Comprehensive Biochemical Profiling of Key Serum Biomarkers in Breast Cancer Patients: Pre- and Post-Mastectomy Comparative Analysis

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ABSTRACT
Breast cancer is, undoubtedly, the leading cause of cancer-related human casualities among females, making early detection and accurate diagnosis increasingly essential for improving outcomes. Molecular advancements have aided in identifying biomarkers that inform therapy and prognosis. This article reports on blood samples taken from 70 females before and after having surgical tumor removal, along with 20 samples from a control group. Many biomarker enzymes were analyzed, including H-Interferon (IFN-γ), Mammaglobin-A (SCGB2A2), Indoleamine-2,3-deoxygenase (IDO), Amyloid Beta peptide (Aβ), Acetylcholine carboxylase 1 (ACC1), Selenium binding protein (SELENBP1), and Acetylcholine Esterase (AChE). The findings indicated that Mammaglobin-A (SCGB2A2) levels increased before surgery and decreased afterward, emphasizing its diagnostic significance and the effectiveness of tumor removal. Indoleamine-2,3-deoxygenase (IDO) and Amyloid Beta peptide (Aβ) levels were elevated prior to surgery, associated with immune evasion and tumor aggressiveness, but showed a decline post-surgery. Acetyl-CoA Carboxylase 1 (ACC1) and Selenium-binding Protein 1 (SELENBP1) indicated metabolic changes, while Interferon-gamma (IFN-γ) levels remained low, particularly in older patients, which may influence immune recovery. These findings significantly emphasize the importance of biomarkers in diagnosis and treatment.

Keywords: Breast Cancer, Mastectomy, Diagnostic Biomarker, Tumor aggressiveness, Prognostic Biomarker.

1. INTRODUCTION

Breast cancer, as commonly known, is one of the major cancers that threaten the lives of women worldwide. It is a very complex disease and its effects vary greatly. [1]. The basis of improving the therapeutic results as well as the survival in patients requires early diagnosis and accurate recognition. Development in molecular technologies has significantly improved our knowledge regarding the biology of breast cancer and thus identification of a number of biomarkers affecting the therapy decisions, diagnosis, and prognosis became possible.[2].

Three biomarkers categories for breast cancer can be presented here: genetic, protein-based, and epigenetic[3]. Such biomarkers have multifaceted uses, like diagnosing cancer, predicting the course of the illness, and measuringhow effective the treatment is. Hormone receptors, like the human epidermal growth factor receptor 2 (HER2)[4], progesterone receptor (PR)[5], and estrogen receptor (ER)[6], can beone of the most distinguished biomarkers. These receptors help determine the mode of treatment. The most critical biomarkers in breast cancer are genes and proteins whose expressions are different from that of malignant to normal tissues, hence assisting diagnosis and prognosis. [7]. Overexpression of CACNG4, EPYC, and PKMYT1 are associated with disease whereas CHRNA6 has diagnostic potentials [8]. Also, circulating miRNAs and DNA methylation patterns could add additional information on prognosis like MAST1 and PRDM14 [3]. Biomarkers at the protein level include TFFs and TK that are useful in the screening and monitoring of metastatic cancer [9]. Other biomarkers include Ki67, cfDNA, and MUC1, which contribute to diagnosis and prognosis by showing tumor aggressiveness and the possibility of recurrence. [10].

Many biomarkers, though in their infancy stage of studies, have been reported to hold a high degree of promise to advance diagnosis and treatment in breast cancer. Among them, H-Interferon or IFN-γ, a cytokine whose main function involves any form of tumor-targeting immune response, has been

