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CLINICAL, HISTOPATHOLOGICAL, HORMONAL EVALUATION AND CHROMOSOMAL ABERRATIONS STUDY IN 50 CASES OF CARCINOMA BREAST

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ABSTRACT

Background - Carcinoma breast is most common cancer in urban indian women. The focus of our study was to evaluate the clinical and histopathological type of breast cancer, the hormonal status, chromosomal aberrations frequently encountered, and any statistical correlation between hormonal and chromosomal aberrations. Material and Methods - Fifty cases of carcinoma breast admitted in our institute between June 2009 - December 2011 were studied. Results - Presenting symptom in 44 (88%) patients was breast lump. Invasive ductal carcinoma was the most common (98%) histopathological type. Majority of the patients (64%) were in stage II followed by stage I (20%). In hormonal study, majority of the cases (48%) were double positive i.e ER+, PR+, