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Alteration in cardiac biomarkers associated with breast cancer therapy-induced cardiotoxicity

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Barzy Karim Hassan [1*] **Sardar Nouri Ahmed** [2]

Abstract

Background and objective: Breast cancer medications, including chemotherapy, radiation, and targeted treatments, are linked to increased cardiovascular risk, frequently leading to early cardiac dysfunction. This study examines cardiac and lipid biomarker alterations throughout breast cancer chemotherapy and their correlation with cardiovascular problems.

Methods: A case-control study involved 90 females, 60 breast cancer patients (30 before and 30 after treatment), and 30 healthy controls. Serum levels of lipid profile (Total Cholesterol, Triglycerides, HDL-C, LDL-C, VLDL-C), the tumor marker CA 15-3, and cardiac biomarkers Brain Natriuretic Peptide (BNP), Troponin T, CK-MB, Gamma-Glutamyl Transferase (GGT), and C-reactive Protein (CRP) were assessed.

Results: The mean age was 44±1.64 for the controls and 46.53±1.14 in patients. The BMI was significantly higher in breast cancer patients (P <0.001). Troponin T levels significantly rose (95% confidence intervals post-treatment were (2.46 to 2.70, P <0.001). Triglycerides, VLDL, and atherogenic index increased significantly P-values (0.011, 0.011, 0.014). CA 15-3 concentrations were significantly elevated in both pre-treatment and post-treatment groups as compared to controls (P-value = 0.016). Post-treatment BNP (95% CI: 52.7 to 131.7) levels were elevated, but lacked statistical significance (P-value 0.081).

Conclusion: Elevated Troponin T and BNP levels after breast cancer treatment, indicating early cardiac stress, which supports the need for routine monitoring of cardiac biomarkers in patients undergoing therapy. Early detection of subclinical cardiotoxicity could allow for timely intervention and help reduce long-term cardiovascular complications.

Keywords: Breast cancer, Cardiac toxicity, Cardiac biomarkers.

[1] **Department of Clinical Pharmacy, College of Pharmacy, Hawler Medical University, Erbil, Kurdistan region, Iraq.**
[2] **Department of Community Medicine, College of Medicine, Hawler Medical University, Erbil, Kurdistan region, Iraq.**
[3] **Department of Pharmacy, Faculty of Pharmacy, Tishk International University, Erbil, Kurdistan region, Iraq.**
Correspondence: iman.ahmed.abdulrahman@gmail.com

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Introduction

Breast cancer is the predominant cause of cancer-related mortality among women globally. It is the primary cause of death in women in undeveloped nations and the second largest cause among women in affluent nations (1).

Progress in breast cancer treatment, encompassing chemotherapy, targeted treatments, and radiotherapy, has markedly enhanced survival rates. Nonetheless, these therapies often have serious cardiovascular side effects, which may affect overall quality of life (2).

Cardiotoxicity arising from breast cancer therapy is a growing issue, as indicated by the rising number of studies emphasizing the cardiovascular hazards linked to contemporary oncological treatments. Anthracyclines, a widespread class of chemotherapeutic drugs, are recognized for their dose-dependent cardiotoxicity, leading to diseases such as cardiomyopathy and heart failure (3).

Brain natriuretic peptide was first found in swine brain tissue as a peptide with natriuretic-diuretic and smooth muscle relaxant characteristics. Soon thereafter, they discovered that human BNP was generated and secreted from the heart as a cardiac hormone (4).

Cardiac troponins (cTn) are structural proteins that bind to actin-containing thin filaments in cardiac muscle and play a crucial role in regulating myocardial contraction by modulating calcium ion interaction and decreasing the ATPase activity of actin-myosin (5).

B-type natriuretic peptides (BNPs) and cardiac troponin levels have been associated with high-dose chemotherapy (6). Serum cardiac biomarker measurement offers a viable and valuable tool for preliminary assessment of risk, early cardiovascular disease diagnosis during and after treatment, identifying cancer patients who might benefit from cardioprotective therapies during oncological treatment, and providing prognostic value to determine patients requiring long-term CVD monitoring (7).

Depending on the cancer therapy being utilized, some cancer treatments might have cardiotoxic side effects that raise plasma troponin levels. Traditionally, immune checkpoint inhibitors cause heart inflammation, while chemotherapy is linked to mitochondrial damage and an increase in reactive species (8).

The study aims to assess how cardiovascular biomarkers are affected by breast cancer treatment and how those changes are related to heart failure, to improve treatment outcomes and quality of life for breast cancer survivors.

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Methods

A case-control study was conducted in Erbil City/Iraq, and the current study was conducted from 29th September to14th January 2025. A total of 90 female blood samples were collected and categorized into the healthy controls (Group I), which were randomly selected and did not exhibit any signs of disorders, and they were age and gender matched to ensure comparability and minimize confounding variables. The patients (Group II) comprised 30 patients before therapy, and Group III included 30 patients after treatment who were diagnosed with breast cancer and were picked from Nanakali Hospital and Rizgary Teaching Hospital. They were aged from 26 to 67 years, including patients undergoing chemotherapy, hormonal therapy, and targeted therapy. A structured questionnaire was specifically developed for the study. The initial section focused on collecting demographic information, including name, age, BMI, marital status, residency, and occupation. The second part consists of questions of cancer treatment history, including (stages, grades, cycles, side of the breast cancer, type of treatment that was received, duration of receiving treatment, and side effects or symptoms related to the cardiovascular system during treatment). The third part was related to medical history (diabetes mellitus, hypertension, heart disease, and other diseases). The fourth part was family history and menopausal status, and the fifth part of the questionnaire was composed of questions related to lifestyle data as smoking and physical activity.

Exclusion criteria

Women who aged less than 26 and more than 67, hemolysis of blood samples, finished their treatment plan for the post-treatment group, patients on medications known to significantly alter cardiovascular biomarkers independently of cancer treatment, those did not complete their investigation and having unknown stage or lymph node, the one who undergoing radiotherapy, and patients with condition such as Down syndrome and those who were previously pregnant with breast cancer.

Ethical consideration

The study was approved by the Ethics Committee of the College of Health Sciences/Hawler Medical University, and verbal approval was obtained from each patient. Confidentiality was assured, and all pieces of information about each patient were kept private.

Blood Collection

Five milliliters (ml) of venous blood samples were collected and placed in a serum separator tube (SST); the serum was then centrifuged for 10 minutes at 3500 rpm after remaining at room temperature for 20 minutes. The serum was transferred into sealed Eppendorf

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tubes and directly stored at -50 °C. The separated blood serum was used to perform biochemical tests, including ELISA for B-type natriuretic peptide, Cobas E411 for Troponin T, CK-MB, and CA 15-3 antigen, and Cobas 6000 for serum total cholesterol (TC), triglycerides (TG), high-density lipoprotein (HDL), and low-density lipoprotein (LDL).

Statistical Analysis

The SPSS (Statistical Package for Social Science) (Version 26) was utilized for statistical analysis. Descriptive statistics were presented as the mean ± standard error for the variables of interest. The association between categorical variables across groups was assessed using the chi-square test; for continuous variables such as BMI and age, the case and control groups were compared using an independent t-test. The ANOVA test was used to differentiate between more than two means. A general linear model (GLM) was used to evaluate the effect of demographic factors on the cardiac biomarker, in addition to 95% confidence intervals, reported to show the proportion of variance. P-values under 0.05 were considered significant.

Results

Age, marital status, residency, smoking, and exercise are among the socioeconomic and demographic traits of the control group that have been observed in the results. No statistically significant changes were found between the patient and control groups, with the P-values (0.205, 0.100, 0.067, 0.213, and 0.473). Meanwhile, other risk factors between the study population's case and control groups, the Chi-square and t-test analysis of differences in risk factors between patients and healthy people revealed statistically significant differences between the two groups of subjects in the majority of risk factors such as occupation, menstrual cycle, family history, and BMI with a P-value of (0.002, 0.03, 0.014, and <0.001). The mean ± standard error (Mean ±SE) age of the control group was 44.00 ± 1.637, also the patient group was 46.53 ± 1.14 with a range of 26 - 67 years old, the mean ± standard error (Mean ±SE) body mass index (BMI) of females with breast cancer was 33.05±0.78 (Kg/m^2), and the control group was 27.512±0.75 (Kg/m^2). This information is provided in Table 1.

There is an increase of 95% confidence intervals in BNP levels after treatment (52.7 – 131.7) compared to both control (56.7 – 69.7) and pre-treatment levels (53.6 – 62.9), but the P value (0.081) is not statistically significant. Troponin T levels significantly increased post-treatment (95% CI: 2.46 – 2.70, P <0.001). There is a decreasing trend in CK-MB levels post-treatment, but the P value (0.155) suggests no statistically significant difference. GGT diminished in post-treatment compared to pre-treatment but remained higher than in the control group. However, the high standard error and P-value (0.32)

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suggest no statistically significant difference. CRP levels increased post-treatment (95% CI: 4.23 – 12.18), which may indicate increased inflammation. However, the P-value (0.088) is not statistically significant. This information can be summarized in Tables 2 and 3.

Table 1. Demographic and social characteristics of participants' groups

Parameters		Control Group n=30 No. (%)	Patient group n=60 No. (%)	P-value
Marital status	Married	25 (83.3)	57 (95)	0.067
	Single	5 (16.7)	3 (5)	
Residency	City	10 (33.3)	31 (51.7)	0.100
	Countryside	20 (66.7)	29 (48.3)	
Occupation	Employed	13 (43.3)	8 (13.3)	0.002*
	Housewife	17 (56.7)	52 (75.4)	
Smoking	Non-smoker	30 (100)	57 (95)	0.213
	Ex-smoker	0 (0.0)	3 (5)	
Exercise	Yes	8 (26.7)	12 (20)	0.473
	No	22 (73.3)	48 (80)	
Menopausal status	Postmenopausal	14 (46.7)	42 (70)	0.031*
	Premenopausal	16 (53.3)	18 (30)	
Family history	Yes	6 (20)	28 (46.7)	0.014*
	No	24 (80)	32 (53.3)	
Age	Mean ± SE 46.53 ± 1.14	44.0 ± 1.64		0.205
BMI	Mean ± SE 33.05 ± 0.78	27.51 ± 0.75		<0.001*

* The mean difference is significant at the 0.05 level.

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Table 2. Chemical parameters of serum BNP, Troponin T, CK-MB, GGT, and CRP in the studied groups

Parameters	Control Mean ± SE	Pre-treatment Mean ± SE	Post-treatment Mean ± SE	*P* value
BNP Pg/mL	63.18 ± 3.17	58.28 ± 2.25	92.18 ± 19.33	0.081
TroponinT Pg/mL	1.73 ± 0.95	2.06 ± 0.01	2.57 ± 0.06	<0.001*
CK-MB ng/mL	1.30 ± 0.25	1.01 ± 0.15	0.76 ± 0.18	0.155
GGT U/L	18.23 ± 2.21	48.43 ± 23.09	33.70 ± 6.92	0.32
CRP mg/dL	3.75 ± 0.92	5.66 ± 1.17	8.20 ± 1.95	0.088

* Significant difference

Table 3. 95% Confidence Intervals of biomarkers across study groups

Parameters	Control 95% (CI)	Pre-treatment 95% (CI)	Post-treatment 95% (CI)
BNP Pg/mL	56.7 – 69.7	53.7 – 62.9	52.7 – 131.7
TroponinT Pg/mL	1.54 – 1.92	1.86 – 2.26	2.46 – 2.70
CK-MB ng/mL	0.75 – 1.76	0.71 – 1.31	0.38 – 1.13
GGT U/L	13.70 – 22.8	1.22 – 95.7	19.55 – 47.85
CRP mg/dL	1.88 – 5.62	3.26 – 8.06	4.23 – 12.18

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Table 4 presents the association between Troponin T levels and the combination of variables: participant groups, age, BMI, and menopausal status; significant differences were only shown between individual groups (P-value ≤0.001) with an R^2 of 0.406, indicating that approximately 40.6% of the variability in Troponin T could be explained by the included variables. In contrast, age (P = 0.093), BMI (P = 0.78), and menopausal status (P = 0.99) were not significantly associated with Troponin T variation. Data from Table 5 of the lipid profile and tumor marker CA 15-3 among breast cancer patients revealed significant modifications post-treatment. While total cholesterol, HDL, and LDL levels did not exhibit statistically significant variations across groups, each of TG, VLDL-c, atherogenic index, and the tumor marker CA15-3 showed significant differences with P-values (0.011, 0.011, 0.014, and 0.016).

Table 4. The combination of (age, BMI, menopausal status, and participant groups) and variation in Troponin T

Variables	F-Value	P-Value
Corrected Model	11.50	<0.001*
Participant groups	14.84	<0.001*
Age	2.89	0.093
BMI	0.081	0.78
Menopausal status	<0.001	0.99

*Significant at the 0.05 level (R squared = 0.406)

Table 5. Serum levels of TC, TG, HDL-c, LDL-c, VLDL-c, LDL/HDL ratio, Atherogenic Index, and CA 15-3 of the studied groups

Parameter mg/dL	Control Mean ± SE	Pre-treatment Mean ± SE	Post-treatment Mean ± SE	P-value
Cholesterol	149.73 ± 5.86	162.16 ± 6.71	167.64 ± 7.94	0.176
Triglyceride	112.79 ± 9.74	125.11 ± 11.52	166.10 ± 16.29	0.011*
HDL-c	44.47 ± 1.59	41.21 ± 2.6	43.21 ± 3.15	0.628
LDL-c	82.87 ± 5.35	93.87 ± 6.13	94.10 ± 5.47	0.281
VLDL-c	22.54 ± 1.95	25.02 ± 2.31	33.22 ± 3.26	0.011*
LDL/HDL ratio	1.95 ± 0.15	2.35 ± 0.21	2.43 ± 0.13	0.101
Atherogenic Index of Plasma (AIP)	0.36 ± 0.49	0.42 ± 0.54	0.57 ± 0.48	0.014*
CA 15-3 u/mL	6.67 ± 0.33	17.055 ± 3.02	23.39 ± 3.62	0.016*

*Significant difference

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Discussion

Cardiotoxic effects of established therapeutic regimes and innovative treatments have contributed to an increased cardiovascular morbidity and death in cancer survivors (9).

In this study, we investigated changes in cardiac biomarkers and lipid parameters before and after breast cancer therapy, compared with a healthy control group, and explored their association with cardiovascular dysfunction. The data analysis demonstrated that statistically significant differences were observed between the control and patient groups regarding several risk factors, including family history, occupation, suggesting that patients were more likely to be housewives than employed, which may reflect differing physical activity levels and stress exposure factors linked to cardiovascular health. Furthermore, a higher incidence of post-menopausal status was noted in the patient group (70%) compared to controls (46.7%), which was statistically significant P-value equal (0.031). This hormonal shift mayincrease cardiovascular risk, consistent with prior studies that menopause to both cancer progression and cardiac disease (10-12).

Body Mass Index (BMI) was significantly higher in the patient groups (33.05±0.78) than in controls (27.51 ± 0.75), with a P- value <0.001. Obesity and overweight in women greatly increase the risk of breast cancer; also, elevated BMI is independently associated with worse cardiovascular outcomes and could potentiate the cardiotoxic effects of chemotherapy (13, 14).

The troponin T and troponin I isoforms are specific to cardiac muscle and are commonly used in the diagnosis and prognosis of cardiovascular disease (5). In the current study, Troponin T levels significantly increased post-treatment (from 2.06 ± 0.01 to 2.57 ± 0.06; P <0.001), which was influenced by treatment-related factors rather than demographic and physiological factors such as age, BMI, and menopausal status, with a non-significant effect (P-value = 0.93, 0.78, and 0.99). Troponins are sensitive markers of cardiac muscle damage and are frequently used to detect chemotherapy-induced cardiotoxicity, which emphasizes the predictive value of elevated troponin levels in anticipating late-onset cardiac dysfunction in breast cancer patients undergoing chemotherapy, targeted treatment, and hormonal therapy. The current study findings suggest that troponin T is a reliable early marker for cardiotoxicity (15).

Zhang et al. (2019) revealed a significant change in brain natriuretic peptide (BNP) levels post radiotherapy. Similarly, Lu et al. (2019) assessed BNP as a predictive marker for anthracycline-induced cardiotoxicity. Although our study examined BNP, a marker of ventricular stress and heart failure, the

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confidence intervals revealed that BNP levels remained relatively stable between control and pre-treatment but showed a wide range post-treatment (95% CI: 52.7 – 131.7). While this increase was not statistically significant (P-value = 0.081), the upward trend aligns with literature reporting BNP elevation in patients receiving cardiotoxic drugs (16-18). Even if BNP increased after chemotherapy, the small sample size, heterogeneity of treatment regimens, and the large standard error may indicate individual variability or delayed response.

C-reactive protein, a marker of systemic inflammation, increased from 5.66 ± 1.17 to 8.20 ± 1.95 post-treatment (95% CI: 4.23 – 12.18), P-value was equal (0.088), in contrast to the findings of prospective analysis showed no association between circulating inflammation and breast cancer risk (19). This may be due to a modest sample size, with disagreement with another study that reported higher CRP levels in patients with reduced left ventricular ejection fraction following chemotherapy, further linking systemic inflammation with cardiac injury (20).

The lipid profile disruptions observed post-treatment support findings, which discuss chemotherapy's role in promoting atherogenic dyslipidemia. The significant rise in the atherogenic index in this study strengthens this link and underscores the need for aggressive lipid monitoring in breast cancer patients (21).

A previous study reported that high blood lipid levels have been linked to chronic inflammation by starting the proinflammatory signaling cascade reaction, according to studies (22), and because the development of breast cancer is linked to inflammation. It is hypothesized that the early onset of hyperlipidemia may be linked to breast cancer in postmenopausal patients. Early screening is recommended for people with postmenopausal breast cancer. This study also reveals that the levels of TG and VLDL were increased significantly during chemotherapy, HDL-c levels fell following treatment, the prevalence of hyperlipidemia also increased significantly ($P < 0.05$), and blood lipid levels were in the direction of growing atherosclerosis (23, 24).

In our study, serum CA 15-3 levels were significantly higher in breast cancer patients compared with healthy controls, in agreement with a previous study that demonstrated CA 15-3 significantly increased in patients with breast cancer compared with controls (25).

In conclusion, a lack of highly associations between these biomarkers and cardiac dysfunction is due to the small sample size because it was difficult to obtain samples within the study requirements and short period, and variability in treatment types, which will require further studies with a larger

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sample size to determine the effect of each of treatments on BNP and the role of inflammation in cardiotoxicity.

Conclusion

The potential cardiac effects of breast cancer therapy were indicated by the considerable increase in Troponin T levels following treatment, as well as the increase in BNP levels relative to both control and pre-treatment values. In patients with breast cancer, Troponin T may be a trustworthy early biomarker for identifying heart problems brought on by treatment.

Competing interest

The authors declare that they have no competing interests.

References

1. Seely J, Alhassan T. Screening for breast cancer in 2018—what should we be doing today? Curr Oncol. 2018;25(s1):115-24. https://doi.org/10.3747/co.25.3770

2. Lyon AR, Dent S, Stanway S, Earl H, Brezden-Masley C, Cohen-Solal A, et al. Baseline cardiovascular risk assessment in cancer patients scheduled to receive cardiotoxic cancer therapies: a position statement and new risk assessment tools from the Cardio-Oncology Study Group of the Heart Failure Association of the European Society of Cardiology in collaboration with the International Cardio-Oncology Society. Eur J Heart Fail. 2020;22(11):1945-60. https://doi.org/10.1002/ejhf.1920

3. Zamorano JL, Lancellotti P, Rodriguez Munoz D, Aboyans V, Asteggiano R, Galderisi M, et al. 2016 ESC Position Paper on cancer treatments and cardiovascular toxicity developed under the auspices of the ESC Committee for Practice Guidelines: The Task Force for cancer treatments and cardiovascular toxicity of the European Society of Cardiology (ESC). Eur Heart J. 2016;37(36):2768-801. https://doi.org/10.1093/eurheartj/ehw211

4. Nakagawa Y, Nishikimi T, Kuwahara K. Atrial and brain natriuretic peptides: Hormones secreted from the heart. Peptides. 2019;111:18-25. https://doi.org/10.1016/j.peptides.2018.05.012

5. Simões R, Silva LM, Cruz ALVM, Fraga VG, de Paula Sabino A, Gomes KB. Troponin as a cardiotoxicity marker in breast cancer patients receiving anthracycline-based chemotherapy: A narrative review. Biomedicine & Pharmacotherapy. 2018;107:989-96. https://www.sciencedirect.com/science/article/pii/S0753332218336400

6. Gulati G, Heck SL, Røsjø H, Ree AH, Hoffmann P, Hagve TA, et al. Neurohormonal blockade and circulating cardiovascular biomarkers during anthracycline therapy in breast cancer patients: results from the PRADA (Prevention of Cardiac Dysfunction During Adjuvant Breast Cancer Therapy) study. J Am Heart Assoc. 2017;6(11):e006513.

https://doi.org/10.15218/zjms.2026.015

https://doi.org/10.1161/jaha.117.006513

7. Pudil R, Mueller C, Čelutkienė J, Henriksen PA, Lenihan D, Dent S, et al. Role of serum biomarkers in cancer patients receiving cardiotoxic cancer therapies: a position statement from the Cardio-Oncology Study Group of the Heart Failure Association and the Cardio-Oncology Council of the European Society of Cardiology. Eur J Heart Fail. 2020;22(11):1966-83. https://doi.org/10.1002/ejhf.2017

8. Romann SW, Giannitsis E, Frey N, Lehmann LH. Troponin Elevation in Asymptomatic Cancer Patients: Unveiling Connections and Clinical Implications. Curr Heart Fail Rep. 2024;21(6):505-14. https://doi.org/10.1007/s11897-024-00681-x

9. Michel L, Rassaf T, Totzeck M. Biomarkers for the detection of apparent and subclinical cancer therapy-related cardiotoxicity. J Thorac Dis. 2018;10(Suppl 35):S4282. https://doi.org/10.21037/jtd.2018.08.15

10. Barac A, Murtagh G, Carver JR, Chen MH, Freeman AM, Herrmann J, et al. Cardiovascular health of patients with cancer and cancer survivors: a roadmap to the next level. Am J Cardiol. 2015;65(25):2739-46. https://doi.org/10.1016/j.jacc.2015.04.059

11. Koene RJ, Prizment AE, Blaes A, Konety SH. Shared risk factors in cardiovascular disease and cancer. Circulation. 2016;133(11):1104-14. https://doi.org/10.1161/circulationaha.115.020406

