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Effects of antineoplastic drugs on oxidative stress and prognosis of hematological and various biochemical parameters in the treatment of breast cancer

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Abstract---Objective: This study aimed to to evaluate the variation and importance of certain hematological, enzymatic, and oxidative stress markers in women with breast cancer under chemotherapy treatment. Methods: The study comprised forty histopathologically proven female breast cancer patients at Omega Cancer Hospital in Visakhapatnam. All subjects were divided into four groups: a control group of 40 healthy females of similar age, a group of 40 breast cancer patients (before chemotherapy, during chemotherapy, and after

chemotherapy), and all subjects were undergoing treatment with anticancer agents. Results: During chemotherapy, lipid peroxidation and Nitric oxide (NO) levels were significantly increased in AC-treated breast cancer patients than in controls. Alanine aminotransferase (ALT), aspartate aminotransferase (AST), and alkaline phosphatase (ALP) were non-significant increase in treatment group than controls. Whereas biochemical profiles, were decreased in treatment group than controls. In AC-treated breast cancer patients, Hematological profiles were found significantly reduced than in controls. Conclusion: Chemotherapy causes a certain amount of systemic oxidative stress, which persists during subsequent clinical interventions and may influence the patients' clinical outcomes. Chemotherapy produced significant adverse effects such as anaemia, neutropenia, leukopenia, thrombocytopenia, and hepatic dysfunction as a side effect of treatment due to disturbed and lowered levels of haematological parameters.

Keywords---breast cancer, AC (Adriamycin cyclophosphamide), oxidative stress, chemotherapy/Treatment.

Introduction

The primary reason for cancer-related fatalities in women is breast cancer [1]. Like other malignancies, breast cancer arises from the breast tissue and is driven by the interaction of an environmental (external) component with a genetically predisposed host [2]. Breast lumps, breast form changes, skin dimpling, fluid seeping from the nipple, and red, scaly patches of skin are all indications of breast cancer [3]. Breast cancer is a complex illness [4]. The incidence of breast cancer has alternately been linked to the effects of some of the most important influencing factors, including human race, family history, age, physical activity, obesity, drinking alcohol, reproductive factors, hormone replacement therapy during menopause, ionising radiation, and the history of benign breast lesions [5]. BRCA1 and BRCA2, HER2/neu, and p53 are some of the genes that have been associated to breast cancer susceptibility and development [6]. The process of carcinogenesis depends heavily on oxidative stress. 3. Reactive oxygen species (ROS) cause cellular damage when they are stimulated by both endogenous and external causes [3].

Reactive oxygen species (ROS) are a crucial component of carcinogenesis and may contribute to the development and growth of malignancies. Free radicals promote oxidative DNA damage and mutagenesis, both of which are necessary for the development of tumours [7]. Both the growth of cancer and metastasis can be impacted by oxidative stress processes, which are also important in the activation of cell signalling pathways, enhanced tumour cell motility, increased tumour cell proangiogenic factors, and tumour cell proliferation [8, 9]. Lipid, protein, or DNA oxidation indicators are used to measure oxidative stress. The measurement of the final lipid peroxidation products yields information about the degree of lipid peroxidation. Lipid peroxidation's byproduct is malondialdehyde (MDA). It serves as a marker for oxidative stress. Malignant transformation may result from lipid

peroxidation brought on by oxygen-free radicals [10]. Breast cancer is exacerbated by oxidative stress, according to numerous research [11].

Superoxide anion, hydrogen peroxide, and hydroxyl radical are just a few examples of the molecules with a significant oxidising potential that are produced as a result of the metabolism of the quinone moiety into DOX, an anthracyclin. Because they cause a disruption in membrane organisation, hydroxyl radicals produced during the DOX redox cycle directly contribute to the process of lipid peroxidation [12, 13]. One of the most used anti-cancer medications, cyclophosphamide, is a bifunctional alkylating member of the nitrogen mustard family that causes chromosomal abnormalities, DNA adducts, and gene alterations. Hematological disorders are a common aspect to take into account in patients with breast cancer. These measures could be employed as one of the crucial biochemical instruments in the therapy monitoring and identification of additional comorbidities in breast cancer patients [16]. Hematological parameters are significant studies that are helpful prognostic indicators for assessing the precision of risk stratification in breast cancer patients, according to studies [17]. In the present study, the liver functioning (LFT) and Kidney functioning (KFT) were assessed to check the level of different components. It determines the healthiness and proper functioning of various organs during chemotherapy treatment. Alkaline phosphatase and liver transaminase (AST, ALT) levels were assessed as part of the liver function tests. Blood urea, creatinine, and uric acid are measured as part of a kidney function test (KFT) to assess how the kidneys are working [18].

Materials and Methods

Study Design

This study was conducted at Omega Cancer Hospital, Visakhapatnam. Ethics Committee approved it, and Informed consent was obtained from the patients.

Exclusion criteria

Alcohol abuse, autoimmune disease, chronic infection and inflammation, hypertension, cardiovascular disease, diabetes mellitus, renal failure, nephrotic syndrome, renal pathology, prior history of breast cancer, and use of antilipidemic, antioxidant, anti-inflammatory, corticosteroid, or immunosuppressive medications were all excluded criteria in both the study and control groups.

Inclusion Criteria

A total of 40 histopathologically confirmed female patients with breast cancer who were given chemotherapy using the AC regimen (adriamycin 60 mg/m2 and cyclophosphamide 600 mg/m2) were selected for the study. Before enrollment, each patient completed an informed consent form, and 40 patients did not get any chemotherapy. Three cycles of 21 days each with intravenous AC were administered to the patients. The study's control group, known as the unexposed group, was made up of participants who had not been exposed to radiation or

chemicals and were clear of any malignant neoplasms as well as any clinical, biochemical, haematological, hepatic, or renal symptoms. At the time of diagnosis, the BC patients in our study had distant metastasis. We assessed their clinic pathological characteristics, including histology, menopausal status, ER and PR status, number of affected axillary lymph nodes, grade, and tumour size and stage in accordance with the American Joint Committee on Cancer staging system. Medical records were consulted for information on clinical features, including cancer site, clinical stage, and HER-2/neu, ER (oestrogen receptor), and PR (progesterone receptor) status.

Methods

All study participants completed a questionnaire from the International Commission for Protection against Environmental Mutagens and Carcinogens [19], which asked about standard demographic information (such as age and gender), medical conditions (such as exposure to X-rays, vaccinations, and medications), lifestyle choices (such as smoking, coffee and alcohol consumption, diet, etc.), and employment, including how many hours per day were worked and protective measures used (PPE). Smokers were defined as those who smoked more than 20 cigarettes per day [20]. Lipid peroxide (malondialdehyde) was evaluated using an estimation of thiobarbituric acid reactive substances (TBARS) technique, and nitric oxide (NO) was measured using the Griess method [21]. The continuous spectrophotometric rate determination method was used to measure catalase. The ferric reducing antioxidant power (FRAP) assay using a spectrophotometric technique was used to measure the overall antioxidant status [22, 23]. Based on the measurement of pyruvate hydrazone and oxaloacetate hydrazonecolorimetrically at 546 nm, the liver enzymes alanine aminotransferase (ALT), aspartate aminotransferase (AST), and alkaline phosphatase (ALP) were analysed. Blood urea, serum creatinine, and uric acid were measured using the Burtis and Ashwood method. The WHO reference range and the results of the CBC SYSMEX XK -21N haematology analyzer were used to determine whether specific hematologic abnormalities in haemoglobin (Hb), platelet count, total white blood cell count (TWBC), neutrophils, and lymphocytes were present.

Blood sampling

7ml of fasting Peripheral venous blood was collected from the Normal control group, and patients and serum were used to determine the levels of catalase, malondialdehyde, nitric oxide (NO), total antioxidant status, blood urea, serum creatinine, and serum uric acid liver enzymes (alanine aminotransferase and aspartate aminotransferase, alkaline phosphatase). Blood sampling is performed, and after serum separation, the samples were immediately transported to the Biochemistry Research Laboratory and stored at−50^{o}C for biochemical analysis. Data were collected for hematological profiles, white blood cell (TWBC) count, platelets (PLT), (hemoglobin (Hb), neutrophils, and lymphocytes). The first collection was performed before the beginning of treatment (C0), 21 days after the second cycle of chemotherapy (C2), and 21 days after the fourth cycle (C4).

Statistical analysis

The Statistical Package for Social Sciences (SPSS) 16.0 for Windows version was used to examine the data. The sociodemographic traits of the study subjects were then evaluated using a descriptive analysis. The mean + SD of various oxidative biomarkers, haematological indices, and metabolic profiles were compared using an independent sample t-test. The substantial variations in haematological parameters between patients and controls were also examined using a Chi-square test. Statistics were considered significant at P-values under 0.05.

Results

Socio-demographic characteristics of the study participants

40 cases and 40 controls were included in this study. The mean ages of the study participants were 30-75 years. Regarding marital status, 55.0 % of the cases and 60.0 % of the controls were married. However, nearly half (22.5%) of the patients were not able to read and write, while 25.0% of the controls had attained secondary education ($P < 0.05$) for cases and controls, respectively. (Table 1). Of the 40 participants aged 30-75, 60.8% had invasive ductal carcinoma. Demographic characteristics of participants with the various classes of breast cancer are shown in Table 2. Demographic distribution of the participants indicated that patients with invasive ductal carcinoma (NST) and invasive lobular carcinoma (NOS) were mainly within 30- 75, ≤35, and ≥75 years, respectively. The majority were married, with a few being single. Most had an informal form of occupation and were overweight as well. Details about clinical features, such as cancer site, clinical stage, and HER-2/neu, ER (estrogen receptor), and PR (progesterone receptor) status, were obtained and analyzed from medical records. The descriptive statistics for such variables are listed in Table 2.

The results of Table 3 demonstrated that the baseline hematological and biochemical characteristics of both control and test participants. The significant differences in the mean values of hematological and biochemical parameters have been identified with the paired t-test analysis. However, the platelet count, tumor side, and lymphocyte count showed insignificant variation between the control and test participants.From Table 4, it was found that there was a highly significant variation was observed between the control and test subjects while studying various hematological and biochemical parameters except for TWBC and neutrophils count. Table 5 shows the significant differences in the mean values of all parameters, except the biochemical parameter uric acid with the paired t-test analysis, which has been identified. Table 6 represents the correlation of various biomarkers levels of renal, hepatic, hematological, and oxidative stress, an antioxidant enzymatic system of patients with breast cancer before, during, and after chemotherapy between controls and test subjects. It was found that there was a significant correlation between the parameters like ALP, TAS, NO, lymphocyte, and neutrophil count between the control and test subjects before treatment and during treatment; a significant difference was noted in lymphocyte and neutrophil count, whereas in case of after treatment the significant difference was noted in NO, lymphocyte and neutrophil count. Figure 1 a-c showed the Photomicrograph showing strong nuclear estrogen receptor (ER), strong nuclear

progesterone receptor (PR) anduniform intense membrane HER2/neuimmunoreactivity in tumor cells. Figure 2 a-c represented the various clinical images of mammograms.

