RESEARCH PAPER Asian Journal of Bio Science, Vol. 4 Issue 2 : 221-225 (October, 2009 to March, 2010)

Bio-chemical adjustment in patients with breast cancer

P. SENTHAMILSELVAN[1] AND V. SIVAKUMARI[2]
[1]Marudu Pandiyar College, THANJAVUR (T.N.) INDIA
[2]Department of Environmental and Herbal Sciences, Tamil University, THANJAVUR (T.N.) INDIA

(Accepted : August, 2009)

Breast cancer is one of the most common cancers in developed and developing countries. In India, it is the second most frequent cancer, which is estimated to be 19.3% of total female cancer with age standardized rate 17.5 per 100,000.The exact cause of breast cancer is not completely known but presumably it represents a complex interplay of genetic susceptibility and environmental factors. Hematology, lipid and lipoproteins have also been associated with risk of breast cancer. The present study was carried out to investigate Total Cholesterol (TC), High Density Lipoprotein Cholesterol (HDL-C), Low Density Lipoprotein Cholesterol (LDL-C), Triglycerides' (TG), sodium, chloride, potassium, SGOT, SGPT, glucose, urea, uric acid and creatinine in female with breast cancer. In the study revealed significant difference between breast cancer and normal subject with respect to most of the biochemical parameter analyzed.

Key words : Total Cholesterol, SGOT, SGPT, Breast cancer.

INTRODUCTION

Breast cancer is a growth of abnormal cells, usually within the ducts (which carry the milk to nipple) or lobules (glands for milk production) of the breast. In more advanced stages of the disease, these out-of-control cells invade nearby tissues or travel throughout the body to other tissues or organs. More than 178,000 women and 2,000 men are diagnosed each year with breast cancer, according to the American Cancer Society (ACS). For women, this rate is second only to non-melanoma skin cancer (Aboud –Pirak *et al.*, 1990).

Normal cell function requires these building blocks to divide and also to die when they grow old - allowing for new cells to take their place in an organized manner. When old cells do not die and the body continues to create new cells it does not need, a mass of cells form a growth or tumor. These tumors do not always signal caner, particularly in the breast (Harris, 1992).

Breast cancer begins with a growth of abnormal cell within the breast tissues. The type of breast cancer is determined by where the cancer began - in the ducts, the lobules or other areas, such as the connective tissue or in the blood vessels. It is also important to determine if the cancer has spread beyond the ducts or lobules and invaded nearby lymph nodes (Therwath, 1994).

Thus, when breast cancer starts to spread, the most common first location is the nearby lymph nodes. If breast cancer has spread to the axillary lymph nodes (located in the underarm region of the body), it can cause swelling of these nodes. After the cancer cells have spread to the lymph nodes it is more likely that the cancer will spread to other areas as well, such as the lungs, bones or brain (Jonrup, 1995).

Breast Cancer is the most frequently diagnosed malignancy in women world wide accounting for 23% of all cancer cases in women with 1.15 million in world wide. A thorough improvements in breast cancer detection treatment contribute to declining breast cancer – specific mortality rates over the last decade, the efficacy of standard systemic therapy is suboptimal ; women still experience relapses despite state –of-the-art adjuvant chemotherapy and hormonal therapy and distend metastic disease most frequently chemotherapy with novel, molecularly targeted therapeutics. However, is showing particular promise. The present study was undertaken to analyze various biochemical parameters such as hematology, lipid profile, kidney function, electrolytes glucose and uric acid in breast cancer patients.

MATERIALS AND METHODS

10 Breast cancer women subjects were taken up for the present study with the age group of 30 – 50 years. These subjects were selected on the basis of oral questionnaire from Thanjavur Medical College (TMC), Thanjavur District. Each people was subjected to detailed interrogation with special reference to marital status and duration of mother feeding. Equal number of women subjects with normal physical and mental health were selected to serve as normal subjects Table 1 shows the case history of perform/ questionnaire for breast cancer

subject.

Specimen collection:

Blood sample was obtained by various arm puncture with EDTA and with out EDTA tube. Blood and serum were used for analysis of various biochemical parameters (Table 2).