12. Liu H, Shi S, Gao J, Guo J, Li M, Wang L. Analysis of risk factors associated with breast cancer in women: a systematic review and meta-analysis. Transl Cancer Res. 2022;11(5):1344. https://doi.org/10.21037/tcr-22-193

13. Pati S, Irfan W, Jameel A, Ahmed S, Shahid RK. Obesity and cancer: a current overview of epidemiology, pathogenesis, outcomes, and management. Cancers. 2023;15(2):485. https://doi.org/10.3390/cancers15020485

14. Tzenios N, Tazanios ME, Chahine M. The impact of BMI on breast cancer–an updated systematic review and meta-analysis. Med. 2024;103(5):e36831.

15. Cardinale D, Colombo A, Bacchiani G, Tedeschi I, Meroni CA, Veglia F, et al. Early detection of anthracycline cardiotoxicity and improvement with heart failure therapy. Circulation. 2015;131(22):1981-8. https://doi.org/10.1161/circulationaha.114.013777

16. Cameron AC, Touyz RM, Lang NN. Vascular complications of cancer chemotherapy. Can J Cardiol. 2016;32(7):852-62. https://doi.org/10.1016/j.cjca.2015.12.023

https://doi.org/10.15218/zjms.2026.015

17. Zhang C, Shi D, Yang P. BNP as a potential biomarker for cardiac damage of breast cancer after radiotherapy: a meta-analysis. Medicine (Baltimore). 2019;98(29):e16507. https://doi.org/10.1097/md.0000000000016507

18. Lu X, Zhao Y, Chen C, Han C, Xue L, Xing D, et al. BNP as a marker for early prediction of anthracycline-induced cardiotoxicity in patients with breast cancer. Oncol Lett. 2019;18(5):4992-5001. https://doi.org/10.3892/ol.2019.10827

19. Cairat M, Rinaldi S, Navionis A-S, Romieu I, Biessy C, Viallon V, et al. Circulating inflammatory biomarkers, adipokines, and breast cancer risk—a case-control study nested within the EPIC cohort. BMC Med. 2022;20(1):118. https://doi.org/10.1186/s12916-022-02319-y

20. Xiao H, Wang X, Li S, Liu Y, Cui Y, Deng X. Advances in biomarkers for detecting early cancer treatment-related cardiac dysfunction. Front Cardiovasc Med. 2021;8:753313. https://doi.org/10.3389/fcvm.2021.753313

21. Izquierdo-Condoy JS, Arias-Intriago M, Becerra Cardona DA, García-Cañarte S, Vinueza-Moreano P. Anticancer Chemotherapy-Induced Atherosclerotic Cardiovascular Disease: A Comprehensive Review. Life. 2025;15(2):245. https://doi.org/10.3390/life15020245

22. Hovland A, Jonasson L, Garred P, Yndestad A, Aukrust P, Lappegård KT, et al. The complement system and toll-like receptors as integrated players in the pathophysiology of atherosclerosis. Atherosclerosis. 2015;241(2):480-94. https://doi.org/10.1016/j.atherosclerosis.2015.05.038

23. Tian W, Yao Y, Fan G, Zhou Y, Wu M, Xu D, Deng Y. Changes in lipid profiles during and after (neo) adjuvant chemotherapy in women with early-stage breast cancer: A retrospective study. PloS one. 2019;14(8):e0221866. https://doi.org/10.1371/journal.pone.0221866

24. Sharma M, Tuaine J, McLaren B, Waters DL, Black K, Jones LM, McCormick SP. Chemotherapy agents alter plasma lipids in breast cancer patients and show differential effects on lipid metabolism genes in liver cells. PloS one. 2016;11(1):e0148049.

25. Varzaru VB, Eftenoiu A-E, Vlad DC, Vlad CS, Moatar AE, Popescu R, Cobec IM. The Influence of Tumor-Specific Markers in Breast Cancer on Other Blood Parameters. Life (Basel). 2024;14(4):458. https://doi.org/10.3390/life14040458