Table 1
Demographic, medical, and lifestyle data of the patients and controls

Characteristics	Control group	Breast cancer
Total of patients	40 (100.0)	40 (100.0)
Age in years (mean ± SD)	30-75 years	30-75 years
Menopause [*n* (%)]		
Premenopausal	28 (70.0)	26 (65.0)
Postmenopausal	12 (30.0)	14 (35.0)
Family history of breast cancer [*n* (%)]		
Yes	6 (15.0)	22(55.0)
No	34 (85.0)	18(45.0)
Physical exercises [*n* (%)]		
Yes	17(42.5)	13(32.5)
No	23(57.5)	27(67.5)
Smoker [*n* (%)]		
Never smoked	35(87.5)	19(47.5)
Smoking	5(12.5)	3(7.5)
Ex-smoker	0(0.0)	18(45)
Marital status [*n* (%)]		
Single	13(32.5)	11(27.5)
Married	22(55.0)	24(60.0)
Divorced	4(10)	3(7.5)
Widow	1(2.5)	2(5.0)
Education Level		
Not able to read and write	3(7.5)	9(22.5)
Able to read and write	7(17.5)	6(15.0)
Primary education	9(22.5)	7(17.5)
Secondary education	10(25.0)	10(25.0)
College and University	11(27.5)	9(22.5)

Table 2
Clinical Characteristics of Patients with Breast Invasive Ductal, lobular carcinoma (n = 40)

Characteristics	Breast cancer
Cancer sites [*n* (%)]	
Left breast	22 (55.0)
Right mama	18 (45.0)
Clinical stage [*n* (%)]	
Grade 1	11 (27.5)
Grade 2	14 (35.0)
Grade 3	15 (37.5)

Estrogen receptor [*n* (%)	
Negative	11 (27.5)
Positive	29 (72.5)
Progesterone receptor [*n* (%)]	
Negative	11(27.5)
Positive	29 (72.5)
HER2/neu (human epidermal growth factor receptor) [*n* (%)]	
Score 0	9(22.5)
Score +1	12(30.0)
Score +2	8(20.0)
Score +3	11(27.5)

Table 3
Paired t-test analysis between the test and control samples before treatment

Variables		Mean	Std. Deviation	Std. Error Mean	T-value	P-value
Urea	Control	24.7000	4.94690	.78217	-30.875	.000
	Test	64.0250	7.05469	1.11544		
creatinine	Control	.9825	.25206	.03985	-47.388	.000
	Test	6.0175	.63038	.09967		
Uric acid	Control	5.0375	.78990	.12489	-12.125	.000
	Test	7.3850	.83160	.13149		
AST	Control	30.6000	7.28469	1.15181	-14.061	.000
	Test	63.0500	12.94555	2.04687		
ALT	Control	27.4000	5.80362	.91763	-17.766	.000
	Test	67.4000	13.36432	2.11308		
ALP	Control	65.2250	14.65411	2.31702	-14.263	.000
	Test	131.1500	25.43927	4.02230		
MDA	Control	1.9225	.53035	.08386	-23.593	.000
	Test	6.2825	1.14777	.18148		
TAS	Control	160.3250	47.58064	7.52316	6.590	.000
	Test	106.5500	29.11599	4.60364		
NO	Control	26.4000	6.42032	1.01514	-22.021	.000
	Test	65.1750	10.02276	1.58474		
CAT	Control	679.3750	55.74838	8.81459	24.719	.000
	Test	328.6500	80.90626	12.79240		
HB	Control	12.1375	1.12105	.17725	7.512	.000
	Test	10.3175	1.31459	.20785		
PLATELETS	Control	3.5100	.38551	.06095	7.116	.000
	Test	2.5800	.64498	.10198		
TWBC	Control	10.0375	.61590	.09738	-.238	.813
	Test	10.0750	.76519	.12099		
Neutrophils	Control	56.1250	9.29623	1.46986	.450	.655
	Test	55.7250	8.44587	1.33541		
Lymphocytes	Control	33.8750	7.73665	1.22327	4.755	.000
	Test	28.6000	7.30578	1.15514		

Table 4
Paired t-test analysis between the test and control samples during treatment

Variables		Mean	Std. Deviation	Std. Error Mean	T-value	P-value
Tumor side	Control	.5000	.50637	.08006	-.902	.372
	Test	.5750	.50064	.07916		
Urea	Control	24.7000	4.94690	.78217	-24.136	.000
	Test	58.6750	7.95625	1.25799		
creatinine	Control	.9825	.25206	.03985	-28.630	.000
	Test	3.9375	.57365	.09070		
Uric acid	Control	5.0375	.78990	.12489	-8.591	.000
	Test	6.8525	1.08060	.17086		
AST	Control	30.6000	7.28469	1.15181	-12.671	.000
	Test	59.5500	12.79413	2.02293		
ALT	Control	27.4000	5.80362	.91763	-16.760	.000
	Test	65.2750	13.45455	2.12735		
ALP	Control	65.2250	14.65411	2.31702	-10.910	.000
	Test	115.7250	19.99870	3.16207		
MDA	Control	1.9225	.53035	.08386	-24.603	.000
	Test	5.0150	.57804	.09140		
TAS	Control	160.3250	47.58064	7.52316	3.428	.001
	Test	126.9750	57.72458	9.12706		
NO	Control	26.4000	6.42032	1.01514	-22.434	.000
	Test	55.2750	9.44888	1.49400		
CAT	Control	679.3750	55.74838	8.81459	23.053	.000
	Test	371.5750	79.99548	12.64840		
HB	Control	12.1375	1.12105	.17725	3.244	.002
	Test	11.3800	1.05956	.16753		
Platelet	Control	3.5100	.38551	.06095	.496	.623
	Test	3.4725	.44949	.07107		
TWBC	Control	10.0375	.61590	.09738	-5.318	.000
	Test	10.9800	.85521	.13522		
Neutrophils	Control	56.1250	9.29623	1.46986	-2.505	.017
	Test	59.6750	7.51200	1.18775		
Lymphocytes	Control	33.8750	7.73665	1.22327	-1.281	.208
	Test	34.8750	7.71009	1.21907		

MDA- malondialdehyde (MDA);Total Antioxidant Status levels (TAS); nitric oxide concentration; catalase activity; Blood Urea; Serum Creatinine; Aspartate aminotransferase; Alanine aminotransferase; alkaline phosphatase ;Serum Uric acid; hemoglobin (Hb); platelets (Plt); Neutrophils; total white blood cell count (TWBC); Leukocytes.

Table 5
Paired t-test analysis between the test and control samples after treatment

Variables		**Mean**	**Std. Deviation**	**Std.Error Mean**	**T-value**	**P-value**
Urea	Control	24.7000	4.94690	.78217	-24.206	.000
	Test	66.5500	8.75873	1.38488		
creatinine	Control	.9825	.25206	.03985	26.222	.000
	Test	6.3950	1.25452	.19836		
Uric acid	Control	5.0375	.78990	.12489	-1.000	.323
	Test	5.0650	.76445	.12087		
AST	Control	30.6000	7.28469	1.15181	-14.317	.000
	Test	75.2000	17.85870	2.82371		
ALT	Control	27.4000	5.80362	.91763	-14.079	.000
	Test	87.6000	25.65031	4.05567		
ALP	Control	65.2250	14.65411	2.31702	-20.990	.000
	Test	68.500	14.7000	2.32500		
MDA	Control	1.9225	.53035	.08386	-28.036	.000
	Test	6.5350	.91807	.14516		
TAS	Control	160.3250	47.58064	7.52316	6.991	.000
	Test	93.8750	36.89291	5.83328		
NO	Control	26.4000	6.42032	1.01514	-29.691	.000
	Test	64.2000	7.25789	1.14757		
CAT	Control	679.3750	55.74838	8.81459	42.640	.000
	Test	211.5000	42.57723	6.73205		
HB	Control	12.1375	1.12105	.17725	16.651	.000
	Test	8.4550	.90948	.14380		
PLATELET	Control	3.5100	.38551	.06095	8.275	.000
	Test	2.4675	.62199	.09834		
TWBC	Control	10.0375	.61590	.09738	4.907	.000
	Test	9.3125	.84039	.13288		
Neutrophils	Control	56.1250	9.29623	1.46986	2.924	.000
	Test	51.6750	9.32707	1.47474		
Lymphocytes	Control	33.8750	7.73665	1.22327	4.666	.000
	Test	27.8500	6.48292	1.02504		

