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Evaluation of serum levels of visfatin and resistin alongside other biochemical indicators in breast cancer

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Abstract

Background and objective: One important modifiable risk factor for breast cancer is obesity. The pathophysiology and advancement of breast cancer are facilitated by the release of chemicals from obese adipose tissue, including proinflammatory and adipocytokines. The two adipocytokines that are the subject of this study are Resistin and Visfatin. To compare breast cancer patients' blood and tissue levels of Resistin, Visfatin, and other biochemical markers to those of healthy controls.

Methods: a case-control study involving breast cancer patients and healthy individuals. The study included 30 women patients with breast cancer before treatment, the same number (30) of women with breast cancer on treatment, and also 30 women in a healthy group, totally collected 90 female blood sample from patients that diagnosed with a breast cancer and healthy control, the level of serum Visfatin and Resistin with biochemical parameters was performed for all participants.

Results: The mean ± SE Visfatin levels in the patients (pre-treatment and post-treatment) were elevated (4.44 ± 0.807, 7.79 ± 1.802) relative to the control (3.53 ± 0.214). With a P-value of 0.025. There was a significant difference in the mean ± SE of serum Resistin levels between the cases and the control group with (P = 0.013).

Conclusion: According to our study, women with breast cancer had significantly greater levels of Visfatin and Resistin than those in good health. This implies that the genesis and progression of breast cancer may be influenced by these adipocytokines**.**

Keywords: Breast cancer, Lipid profile, Resistin, Visfatin.

Introduction

Breast cancer is the most prevalent malignancy among women globally, accounting for the majority of cancer cases in both developing and developed countries. Breast cancer is an illness characterized by a range of symptoms and characteristics. Specific genetic defects in breast epithelial cells give rise to molecular traits, which each person experiences in various clinical manifestations (1).

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The uncontrolled development or multiplication of cells inside the breast might result in a condition referred to as breast disease. Oncology The anatomical site of origin often determines the designation. Breast cancer refers to the situation in which breast cells proliferate beyond their typical boundaries. Numerous risk factors may facilitate the onset of breast cancer. The classification of a patient's breast cancer will be based on the specific kinds of cells affected by the tumor. Cancer may develop in any region of the breast. The three primary anatomical components of the breasts are lobules, ducts, and soft tissue. The lobules produce milk, which the ducts then transport to the nipple. Connective tissue either envelops or unites all structures. Breast cancer often originates in the lobules and disseminates via the ducts. As stated in, arteries facilitate the dissemination of cancer to other organs (2). Breast cancer is believed to develop due to a multitude of factors. Breast cancer is thought to be caused by a combination of lifestyle factors and environmental ones. These include exposure to chemicals and radiation, being overweight or inactive, eating a lot of fat, smoking, having diabetes or high blood pressure, not breastfeeding or breastfeeding for a short time, having irregular periods, and using birth control. Experts believe that inherited defective genes initiate breast cancer, while a familial history of the illness exacerbates its progression (3).

Adipose tissue mostly produces adipocytokines, a specific subclass of cytokines, albeit not exclusively. It is an adipocytokine that works as both an outside secretory factor and an inside enzyme in mammalian cells. It is located in visceral adipose tissue. It has a pivotal function in inflammatory cytokine activity and considerably contributes to cellular energy and stress responses. It significantly contributes to the promotion of inflammation via the synthesis of Visfatin. Macrophages and adipocytes secrete it inside adipose tissue (1).

Visfatin, also known as preB cell colony-enhancing factor, is a new adipokine that has been found in visceral fat. Visfatin is an NAMPT (nicotinamide phosphoribosyl transferase) that helps cells use energy. It also takes part in a number of metabolic and stress responses (4). Research indicated markedly elevated blood levels of Visfatin in individuals with different types of breast cancer, suggesting its possible role in the illness. The breast cancer stages were elevated in comparison to the benign and control groups. Moreover, Visfatin levels escalated in tandem with the tumor's growth throughout time (5).

Resistin is an adipokine composed of 108 amino acids and contains a significant amount of cysteine. The RETN gene, located on chromosome

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19 (19p13.2), produces Resistin, a discovery in 2001. Resistin is involved in glucose and insulin metabolic pathways, as its name suggests a connection to insulin resistance. Also, when its receptor TLR4, which is part of the Toll-Like Receptor family, is activated, it happens in pathways that promote inflammation (NF-κB, PI3K). Resistin is released by both adipocytes and inflammatory cells (6). A significant correlation between elevated levels of Resistin and the risk of breast cancer was observed, regardless of age, body mass index, menopausal status, serum glucose, and adiponectin levels (4). Lipid metabolism disorders are often associated with a higher prevalence of neoplastic diseases. Epidemiological studies demonstrate that the lipid profile is a crucial determinant in breast cancer. Lipoprotein and cholesterol levels have been linked to an increased risk of breast cancer (7).