associated with increased immune activities in patients with breast cancer [11]. Increased levels of this cytokine could be a useful indicator for tracking the effectiveness of treatment. Mammaglobin, which is mostly expressed in breast tissue, has also demonstrated potential as a diagnostic tool, especially when detected in serum samples to identify recurrence of breast cancer[12].Indoleamine-2,3-dioxygenase has been wedded to the capability of the immune system to escape the tumor cells. Tumor aggressiveness and higher IDO expression have been associated, indicating that IDO may be used as a predictor [13]. In a similar way, amyloid beta peptide, which traditionally has been associated with neurological diseases, its role in tumor biology is under investigation regarding its expression in breast cancer tissues.[14].Further, the role of acetyl-CoA carboxylase 1 in neurotransmitter metabolism in tumor growth and metastasis in breast cancer has also been discussed [15]. A component of antioxidant defense system, that is, selenium-binding protein, has equally been said to be differentially represented in a host of malignancies including breast cancer [16]. This is followed by acetylcholinesterase, which, besides the role of neurotransmission, has also been implicated in tumor development. The expression of AChE can thus become an important marker of tumor behavior and patient outcome..

The current research article outlines the activities of some of these biomarkers like H-Interferon, Mammaglobin-A, Indoleamine-2,3-deoxygenase, Amyloid Beta peptide, Acetylcholine carboxylase 1, Selenium binding protein, and Acetylcholine Esterase enzyme, and their relation with the latter in females before and after mastectomy.

2. MATERIALS AND METHODS

2.1. Patients and Blood Sample Collection

A. Control Group

Twenty female blood samples were selected at random to act as the study's control group for comparative analysis.

B. Patients Group

The study included blood samples that were obtained from people between the ages of 33 and 68. Samples were collected in the duration between February 2023, and May 2023. Patients' samples were obtained both before and after the procedure to remove the tumor. The samples included 70 individuals with breast cancer; 35 patients were taken before to surgery, and 35 patients who had their tumors removed and were receiving chemotherapy treatment dosages at different intervals were included in the sample set. The samples were collected after oncologists working with Al-Amal Cancer Hospital in Baghdad made an appropriate diagnosis.

C. Blood Sample Collection

Blood was drawn from the vein using a single-use syringe (5 ml) and placed into GEL tubes. After letting the blood sit for thirty to fifty minutes, serum was extracted using a centrifuge set to run at 5,000 rpm for five minutes. Subsequently, the clear serum was transferred into Eppendorf tubes and refrigerated.

2.2. General Principle of The Assay

The human ELISA kit is centred on a quantitative sandwich enzyme-linked immunosorbent assay (ELISA) method to measure the concentration of the enzyme in samples[17]. A monoclonal antibody specified for the enzyme is pre-coated onto the microplate wells. Samples or standards are added to the wells, followed by the addition of a biotin-labeled detection antibody specific for each enzyme. After the first incubation, the enzyme present in the samples binds to both the immobilized antibody and the detection antibody. Afterremoving unbound substances, Horseradish Peroxidase (HRP) is put for the next incubation. Following the process of washing, the substrate solution responds with HRP, and a color develops in proportion to the amount of the enzyme bound to the immobilized antibody. The development of color is halted by adding the acid, and the optical density is measured using a microplate reader. The table (1) below lists the materials provided in the kit.

Table 1. Kit components.

No.	Provided Kit Components	Determinations	Storage
1	Membrane/Closure plate	2	Room Temp.
2	Micro Elisa/Strip plates	1	2 to 8°C
3	Standard: 54 ng	0.5 x 1	2 to 8°C
4	Standard Diluent	1.5 x 1	2 to 8°C
5	Horseradish Conjugate	6 mL x 1	2 to 8°C
6	Sample Diluent	6 mL x 1	2 to 8°C

7	Chromogen A Solution	6 mL x 1	2 to 8°C
8	Chromogen B Solution	6 mL x 1	2 to 8°C
9	Stop Solution	6 mL x 1	2 to 8°C
10	Wash Solution	20 mL (30X) x 1	2 to 8°C

2.3. Procedures

The same ELISA method was employed for measuring the following variables: Interferon-γ (IFN-γ) activity, mammaglobin (SCGB2A2), indoleamine 2,3-dioxygenase (IDO), amyloid beta peptide (Aβ), acetyl-CoA carboxylase (ACC1), selenium-binding protein, and acetylcholinesterase (AChE). However, the antibody used in each measurement varied.

