient; NS: Not significant (p > 0.05)

Table 5. ROC characteristics of serum endothelin converting enzyme activity and Mannan binding lectin as a predictor of malignant and benign breast tumor

Characteristics	Endothelin converting enzyme	Mannan binding lectin
Cut off value	24.05	134.16
Area under curve (AUC)	0.773	0.717
Sensitivity %	72.80%	67.20%
Specificity %	93.10%	89.70%
***p* value**	0.0001	0.001

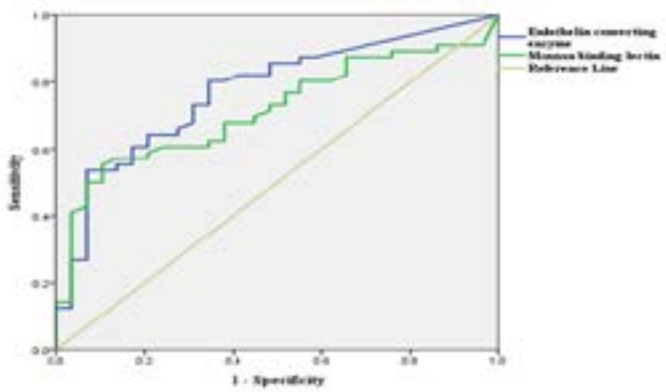

Figure 5. ROC curve for Endothelin converting enzyme and Mannan binding lectin between patients (benign and malignant) and controls

DISCUSSION

Evaluated 2,910 women with breast cancer and found higher pre-treatment CRP levels were significantly associated with increased risk of breast cancer-specific mortality, overall mortality, and distant recurrence[12]. Meta-analysis found that elevated CRP levels were significantly associated with worse overall survival (OS) and disease-free survival (DFS) in breast cancer[13]. In a case-control study, women with breast cancer had significantly lower levels of HDL-C compared to healthy controls. Suggests a protective role of HDL-C and potential involvement of lipid metabolism in cancer pathogenesis[14]. Analyzed the association between total cholesterol, HDL, LDL, triglycerides and breast cancer. Reported a possible inverse relationship between HDL-C and breast cancer risk, and a positive association between triglycerides and breast cancer in some subgroups[15]. Investigated lipid levels in different breast cancer subtypes. Found that triple-negative breast cancer patients had higher total cholesterol and LDL-C compared to other subtypes, indicating a possible link with tumor aggressiveness[16]. Although there are studies proving the effect of lipid levels on breast cancer patients, the current study did not demonstrate any significant change in lipid levels in breast cancer (benign and malignant) patients. The results of this study reveal a significant elevation in serum Endothelin-Converting Enzyme-1 (ECE-1) levels among women diagnosed with Invasive Ductal Carcinoma (IDC) compared to those with benign breast disease (BBD) and healthy controls[17]. This finding supports the growing body of evidence that implicates ECE-1 as an active molecular participant in breast tumor genesis[18]. Given that ECE-1 catalyzes the conversion of big endothelin-1 into its biologically active form, ET-1, its overexpression is functionally tied to increased vasoconstriction, angiogenesis, and cell proliferation—key hallmarks of cancer progression[19].

The markedly higher serum ECE-1 levels in IDC patients align with previous immunohistochemical and molecular studies which demonstrated ECE-1 up-regulation in high-grade and metastatic breast tumors[20]. This overexpression enhances tumor aggressiveness not only by promoting neovascularization but also by supporting cellular escape from apoptosis and immune surveillance[21]. Importantly, the data indicate that ECE-1 can distinguish malignant from benign lesions with a moderate diagnostic performance (AUC = 0.650), making it a potentially useful adjunct marker in diagnostic workflows, especially when radiological findings are inconclusive[22].

Despite this, the modest sensitivity and specificity values (62.5% and 67.5%, respectively) suggest that ECE-1 alone may not be sufficient for definitive diagnosis. This is consistent with the view that no single biomarker, especially one related to a pleiotropic pathway such as the endothelin system, can fully reflect the complexity of breast cancer pathophysiology[23]. However, the magnitude of difference in ECE-1 levels between IDC and control groups indicates that it could serve as part of a multi-marker diagnostic panel perhaps alongside angiogenic or immune markers to improve early detection rates.

The partial overlap in ECE-1 levels between IDC and BBD cases also warrants attention. It suggests that while ECE-1 is predominantly associated with malignancy, some benign proliferative lesions may exhibit elevated activity due to localized inflammation or stromal remodeling. This echoes findings in endometrial and ovarian neoplasms, where elevated ECE-1 was also seen in certain pre-malignant conditions[22].

Moreover, ECE-1's role may not be limited to tumor progression but could extend to therapeutic resistance. Recent in vitro evidence has shown that ET-1 signaling, enhanced by ECE-1, activates anti-apoptotic cascades and interferes with chemotherapeutic efficacy[24]. If serum ECE-1 levels reflect this intratumoral activity, they may eventually serve not only for diagnosis but also for treatment monitoring or prognosis.

Investigated serum MBL levels in women with breast cancer compared to healthy controls found that low MBL levels were significantly more frequent in breast cancer patients. Suggests that MBL deficiency may impair innate immunity and potentially contribute to tumor development or progression[25]. Compared levels of lectin pathway components (including MBL, MASP-2, ficolins) between healthy controls and cancer patients (including breast cancer). found altered MBL and MASP-2 levels in cancer patients, indicating dysregulation of the complement system. Suggests a role for MBL in cancer immune surveillance and possibly in modulating tumor microenvironment[26]. Explored MBL2 gene polymorphisms and their influence on cancer risk. Certain polymorphisms leading to low MBL expression were associated with greater susceptibility to breast and other cancers, suggesting genetic variation in MBL may influence immune response to tumors[27]. Both MBL and ECE/ET-1 influence the tumor immune landscape. Low MBL levels may impair complement-mediated tumor cell lysis and allow immune evasion. High ECE/ET-1 activity promotes a pro-tumorigenic microenvironment, including angiogenesis and macrophage recruitment[28,29].

CONCLUSION

This study demonstrates that serum levels of Endothelin-Converting Enzyme-1 (ECE-1) and Mannan-Binding Lectin are significantly elevated in patients with Invasive Ductal Carcinoma (IDC) compared to both benign breast disease cases and healthy individuals. These findings underscore the potential role of ECE-1 and Mannan-Binding Lectin as a non-invasive biomarker for the early detection of breast cancer malignancy.

Authorship Contribution: All authors share equal effort contribution towards (1) substantial contributions to conception and design,

acquisition, analysis and interpretation of data; (2) drafting the article and revising it critically for important intellectual content; and (3) final approval of the manuscript version to be published. Yes.

Potential Conflicts of Interest: None

Competing Interest: None

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