Methodology and data collection

A case-control study aims to investigate Visfatin, Resistin, and various biochemical parameters, including lipid profile, glucose, and insulin, in participants with breast cancer compared to those without. A total of 90 female blood samples were collected from patients diagnosed with breast cancer and healthy controls. The samples were categorized into Group I (30 apparently healthy controls), Group II (30 pre-treatment patients), and Group III (30 post-treatment patients), aged between 26 and 67. The study population was selected from Rizgary Teaching Hospital and Nanakali Hospital, while healthy controls were randomly selected volunteers with no evidence of any disease.

We obtained written informed permission from both patients and control individuals once they agreed to participate. We used comprehensive questionnaires to gather medical information for all individuals. We acquired a comprehensive history, which encompassed menstrual cycle, lifestyle habits, medical history, and family history of breast cancer and other malignancies. We collected questionnaire data and specimens prior to the initiation of radio- or chemotherapy, minimizing the potential impact of treatment. All participants, including cases and controls, were thoroughly apprised of the study's objective and provided signed permission for their involvement.

Collection of Specimen

Blood samples are taken from all participants using a disposable syringe; 5 ml of blood is aseptically extracted from each person and maintained at room temperature for 15 minutes. Centrifugation at 5000 rpm for 10 minutes isolates the serum, which we then promptly test. After that, the above parameters (Visfatin and Resistin) were measured with an enzyme-linked immunosorbent assay (ELISA), and

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the lipid profile was checked with a spectrophotometer.

Statistical Analysis

The data analysis was conducted using SPSS software (version 25). All descriptive data were reported as the mean ± standard error of the mean (SEM) for the selected variable. The chi-square test was used to evaluate association between categorical variables between groups. An independent t-test was used to compare numerical data, including BMI and age, between the control and case groups. A P-value below 0.05 was deemed statistically significant, but values beyond 0.05 were regarded as not significant.

Results

Demographic and Social characteristics of participants group: Results have been observed that demographic and social characteristics of the participant group, including Mstatus, Residency, Smoking, Activity, and Age there were no statistically noteworthy differences between groups with P-values of (0.07, 0.17, 0.2, 0.47, 0.6) respectively. In relation to the other risk factors among the control and patient groups of the study population, an analysis of the differences in risk factors between patients and healthy individuals using the chi-square and t-test revealed statistically significant disparities among the groups for most risk factors, including occupation, family history, menstrual cycle, BMI, and another clinical disorder with P-values of 0.004, 0.03, 0.03 0.037, 0.01, respectively. The mean ± SE of body mass index (BMI) for the control group was 27.5±0.75, whereas the patient group value was 33.1±0.77. This information is presented in Table 1.

Serum levels of Visfatin, and Resistin in the studied groups:

The mean ± SE Visfatin levels in the patients (pre-treatment and post-treatment) were elevated (4.44 ± 0.807, 7.79 ± 1.802) relative to the control (3.53 ± 0.214). This variation was statistically significant with a p-value of 0.025. There was a significant difference in the mean ± SE of serum Resistin levels between the cases and the control group. Unsurprisingly, the cases (pre-treatment and post-treatment) had a higher Resistin level (1.52 ± 0.091, 2.38 ± 0.434), respectively, compared to the control (1.35 ± 0.076). This improvement was also significant (P = 0.013). Both Visfatin and Resistin show a significant increase after treatment (post-treatment) compared to the control and pre-treatment groups, as indicated by the P-values less than 0.05. This information can be seen summarized in (Table 2).

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Table 1. Demographic and Social characteristics of participants group

Parameter		Control Group n=30 No. (%)	Patients' group n=60 No. (%)	Test	value	P-value
Marital status	Married	25 (83.3)	57 (95)	Chi-Square	3.4	0.07
	single	5 (16.7)	3 (5)			
Residency	City	9 (30)	27 (45)	Chi-Square	1.9	0.17
	countryside	21 (70)	33 (55)			
Occupation	Employed	12 (40)	8 (13.3)	Chi-Square	8.2	0.004*
	Unemployed	18 (60)	52 (86.7)			
Smoking	Non-smoker	30 (100)	57 (95)	Chi-Square	1.55	0.2
	Ex-smoker	0 (0.0)	3 (5)			
Family history	Yes	6 (20)	26 (43.3)	Chi-Square	4.6	0.03*
	No	24 (80)	34 (56.7)			
Menstrual cycle	postmenopausal	14 (46.7)	42 (70)	Chi-Square	4.6	0.03*
	premenopausal	16 (53.3)	18 (30)			
Exercise	Yes	8 (26.3)	12 (20)	Chi-Square	0.51	0.47
	No	22 (73.3)	48 (80)			
BMI		27.5±0.75	33.1±0.77	T -test	4.5	0.037*
Age		44± 1.6	46.5±1.14	T -test	0.26	0.6
Another clinical disease	Diabetes	0 (0.0%)	10 (16.7)	Chi-Square	12.9	0.01*
	Hypertension	0 (0.0%)	6 (10)			
	Heart disease	0 (0.0%)	3 (5)			
	Other disease	0 (0.0%)	1 (1.7)			
	None	30 (100)	40 (66.7)			

*. Significant difference

Table 2. Chemical parameters of serum Visfatin and Resistin in the studied groups

Parameters	Control (Mean ± SE)	Pre-treatment (Mean ± SE)	Post-treatment (Mean ± SE)	F value	P value
Visfatin (ng/ml)	3.53 ± 0.214	4.44 ± 0.807	7.79 ± 1.802	2.79	0.025*
Resistin (ng/ml)	1.35 ± 0.076	1.52 ± 0.091	2.38 ± 0.434	2.73	0.013*

*. Significant difference

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Serum level of Cholesterol, TG, HDL, LDL, VLDL, AIP, CRP of the studied group

As below (Table 3) healthy control and patients of breast cancer were further analyzed according to some other important serum biomarker cholesterol, triglyceride, HDL, LDL, VLDL, API, LDL/HDL ratio, CRP. The results revealed that they were not statistic significant in cholesterol, HDL, LDL, LDL/HDL Ratio, CRP, And P-value were (0.17, 0.29,0.24, 0.08,0.09) respectively, on another hand (triglyceride, VLDL, API) were significant with P-value (0.01, 0.01, 0.011) respectively.

Table 3. Serum level of Cholesterol, TG, HDL, LDL, VLDL, AIP, CRP of the studied group

Parameters	Control Mean ± SE	Pre-treatment Mean ± SE	Post-treatment Mean ± SE	F-value	P-value
Cholesterol (mg/dl)	149.7 ± 5.855	162.2 ± 6.706	168.6 ± 8.379	1.84	0.17
TG (mg/dl)	112.7 ± 9.75	125.1 ±11.5	166.1± 16.3	4.76	0.011*
HDL (mg/dl)	44.5 ± 1.59	43.2 ± 2.06	40.0 ±2.35	1.27	0.29
LDL (mg/dl)	82.9 ± 5.35	93.9 ± 6.13	95.3 ±5.45	1.45	0.24
VLDL (mg/dl)	22.535 ± 1.949	25.022 ± 2.305	33.2 ± 3.26	4.76	0.011*
API (mg/dl)	0.36 ± 0.049	0.420 ± 0.054	0.58 ± 0.05	4.78	0.011*
LDL/HDL ratio	1.95 ± 0.15	2.35 ± 0.21	2.45 ± 0.12	2.63	0.08
CRP (mg/l)	3.74 ± 0.91	5.63 ± 1.18	8.20 ±1.94	2.51	0.09

*. Significant difference

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Correlation between Serum level of Visfatin and Resistin with BMI, Glucose, API, in patients' group

A Pearson correlation analysis was done to examine the relationship between visfatin (ng/ml) levels and various clinical factors, such as BMI, high blood sugar, and Atherogenic Index of Plasma (API) in the patient group. Of the characteristics evaluated, only a weak positive correlation was observed between serum visfatin and glucose (r = 0.277, P = 0.03), exhibited a statistically significant connection. No notable correlations were detected between visfatin and BMI (r = 0.023, P = 0.86) or API (r = 0.086, P = 0.51). And also no significant correlation were detected between Resistin with BMI (r = 0.062, P = 0.64), glucose (r = 0.077, P = 0.56), or API (r = -0.008, P = 0.95). All correlations were weak to negligible in strength. This information is referenced in Table 4 and 5.

Table 4. Correlation between serum level of Visfatin with BMI, Glucose, API, in patients' group represented by correlation coefficient (r) and P-value

Parameters	Visfatin (ng/ml)	
	r	P-value
BMI	0.023	0.86
Glucose	0.277**	0.03*
API	0.086	0.51

*Significant difference at P <0.05 **Correlation is significant at the 0.01 level.

Table 5. Correlation between serum level of Resistin with BMI, Glucose, API, in patients' group represented by correlation coefficient (r) and P-value

Parameters	Resistin (ng/ml)	
	r	P-value
BMI	0.062	0.64
Glucose	0.077	0.56
API	-0.008	0.95

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Discussion

When cells in the breast start to grow uncontrollably, it can lead to what's known as breast disease. If this growth is cancerous, it's typically named based on where it starts. In the case of breast cancer, it means the cells in the breast are multiplying and spreading beyond their usual limits (2).

This research examined the concentrations of two molecules, Resistin and Visfatin, in the blood of breast cancer patients relative to healthy individuals. Visfatin, which is also called nicotinamide phosphoribosyl transferase (Nampt) or pre-B-cell colony-enhancing factor (PBEF), is a protein made by fat tissue near the organs. It is a distinctive molecule with three functions: it serves as a growth factor, an enzyme, and a cytokine (8).

We demonstrate that both Resistin and Visfatin were significantly higher in those with breast cancer, suggesting these molecules could be involved in how the disease develops and progresses. The present findings corroborate the previous study's results, which shown that serum Visfatin levels were considerably elevated in breast cancer patients relative to healthy controls (1).

Resistin, which is a pro-inflammatory adipokine also called adipose-tissue-specific secretory factor (ADSF), is made up of 108 amino acids and is produced in fat cells, pancreatic cells, muscle tissue, and some blood cells (9).

The findings indicated a considerably elevated level in the breast cancer patient group relative to the controls. This conclusion aligns with the prior study's findings, which statistically (P-value ≤0.05) identified a significant correlation between increased Resistin levels and breast cancer risk, independent of body mass index, menopausal status, age, serum glucose, and adiponectin levels (4).

The data analysis in this study demonstrated that statistically significant differences were observed between the patient and control groups regarding several established risk factors, including family history of cancer and menstrual cycle. The result is in accordance with a previous study that has shown menopause had a 6.43-fold risk for having breast cancer compared to menstruating women, and family history of a first-degree relative showed increased risk compared to women without family history of breast cancer (10).

This study demonstrates a significant disparity in BMI between breast cancer patients and healthy controls. This result supports the findings of a prior study, which indicated that women with a high BMI faced a markedly increased risk of breast cancer relative to those with a normal BMI. Research found that for every 5 kg/m² increase in BMI, the

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probability of having breast cancer rose by 31% (11).

Our results revealed significantly elevated mean levels of blood triglycerides and very-low-density lipoprotein, together with reduced high-density lipoprotein in all breast cancer patients compared to healthy controls. Cholesterol and LDL readings were elevated, albeit not to a statistically significant degree. Prior research indicated that cholesterol, triglycerides, LDL-C, and VLDL levels are elevated in breast cancer patients, suggesting a correlation between increasing cholesterol levels and breast cancer incidence. A correlation exists between cellular proliferation and cholesterol production. Breast cancer stimulates an increase in cholesterol due to its activation of tumor proliferation and metastasis. It has been shown that elevated LDL levels augment the risk of breast cancer. HDL-C did not exhibit any significant differences between cancer patients and controls; HDL limits the absorption of LDL-C from arterial walls and enhances the transfer of cholesterol from peripheral tissues to the liver (12).

The statistical study revealed a substantial positive association between Visfatin and glucose, as shown by a P-value of (≤0.05) in serum breast cancer patients. The latest findings align with the previous study's results, which demonstrated a substantial positive association between serum Visfatin levels and glucose (4).

The Visfatin connection showed that Visfatin was positively correlated with both API and BMI. There was no statistically significant relationship between serum Visfatin levels and either API or BMI (P-value >0.05). This finding is consistent with other studies showing no meaningful correlation between serum Visfatin and body mass index (BMI) (13).

The connection between Resistin and BMI showed a positive association. No statistically significant link was detected between serum Resistin levels and BMI (P-value >0.05). Consistent with our investigation, no significant correlation was seen between serum Resistin and BMI (13).

Additionally, this research discovered a favorable association between glucose and Resistin. Serum Resistin level and glucose did not show a statistically significant connection (P-value > 0.05). The new study's connection finding contradicts earlier research that found a positive link between insulin resistance and Resistin (14).

Conclusion

The results indicated a substantial increase in serum Visfatin and Resistin levels in breast cancer patients compared to the control group. Our findings indicated that serum Resistin and Visfatin levels may serve as risk factors for breast cancer.

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Competing interest

The authors declare that they have no competing interests.

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