2.3.1. General Procedure

1. **Dilution of Standard Solutions:** The standard solution was first diluted using small test tubes. Then, 50 µL is drawn from each tube and transferred into the wells of the microplate. Each tube corresponds to one well, resulting in a total of five wells, as illustrated in Table (2) and Figure (1).

Table 2. Dilution of standard solutions and concentrations.

Dilution	Resulting	Conc.
300 µL of the original standard solution + 150 µL of the standard diluent	Standard 1	1800 pg/mL
300 µL of the standard solution 1 + 150 µL of the standard diluent	Standard 2	1200 pg/mL
150 µL of the standard solution 2 + 150 µL of the standard diluent	Standard 3	600 pg/mL
150 µL of the standard solution 3 + 150 µL of the standard diluent	Standard 4	300 pg/mL
150 µL of the standard solution 4 + 150 µL of the standard diluent	Standard 5	150 pg/mL

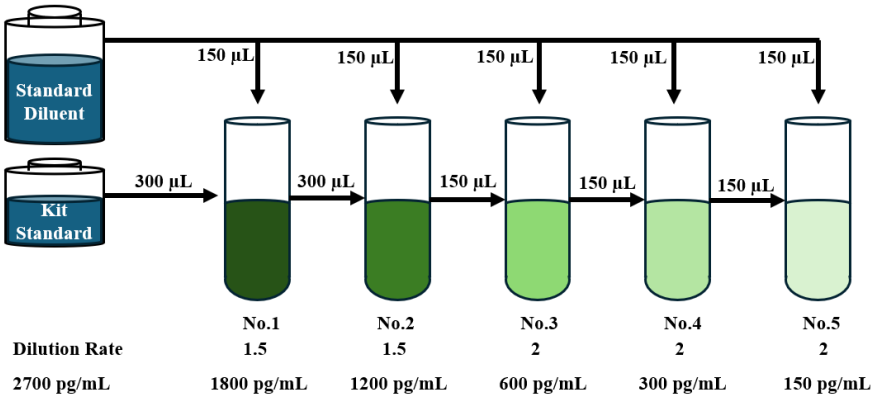

Figure 1. Dilution instructions.

2. **Sample Addition:**Blank wells left separate (no sample or HRP-Conjugate were added to blank wells). 40 µL of the diluted standard solution has been diluted into the test sample well, followed by the addition of 10 µL of the test sample (final dilution factor of the sample is x 5). The sample was added into the wells avoiding contact with the well walls as much as possible and mixed gently.
3. **Incubation:** the plate waswrappedwith a cover and incubated for 30 minutes at 37℃
4. **Dilution of Buffer:** The buffer solution is diluted (either 20x or 30x) with distilled water and stored.
5. **Washing:** The plate cover was removed, and the liquid was discarded, and dried by gently tapping. the washing buffer was added (350-400 µL or completely filled; overflow was acceptable) to each well, and sat for 30 seconds, then discard the liquid. the washing step was repeat 5 times and dried by blotting.
6. **Enzyme Addition:** 50 µL of the HRP-Conjugate reagent was put to every single well, except for the blank one.
7. **Incubation:**The same incubation procedure as in step 3 has been followed.
8. **Washing:**The washing procedure was performed as described in step 5.
9. **Color Development:** 50 µL of Chromogen A solution and 50 µL of Chromogen B solution were addedto every single well. The plate was protected from light and incubated for 10 minutes at 37℃
10. **Reaction Termination:** 50 µL of the stop solution was added to each well, stopping the reaction as the color changes from blue to yellow.

11. **Analysis:**The blank well has been used as the reference (zero) and the absorbance was read within 15 minutes at 450nm after adding the stop solution.

The briefed Procedure is depicted in Figure (2).