Table 1 : Preformed/questionnaire for breast cancer subjects

Sr. No.	Character	Normal subject	Breast cancer subject
	Total no. of subjects	10	10
1.	Sex	Female	Female
2.	Age	30 – 50	30 - 50
3.	Location	TMC	TMC
4.	Period of suffering	Nil	5 – 10 Yrs
5.	Marital status	Married	Married
6.	Type of abnormality	Normal breast	Abnormal of breast stage
7.	Food habits	Non veg /Veg	Non veg /Veg
8.	Stable food	Rice	Rice
9.	Source of drinking water	Bore well	Bore well
10.	Habits		
	i). Tea/coffee	Both	Both
	(No% of cups)	(2-4)	(5 – 8)
	ii). Mother feeding after birth	8 month	8 month

Table 2 : Biochemical parameters

Subjects	Parameter
	HEMATOLOGY
	Hb
	ESR
	TC
	DC
	PCV
Normal	TWBC
	TRBC
	BIOCHEMICAL
	SUGAR
	UREA
	Ureic acid
	Lipid profile
	TG
	HDL
	VLDL
Breast cancer	LDL
	RF_1
	RF_2
	SGOT
	SGPT
	ELEECTEOLYTES
	Sodium
	Potassium
	chloride

The biochemical parameters *viz.*, Hemoglobin (Seeley *et al.*, 1989), Platelet (Nowell *et al.*, 1967), WBC (Henderson, 1991), leucocytes (Weber and Lea, 1966), erythrocytes (Seeley *et al.*, 1989), Packed Cell Volume (PCV) (Grunberg, 2002), glucose (Groenwald *et al.*, 1990), urea (Burti's and Ashwood, 1994), creatinine (Beutler, 1971), uric acid (Beare and Myers, 1990), bilirubin (Baird *et al.*, 1991), cholesterol (Ramaswamy *et al.*, 2000), triglycerides (Shonk and Boxer, 1964), Serum Glutamate Oxaloacetate Transaminase (SGOT) (Bobak *et al.*, 1989), Plasma chloride (Chipps *et al.*, 1992), potassium (Corinne *et al.*, 1982), sodium (Dacie and Lewis, 1994), Serum Glutamate Pyuvate Acetate Transaminase (SGPT) (Garber, 1991) were analysed. The results were presented as mean ± SD. Data were statistically analyzed using student "t" test. P value set a lower than 0.001, 0.01, 0.05 were considered as statistically significant.

RESULTS AND DISCUSSION

The present study was carried out to analyze the various biochemical parameters in normal and breast cancer subjects. The observation made on different subjects of normal and breast cancer was compared. Table 3 shows the level of glucose, RBC, WBC, neutrophiles and lymphocytes in normal and breast cancer subjects. Hemoglobin (Hb), WBC and neutrophiles increased significantly, while, RBC and lymphocytes not increased significantly in breast cancer subjects when compared to normal subjects.

Table 4 shows the level of eosinophil, monocytes, basophile, E.S.R (1/2h) and E.S.R (1h) in normal and breast cancer subjects. Eosinophil, monocytes, basophile and E.S.R (1/2h) were not increased, significantly E.S.R (1h) increased significantly in breast cancer subjects when compared to normal subjects.

Table 5 shows the level of P.C.V, glucose, urea, uric acid and creatinin in normal and breast cancer subjects. P.C.V, glucose, urea, uric acid and creatinin were not increased significantly in breast cancer subjects when compared to normal subjects.

Table 6 shows the level of total cholestrol, HDL-cholestrol, LDL- cholestrol, VLDL and triglyceridein normal and breast cancer subjects. Total cholestrol, HDL-cholestrol, LDL- cholestrol, VLDL and triglyceride significantly increased in breast cancer subjects when compared to normal subjects. Increased level of l i p i d

Table 3 : The level of hemoglobin, RBC, WBC, neutrophiles and lymphocytes in normal and breast cancer

Subject	Hemoglobin (g%)	RBC	WBC	Neutrophiles(g%)	lymphocytoes(g%)
Normal	16.09± 1.98	3.04 ± 1.08	3,744 ± 632.77	52.41 ± 2.31	25.14 ± 3.10
Brest Cancer	9.16 ± 1.97*	3.37 ± 0.50*	1192 ± 279.25*	65.61± 3.58*	28.6± 3.91*

The values expressed as mean ± SD
*Not significantly different from normal subjects