Table 6
Pearson Correlation for Control Vs. Before, During and After Treatment

	Before Treatment (C/T)		During Treatment (C/T)		After Treatment (C/T)	
Variable	r-value	P-value	r-value	P-value	r-value	P-value
Urea	.108	.506	.134	.410	-.212	.189
Creatinine	-.116	.476	.029	.857	-.106	.516
Uric acid	.004	.982	-.140	.389	.975	.000
AST	.043	.794	.040	.804	-.062	.703
ALT	.067	.682	.061	.707	-.133	.412
ALP	-.413	.008	.010	.952	.237	.251
MDA	-.027	.868	.191	.238	.043	.794
TAS	.330	.038	.162	.319	.003	.985
NO	.529	.000	.137	.399	.312	.050
CAT	.266	.097	.177	.273	.022	.893

HB	.084	.607	.216	.180	.063	.701
Platelet	.352	.026	-.238	.138	-.207	.200
TWBC	-.138	.395	-.032	.846	.205	.204
Neutrophils	.448	.004	.803	.000	.466	.002
Lymphocytes	.796	.000	.566	.000	.351	.026

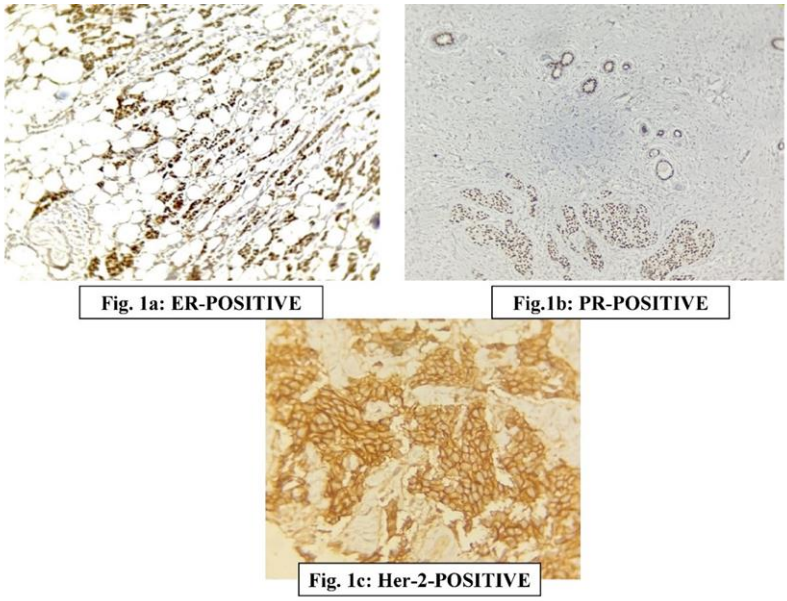

Figure 1. a-c: Photomicrograph showing strong nuclear estrogen receptor (ER), strong nuclear progesterone receptor (PR) and uniform intense membrane HER2/neuimmunoreactivity in tumor cells