Figure 2. Briefed Procedure according to the kit manufacturers.

2.4. Precision, Test Range, and Sensitivity

Intra-assay Precision: Three samples with low, medium, and high levels of the studied enzyme were verified 20 times consecutively on a single plate.

Inter-assay Precision: Three samples with low, medium, and high levels of studied enzyme were verified on triplevarious plates, with eight replicates conducted on each plate.

$$CV\% = \frac{SD}{Mean} \text{ x } 100$$

Where; Intra-Assay: CV<10%, and Inter-Assay: CV<12%.

Test Range:30 pg/ml - 2000 pg/ml.

Sensitivity:5 pg/ml.

3. RESULTS AND DISCUSSIONS

3.1. Comparing the Biochemical Variables of Breast Cancer Patients with Healthy Controls Before and After Surgical Resection

Results show the statistical values of the clinical variables assessed in this paper from the serum of breast cancer patients and the control group represented as (Mean ± SD). Table (3) provides a summary of the results.

Table 3. Measured variable results for patients with breast cancer and the control group.

Variable	**Control Group n=20 Mean ± SD**	**Patients Pre- mastectomy n=35 Mean ± SD**	**Patients post-mastectomy n=35 Mean ± SD**
Interferon Activity (IFN- γ)	7.845 ± 0.418	4.889 ± 0.400	6.971 ± 0.622
Mammaglobin-A (SCGB2A2)	0.354 ± 0.011	0.532 ± 0.056	0.366 ± 0.037
Indoleamine-2,3-deoxygenase (IDO)	78.433 ± 2.691	88.312 ± 5.072	74.104 ± 8.466
Amyloid Beta Peptide (Aβ	73.908 ± 2.180	83.196 ± 5.945	72.907 ± 3.932
Acetylcholine Carboxylase (ACC1)	116.924 ± 4.551	167.489 ± 1.899	131.574 ± 3.162
Selenium Binding Protein 1 (SELEBP1)	212.603 ± 2.504	162.404 ± 17.811	201.330 ± 4.572
Acetylcholine Esterase (AChE)	5.058 ± 0.174	3.055 ± 0.228	4.108 ± 0.326

In general, table (3) shows that, both before and after a mastectomy, the levels of interferon-gamma (IFN-γ) in patients with breast cancer were considerably lower than in healthy individuals. The values were 4.889 ± 0.403, 6.971 ± 0.622, and 7.845 ± 0.418, respectively. In patients undergoing pre-operative care

and those with stage III breast cancer, there was a significant decrease in IFN-γ, which was correlated with more advanced tumor stages. These results are consistent with other research, such as a 396-patient analysis that connected decreased IFN-γ levels to the development of tumors. [18].In comparison to healthy controls (0.011 ± 0.354), Table (3) demonstrates a large increase in mammaglobin (SCGB2A2) levels in pre-mastectomy patients (0.056 ± 0.532), followed by a slight increase in post-mastectomy patients (0.037 ± 0.366) and a notable drop following surgery. The predictive value of mammography can be enhanced by early identification, especially in high-risk individuals, thanks to the higher levels of mammaglobin, a marker specific to breast cancer [19]. It is a useful biomarker for prognosis and diagnosis because it is also connected to high-grade and metastatic cancers [20]. Research validates that following surgery, mammaglobin levels drop, signifying the tumor's excision and the efficacy of the treatment [21].The results in Table (3) have also demonstrate that, in comparison to healthy individuals (78.433 ± 2.691), breast cancer patients had higher levels of Indoleamine 2,3-Dioxygenase (IDO) prior to mastectomy (88.312 ± 5.072) and lower levels following surgery (74.104 ± 8.466). Through the recrutiment of regulatory T cells (T-regs), the suppression of local immunological responses, and the promotion of tumor dissemination, elevated IDO levels are associated with both tumor growth and immune evasion. Elevated levels of IDO have been linked to unfavorable survival results, especially in cases of basal-like breast cancer. Additionally, these levels are more prevalent in tumors that are estrogen receptor-positive (ER+), which may have an impact on treatment outcomes [22].According to Table (3), compared to healthy individuals (73.908 ± 2.180), the levels of Amyloid Beta Peptide (Aβ) are higher in pre-mastectomy patients (83.196 ± 5.945) and lower in post-mastectomy patients (72.907 ± 3.932). These findings are consistent with research indicating that higher Aβ levels before surgery are linked to tumor aggressiveness and progression. Amyloid Precursor Protein (APP) is the source of Aβ accumulation, which increases the invasiveness of breast cancer cells and suggests possible treatment paths connecting Aβ to the biology of breast cancer[23,24].