Table 4 : The level of eosinophil, monocytes, basophile, E.S.R (1/2h) and E.S.R (1h) in normal and breast cancer

Subject	Eosinophil (g%)	Monocytes(g%)	Basophile (g%)	E.S.R (1/2h) (mm/h)	E.S.R (1h) (mm/h)
Normal	4.74 ± 0.72	0.7 ± 0.14	0-1%	18.42 ± 8.40	44.61 ± 1.74
Brest cancer	5.2± 0.52*	0.6± 0.5*	0%*	17.6 ± 14.70*	51.4 ± 2.97*

The values expressed as mean ± SD
*Not significantly different from normal subjects

Table 5 : The level of P.C.V, glucose, urea, uric acid and creatinin in normal and breast cancer

Subject	P.C.V	Glucose (mg/dl)	Urea (mg/dl)	Uric acid (mg/dl)	Creatinin (mg/dl)
Normal	28.64 ± 1.87	94.96±13.65	29.44± 5.19	4.21 ± 1.04	0.93± 0.35
Brest cancer	32.4 ± 2.97*	141 ± 71.42 *	36.2± 9.20*	3.6± 0.8*	1.3± 14 *

The Values expressed as mean ± SD
*Not significantly different from normal subjects

Table 6 : The level of total cholestrol, HDL- cholestrol, LDL- cholestrol, VLDL and triglyceride in normal and breast cancer

Subject	Total cholestrol (mg/dl)	HDL- cholestrol (mg/dl)	LDL- cholestrol (mg/dl)	VLDL cholestrol (mg/dl)	Triglyceride (mg/dl)
Normal	191.82± 2.15	43.97±6.27	163.77 ± 18.48	24.08 ± 4.22	119.81 ± 2.05
Brest cancer	182± 2.7*	30± 11.06*	110.6 ± 27.44*	32.04 ± 3.5*	160.2 ± 3.5*

The values expressed as mean ± SD
*Not significantly different from normal subjects

profile such cholesterol, triglyceride, HDL, VLDL, LDL-cholesterol and decreased level of HDL-C were absorbed in breast cancer as compared to normal subjects. This may indicate the sex hormone that alter the lipid metabolism in breast cancer.

Table 7 shows the level of risk factor1 (RF1), risk factor 2(RF2), sodium and potassium in normal and breast cancer subjects. Risk factor 2(RF2) and potassium significantly increased, while Risk factor1 (RF1) and sodium not increased significantly in breast cancer subjects when compared to normal subjects.

Table 8 shows the level of chloride, SGOT and SGPT normal and breast cancer subjects. Chloride increased significantly, while SGOT and SGPT not increased significantly in breast cancer subjects when compared to normal subjects.

The above result confirms that the breast cancer patients increase the risk of cancer. Preventive therapy including chemotherapy and radiation therapy are used to decrease the prognosis of breast cancer. Fats being insoluble in water, their transport in aqueous media the blood plasma become a problem. This is made possible by formation of water miscible lipoproteins that are combinations of lipids with water miscible proteins. The major lipids in plasma namely, triglycerides, cholesterol and phospholipids are combined with specific apoproteins to form complexes called lipoproteins and circulate in blood in combination with plasma proteins, they are synthesized in the intestines and in liver. Their main function is transport of lipids from the intestines and liver to extra

Table 7 : The level of risk factor1 (RF1), risk factor 2(RF2), sodium and potassium in normal and breast cancer

Subject	RF1 (mg/dl)	RF2 (mg/dl)	Sodium (mmol/L)	Pottasium (mmol/L)
Normal	4.41 ± 0.63	1.56 ± 0.057	138.79 ± 7.05	5.92± 1.05
Brest cancer	4.55 ± 0.62*	2.82 ± 0.62*	135.8 ± 24.36*	3.62 ± 0.66*

The values expressed as mean ± SD
*Not significantly different from normal subjects

Table 8 : The level of chloride, SGOT and SGPT in normal and breast cancer

Subject	Chloride (mmol/L)	SGOT (IU/L)	SGPT (IU/L)
Normal	104± 4.21	33.55 ± 6.50	36.36 ± 6.43
Brest cancer	73.6 ± 3.7*	36 ± 8.9*	42.4 ± 10.64*

The values expressed as mean ± SD
*Not significantly different from normal subjects

hepatic tissue where they can be stored or oxidized for energy (Ambile and Shanmugam, 1999).