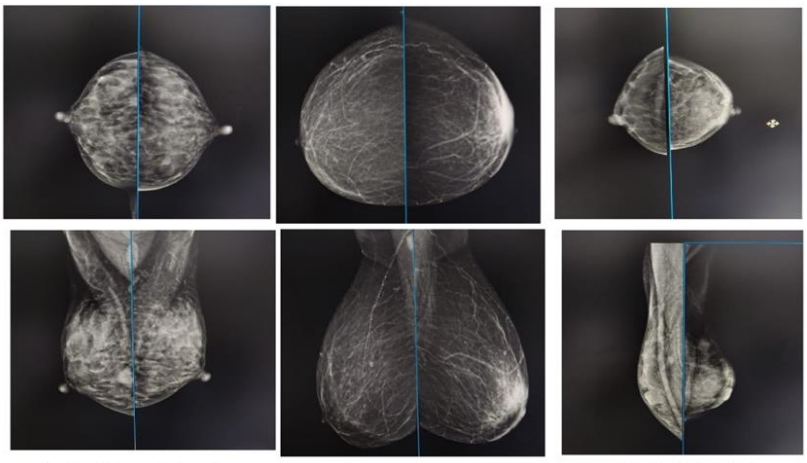

Fig. 2a: Hypoechoic lesion extending from 7'o - 8'o clock in right breast 3 cm present malignant lesion BRADS-6.

Fig. 2b: Well defined oval hypo echoic lesion from 8'o clock -9'o clock position BRADS-5. Enlarge left axillary lymph nodes with thickened cortex likely metastatic. BRADS- 4

Fig. 2c: Lobulated hyper dense lesion with multiple clusters of tiny calcific foci in left retroaerolar region. malignant lesion BRADS-6

Figure 2. a-c: Various clinical images of mammograms

Discussion

Chemotherapy continues to be the anti-cancer treatment of choice for hundreds of thousands of individuals who are diagnosed with cancer each year [24]. The most efficient anti-neoplastic therapy medications available today are cyclophosphamide, adriamycin, and 5-fluorouracil (CAF), which are recommended to millions of women worldwide for adjuvant or palliative treatment of breast cancer. The chemotherapy (AC regimen), according to the current study, has a negative impact on the haematological and biochemical profile, leading to neutropenia, thrombocytopenia, anaemia, hyperuricemia, and liver dysfunction [25]. In a similar line, our work supports these conclusions on the detrimental effects of chemotherapy on haematological and biochemical parameters. Hemoglobin increased during cycles while decreasing following treatment, according to the study, which was carried out throughout the cycles. Following treatment, there was a reduction in white blood cells (WBC), neutrophils, platelets, and lymphocytes. While after therapy, AST, ALT, and ALP improved.

Throughout the cycles, there are also considerable changes in the amounts of uric acid and blood urea creatinine. In breast cancer patients receiving chemotherapy, anaemia has been noted to occur more frequently with each dose of doxorubicin [25]. Despite the fact that haemoglobin levels varied during the chemotherapy rounds in our study, none of the individuals had anaemia. The medications given may be the cause of this variation. Doxorubicin was administered in the majority of past trials by various researchers, however in the current study, multivitamins and fersolate (ferrous sulphate) were given in addition to the drug to reduce the risk of anaemia in our patients. The WBC levels seen in our study's third cycle support the findings of other studies [26, 27]. Oxidative stress is an essential factor in cancer devolvement and a key factor for disease evolution [28]. Increased oxidative stress has been linked to several cancer types, including breast cancer, according to earlier research [29]. The ability to cause genetic alterations in antioxidant enzymes, oestrogen treatment, excessive production of reactive oxygen species, as well as a compromised antioxidant system, are anticipated causes for increased oxidative stress in breast cancer [30].

Lipid peroxidation can produce reactive and hazardous compounds, and studies have suggested that this process may play a role in the development of tumours. Proteins, DNA, and other biomolecules can react with MDA, one of the most prevalent and crucial aldehydes of lipid peroxidation, changing their structure and function. MDA assessment of tissues and plasma has been widely employed in several malignancies, including breast cancer, in recent years [31, 32, 33]. Our results confirmed the widely held belief that elevated MDA levels are associated with breast cancer. The findings demonstrated that, when compared to healthy persons, breast cancer patients have significantly higher serum levels of MDA. Since the majority of the patients in our patient group had just received a breast cancer diagnosis, the elevated level of oxidative agents may be indicative of the early stages of cancer development. The aggregation of ROS, which could lead to significantly heightened cellular lipid peroxidation, may be the cause of increased MDA levels in the serum of breast cancer patients.

Repeated polychemotherapy with radical-generating substances caused cancer patients to have significantly lower antioxidant capacities, which resulted in oxidative stress in a different study by Look and Musch [34]. Patients with breast cancer may experience increased cytotoxic activity as a result of elevated TBARS concentrations and decreased TAS in treatment. Therefore, controlling MDA and TAS production is crucial for reducing breast cancer. We found it fascinating that there was a substantial difference in MDA levels before, during, and after the treatment across the three groups when we compared our data at different phases, such as II and III. Besides, these patients had dramatically reduction in the amount of TAS. In the current study, the levels of TAS were found to significantly decrease after treatment than before and during after treatment of breast cancer patients when compared with controls.

Apoptosis can be induced or inhibited by the short-lived, extremely reactive free radical nitric oxide. Nitric oxide levels were observed to be significantly greater in the current study's stage III and stage IV postoperative breast cancer patients than they were in healthy controls both before and throughout chemotherapy (p 0.0001). In serum samples from patients with breast cancer, several researchers have shown higher nitric oxide levels at an operable stage [35, 36]. In our study, catalase activity is significantly decreased after treatment than before, during treatment of breast cancer patients.Our results indicated that enhanced liver enzymes AST and ALT after AC-T treatment($P<0.05$), which evidenced that Serum levels of GOT and GPT are used commonly as parameters of hepatic damage as intracellular enzymes are released into the blood after hepatic injury [37].

Due to a high rate of cell turnover (tumourlysis syndrome), hyperuricemia during cancer treatment results in renal impairment [38]. Even while uric acid levels rose during the treatment cycles, the rise was insufficient to have a noticeable impact on renal function. When compared to before and for controls in the current investigation, uric acid levels were significantly higher ($P<0.05$) after and during the treatment. The majority of chemotherapy drugs impact normal cells instead of just cancer cells. It causes a wide range of negative side effects in almost all body tissues, including alopecia, exhaustion, widespread rash, diarrhoea, and dizziness.

Conclusion

The current investigation came to the conclusion that haematological parameters are out of whack in breast cancer patients. In patients with breast cancer, we found anaemia, neutropenia, lymphocytopenia, and a low monocytic count. Hematological tests are a crucial component in treating and monitoring patients with breast cancer. It can be used to evaluate the course of a disease and the behaviour of various cancers. The increased circulation of oxidative and nitro radicals and a general deterioration of cellular redox equilibrium are signs of oxidative stress, which occurs in a variety of malignancies and results in carcinogenesis. The administration of antioxidants may increase the effectiveness of the treatment because many chemotherapy medications for cancer generate oxidative stress, which can impair anti-neoplastic activity. According to our findings, chemotherapy causes a certain amount of systemic oxidative stress, which is sustained throughout subsequent clinical treatments and may have an

impact on the patients' clinical outcomes. The main factors contributing to the spread of breast cancer are increased lipid peroxidation and oxidative stress. Our results generally support the importance of endogenous antioxidants in the genesis of breast cancer at all anticipated risk levels.

Ethics approval and consent to participate

The Ethics Committee of Omega Cancer Hospital, Visakhapatnam, India reviewed and approved this study.

Human and animal rights

No animals were used in this research. All human research procedures were followed by the ethical standards of the committee responsible for human experimentation (institutional and national) and the Helsinki Declaration of 1975, revised in 2013.

Consent for publication

The authors had taken the written informed consent from the patients.

Availability of data and materials

The authors declare that the data used in the present study were collected from the Omega Cancer Hospital, Visakhapatnam. The study doesn't use any licensed material or any previously published material. It is purely the authors' work.

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Conflict of interest

The authors have no conflicts of interest, financial or otherwise.

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