Breast cancer patients had significantly higher levels of Acetyl-CoA Carboxylase 1 (ACC1) pre-mastectomy (167.489 ± 1.899) and post-mastectomy (131.574 ± 3.162) than healthy controls (116.924 ± 4.551), as shown in Table (3). The rise is particularly noticeable in pre-surgery patients. These resultsagree with the research that found higher ACC1 levels in breast cancer patients, highlighting its function in satisfying tumor metabolic needs and its potential as a therapeutic target and prognostic marker. Furthermore, breast cancer cells exhibit dysregulated lipid metabolism, as evidenced by greater ACC1 activity than normal mammary epithelial cells[25].In comparison to healthy controls (212.603 ± 2.504), Table (3) shows that selenium concentration with selenium-binding protein 1 (SELENBP1) was lower in patients prior to mastectomy (162.404 ± 17.811) and increased after the procedure (201.330 ± 4.572). Studies reveal a correlation between decreased selenium binding to SELENBP1 in breast cancer tissues and treatment resistance as well as poor survival rates. This relationship affects hormone signaling, complicates illness outcomes, and is essential for selenium's anticancer activities, which include inducing apoptosis and arresting the cell cycle [16]. Tracking SELENBP1 levels in breast cancer patients may aid in survival rate prediction and direct selenium-based therapy approaches [26].Table (3) shows a significant decrease in Acetylcholine Esterase (AChE) levels in breast cancer patients, with values of (3.055 ± 0.228) before surgery and (4.108 ± 0.326) after surgery, compared to healthy controls (5.058 ± 0.174). This reduction is more pronounced before surgery. These results are consistent with two studies highlighting elevated AChE levels in breast cancer patients, emphasizing its role in meeting the metabolic demands of tumor cells and its potential as a therapeutic target and predictive marker. Additionally, breast cancer cells demonstrate dysregulation in lipid metabolism and exhibit higher AChE activity than normal breast epithelial cells[25].

3.2. Comparing the Biochemical Variables of Breast Cancer Patients with Healthy Controls based on Age Group

The variables listed in Table (4) were examined, with the results presented according to the studied age groups, as illustrated.

Table 4. Results ofstudied variables in breast cancer patients and the control group by age group.

Variable	Age Group	Control Group n=20 Mean ± SD	Patients Pre- mastectomy n=35 Mean ± SD	Patients post-mastectomy n=35 Mean ± SD
Interferon Activity (IFN- γ)	39-30	9.819 ± 0.095	6.541 ± 0.461	8.683 ± 1.192
	49-40	7.967 ± 0.537b	4.938 ± 0.371	7.456 ± 0.666
	59-50	6.923 ± 0.326	3.190 ± 0.355	6.758 ± 0.424