The underlying cause of cardio vascular diseases involving a complex array of circulating lipoproteins and cells and their interactions with the cell and metric proteins of the arterial wall. It is well established that high circulatory serum cholesterol, LDL-C and low level of circulating HDL-C are the main causative of this disease (Sabari *et al.*, 2002). Excess cholesterol can accumulate and form deposits, called plaques on artery wall. This leads the atherosclerosis (hardening of the arteries). As plaques continue to grow, blood flow in affected arteries may slow (or) even stop. There is also a risk that a plaque can form bigger local clotting (or) thE parts of plaque can become dislodged leading AS an ischemic event.

Triglycerides are composed of fatty acid and glycerol like cholesterol, they circulate in the blood but are stored in body fat. When a fatty meal is eaten triglyceride (and glucose) levels increase significantly. Gradually as the body processes efficiently, the level of triglyceride will decrease (Ohlsen, 2004).

The increase in serum cholesterol level was attributable mainly to an increase in cholesterol in the LDL fraction. This observation is consistent with the finding that age related hyper cholesterolemia was caused by LDL and HDL. Serum LDL cholesterol levels are known to be regulated by receptor mediated clearance of the lipoprotein (Ginsberg, 1998) so the increase of serum LDL-C is thought to be caused by the reduction of catabolic pathways. High levels of LDL cholesterol show a positive correlation with atherosclerosis whereas high level of HDL cholesterol have a negative correlation. HDL inhibits the uptake of LDL by the arterial wall and aueilitates the transport of cholera I born perbheral tissue to the liver where it catabolised and excretes from the body (Buring *et al.*, 1992).

Human mammary tissue metabolizes lipids from plasma effected by female gondola hormones malignant proliferation of breast tissue in women has been associated with changes in plasma lipoid and lipoprotein levels. In the present studies elevated level of total cholesterol has been observed in breast cancer patients overall elevated total cholesterol was highly significant in breast cancer of women than control group as also reported by Mady (2000) and Ray *et al.* (2000). HDL-C level way significantly decreased in breast cancer patients. Present observations medicate that patients with breast cancer have significantly lower concentration of HDL-C than the patients having advanced diseases.

The human haemopietic system is extremely sensitive to some environmental influence because of the rapid synthesis and destruction of cell with consequent heavy metabolic demands. Hematological parameters are a sensitive index to change in ecological conditions and can constitute an important diagnostic tool in poxicologyics studies (Allistur, 2001).

REFERENCES

Aboud-Pirak, E., Hurwitz, E., Pirak, M.E., Bellot, F., Schlessinger, J. and Sela, M. (1990). Efficacy of antibodies to epidermal growth factor receptor against KB carcinoma in vitro and in nude mice. *J. Natl. Cancer. Inst.*, **80**: 1605-1611.

Allistur, A.H. (2001). Glutathione and its metabolizing enzymes in patients with different benign and malignant diseases. *Clin. Biochem.*, **33** (8): 657-62.

Ambile Shanmugam, N. A. (1999). Nutrition dietary factors and breast cancer. *Indian J. Cancer*, **31**:218-285.

Baird, S.B., McCorkle, R. and Grant, M. (1991). Cancer nursing: a comprehensive textbook, Philadelphia, WB Saunders.

Beare, P.G. and Myers, J.L. (1990). Principles arid practice of adult health nursing, St. Louis, Mosby- Year Book.

Beutler, E. (1971). Red cell metabolism: A manual of biochemical methods. Grune and Stration. New York. 103-5.

Bobak, I.M., Jensen, M.D. and Zalar, M.K. (1989). *Maternity and Gynecologic Care: the nurse and the family*. (Ed.4), St. Louis, Mosby-Year Book.

Buring, A.U., Ibrahim, N.K. and Francis, O. (1992). Significantly higher pathologic complete remission rate after neoadjuvant therapy with trastuzumab, paclitaxel, and epirubicin chemotherapy: results of a randomized trial in human epidermal growth factor receptor 2-positive operable breast cancer. *J. Clin. Onco1.*, **23**: 3676-3685.

Burti's, C.A. and Ashwood, E.R. (1994). *Tietz textbook of clinical chemistry*. Second edition, W.B. Saunders Company, Philadelphia pp. 814-15,831,848-49,897-99.

Chipps, E., Clanin, N.J. and Campbell, V.G. (1992). *Neuro- logic disorders*, St. Louis, Mosby-YearBook.

Corinne H., Robbinson, M., Marilyn, N. and Laweley, R. (1982). *Lipids Metabolism and Dietary Fats, Normal and Therapeutic Nutrition*. 16th Ed. 105-109.

Dacie, J.V. and Lewis, S.M. (1994). *Practical Haematology*. Churchill Livingston, Edinburgh, (8th Ed.). pp. 49-82.

Garber, J.E. (1991). Familial Aspects of Breast Cancer, *in Breast diseases*, (2ndEd.) Harris, J.R. Philadelphia, Lippincott, p. 142. Endocrine factors.

Ginsberg, R.S. (1998). Review of the comparative pharmacology and clinical activity of cisplatin and carboplatin. *J. Clin. Onco.*, **17**: 409-422.

Groenwald, S.L., Frogge, M.J., Goodman, M. and Yarbro, C.H. (1990). *Cancer nursing: principles and practice, Boston*, Jones & Bartlett.

Grunberg (Ed.). (2002). Proceedings of the 38th Annual Meeting of the American Society of Clinical Oncology, Orlando, FL. Baltimore, MD, Lippincott Williams & Wilkins, 18-21.

Harris, C. (1992). Effects of coping style and relaxation on cancer chemotherapy side effects and emotional responses. *Oneol. Nurs. Forum.*, **13**(5):308-315.

Henderson, I.C. (1991). *Endocrine Therapy of Metastatic Breast diseases*. (2nd Ed.), Harris, J.R. *et al.* (Eds). Philadelphia, Lippincott. p. 559.

Jonrup, I. L.R. (1995). New developments and approaches in the *platinum arena*. *Drugs*, **59**(4): 29-36.

Mady, M. (2000). Platinum compounds in the treatment of advanced breast cancer. *Clin. Breast. Cancer*, **2**: 190-208.

Nowell, P.C., Morris, H.P. and Potter, V.R. (1967). Chromosomes of "Minimal Deviation" hepatomas and some other transplantable rat tumors. *Cancer Res*., **27**: 1565-1579.

Ohlsen, A.B. (2004). Canadian national breast screening study: I. Breast cancer detection and death rates among women aged 40-49 years. *Canadian J. Med. Assoc*., **147**:1459.

Ramaswamy, G., Rao, V.R, Krishnamoorthy, L., Ramesh, G., Gomathy, R. and Renukadevi, D. (2000). Serum levels of bone alkaline phosphatase in breast and prostate cancer with bone metastasis. *Indian J. Clin. Biochem*., **15** (2) : 110-13.

Ray, A., Jain, D., Yadav, R., Naik, S.L., Sharma, S., Bhadur, A.K. and Sharma, B.K. (2000). Effect of cancer treatment modalities on serum lipid and lipoprotein among women with carcinoma of breast. *Indian J. Physiol. Pharmacol.*, **45**: 337-44.

Sabari, M.M., Dosoky, I., Horia, H. and Khalaf, A.H. (2002). Elevation of glutathione, glutathione reeducates and nucleic acids in both normal. tissues and tumor of breast cancer patients. *Cancer Leu.*, **72**: 11-15.

Seeley, P., Mangal, D., Ritzke, C., Rossler, N., Kirsch, C.M. and Eiermann, W. (1989). Significance of bone alkaline phosphatase. CA 15-3 and CEA in detection of bone metastasis during follow up of patients suffering from breast carcinoma. *Eur. J. Clin. Chem. Clin. Biochem.*, **30**: 809-14.

Shonk, C. E. and Boxer, G. E. (1964). Enzyme Patterns in Human Tissues. I. Methods for the Determination of Glycolytic Enzymes. *Cancer Res.*, **24**: 709-721.

Therwath, C.L. (1994). Efficacy and safety of trastuzumab as a single agent in first-line treatment of HER2-overexpressing metastatic breast cancer. *J. Clin. Onco.*, **20**: 719-726.

Weber, G. and Lea, M. A. (1966). The Molecular correlation concept of neoplasia. In: G. Weber (Eds.). *Advances in Enzyme Regulation* Vol. 4, 115-145. London: Pergamon Press, Ltd.