	>60	6.668 ± 0.711		5.175 ± 0.007
P Value		<0.05	<0.05	<0.05
Mammaglobin-A (SCGB2A2)	39-30	0.403 ± 0.031	0.594 ± 0.058	0.350 ± 0.013
	49-40	0.352 ± 0.013	0.519 ± 0.055	0.366 ± 0.042
	59-50	0.332 ± 0.001	0.485 ± 0.055	0.365 ± 0.042
	>60	0.330 ± 0.007		0.384 ± 0.053
P Value		<0.05	<0.05	<0.05
Indoleamine-2,3-deoxygenase (IDO)	39-30	68.621 ± 1.986	80.325 ± 3.458	71.868 ± 4.404
	49-40	73.114 ± 1.526	91.134 ± 6.549	73.161 ± 8.823
	59-50	83.340 ± 6.149	93.477 ± 5.208	73.421 ± 9.774
	>60	88.654 ± 1.099		77.967 ± 10.864
P Value		<0.05	<0.05	<0.05
Amyloid Beta Peptide (Aβ	39-30	66.711 ± 2.537	73.028 ± 2.938	69.978 ± 1.456
	49-40	68.227 ± 1.644	86.236 ± 7.470	72.229 ± 4.438
	59-50	78.873 ± 4.456	90.325 ± 7.428	72.604 ± 3.761
	>60	81.820 ± 0.080		76.818 ± 6.072
P Value		<0.05	<0.05	<0.05
Acetylcholine Carboxylase (ACC1)	39-30	126.682 ± 0.947	171.120 ± 1.944	140.236 ± 3.904
	49-40	119.663 ± 2.799	167.219 ± 1.631	135.040 ± 3.300
	59-50	110.278 ± 4.672	164.128 ± 2.122	125.764 ± 3.201
	>60	111.070 ± 9.784		125.256 ± 2.243
P Value		<0.05	<0.05	<0.05
Selenium Binding Protein 1 (SELEBP1)	39-30	220.771 ± 3.747	176.927 ± 24.222	219.627 ± 10.517
	49-40	217.736 ± 0.982	163.162 ± 16.331	214.319 ± 10.517
	59-50	208.500 ± 4.619	147.617 ± 13.608	193.368 ± 1.878
	>60	203.403 ± 0.664		191.203 ± 1.319
P Value		<0.05	<0.05	<0.05
Acetylcholine Esterase (AChE)	39-30	5.882 ± 0.214	3.423 ± 0.202	4.466 ± 0.297
	49-40	5.253 ± 0.159	3.129 ± 0.151	4.656 ± 0.305
	59-50	4.710 ± 0.270	2.612 ± 0.331	3.641 ± 0.322
	>60	4.385 ± 0.049		2.668 ± 0.378
P Value		<0.05	<0.05	<0.05

Interferon-gamma (IFN-γ) efficacy significantly decreased with age in all tested groups (healthy individuals, patients undergoing pre-mastectomy, and patients following mastectomy), as Table (4) illustrates. IFN-γ levels in healthy persons aged 30-39 were (0.095 ± 9.819), whereas those aged 60 and beyond had (0.711 ± 6.668) values. Due to decreased T-cell reactivity and greater regulatory T-cell activity, older adults produce less IFN-γ, which suppresses immunological responses [27]. Patients with breast cancer, especially those who are older, also have similar reductions in IFN-γ, which affect immune function and treatment outcomes because of decreased naïve T-cells and increased immunological exhaustion [28]. This pattern continues after surgery, indicating that the immune system's capacity to generate IFN-γ might not fully recuperate, particularly in elderly people, which could have an impact on immunotherapy and other treatments that come after [29].Table (4) demonstrates that in all three groups (healthy individuals, patients undergoing pre-mastectomy, and patients recovering from mastectomy), the effectiveness of mammaglobin diminishes modestly with age. The concentration was (0.031 ± 0.403) in healthy people aged 30-39, but it dropped to (0.007 ± 0.330) in people over 60. Patients who had had mastectomy before and after likewise showed this trend. Mammaglobin levels in breast cancer patients are not greatly impacted by age, however these results show that mammaglobin may probably be employed as a reliable biomarker for the diagnosis and follow-up of breast cancer in a variety of age groups. Mammaglobin concentrations may be further investigated in future studies in connection to additional clinical and demographic variables[30].Table (4) demonstrates that in all examined groups—healthy individuals, patients undergoing surgery, and patients recovering from surgery—the activity of Indoleamine 2,3-Dioxygenase (IDO) increases dramatically with age. IDO concentration was (1.986 ± 68.621) in healthy persons aged 30-39, and (1.099 ± 88.654) in those aged 60 and above. Age-related disorders including dementia and atherosclerosis may be exacerbated by increasing IDO activity, which has been shown to impact immunological functions and exacerbate inflammation. It may also change T-cell populations and elevate inflammatory markers. Controlling IDO activity may help treat these illnesses and improve immunity in the elderly[31,32].Table (2.3) shows that, across all study groups (healthy individuals,

patients undergoing pre-mastectomy, and patients recovering from mastectomy), the effectiveness of amyloid beta peptide (Aβ) increases significantly with age. The concentration was (2.537 ± 66.711) in healthy adults aged 30-39, and (0.080 ± 81.820) in those over 60. The hallmark of Alzheimer's disease, increased amyloid deposition, is linked to aging. Additionally, changes in metabolism and hormones may make Aβ accumulation in breast cancer worse. These modifications affect oxidative stress, inflammation, and lipid metabolism, which in turn affect APP processing and Aβ generation. Hormonal changes also impact the course of breast cancer and the metabolism of amyloid, particularly in postmenopausal women. Estrogen has an impact on the concentration and processing of APP[24,33].

The results in Table (4) indicate that the activity of Acetyl-CoA Carboxylase (ACC1) significantly decreases with age across all studied groups (healthy individuals, pre-mastectomy patients, and post-mastectomy patients). In healthy individuals aged 30-39, the ACC1 concentration was (0.947 ± 126.682), while it decreased to (9.784 ± 111.070) in those aged 60 and above. A similar trend was observed in both pre- and post-mastectomy patients. This decline in ACC1 enzyme levels with advancing age represents a significant metabolic change that affects lipid metabolism and may contribute to age-related health issues[34].As individuals age, the effectiveness of Selenium-binding Protein (SELENBP1) declines considerably across all study groups (healthy adults, pre- and post-mastectomy patients), as demonstrated in Table (4). The concentration was (3.747 ± 220.771) in healthy adults aged 30-39, and it decreased to (0.664 ± 203.403) in those aged 60 and above. In the other patient groups, comparable patterns were seen. Reduced SELENBP1 levels have been linked to aging and changes in selenium metabolism. Antioxidant defenses and healthy aging depend on maintaining sufficient selenium levels, as a deficit can hasten aging and increase susceptibility to age-related illnesses[35].

Acetylcholine Esterase (AChE) activity significantly decreased with age in all study groups (healthy adults, pre-mastectomy patients, and post-mastectomy patients), as Table (4) demonstrates. In healthy persons aged 30-39, AChE activity was (0.214 ± 5.882); in those over 60, it dropped to (0.049 ± 4.385). Because of a decrease in AChE activity, aging is linked to decreased cholinergic neurotransmission, which has a negative impact on cognitive abilities. The concentration of AChE protein does not change, but its function is disrupted by age-based oxidative stress caused by reactive O_2 species. AChE inhibitors are used to improve cholinergic neurotransmission and preserve cognitive function in neurodegenerative disorders like Alzheimer's disease, where this decrease in AChE activity is significant[36].

4. CONCLUSION

According to the findings, breast cancer patients' pre- and post-mastectomy biomarker levels changed significantly. Advanced stages were associated with reduced levels of interferon-gamma (IFN-γ), whereas mammaglobin (SCGB2A2) increased prior to surgery and decreased following it, suggesting a successful excision of the tumor. While changes in Amyloid Beta (Aβ), Acetyl-CoA Carboxylase (ACC1), and Selenium-binding Protein (SELENBP1) reflect their functions in tumor metabolism, elevated levels of Indoleamine 2,3-Dioxygenase (IDO) signal immune evasion. Age also affects immunological response, inflammation, and changes in cognition, which highlights the necessity for age-appropriate treatment plans.

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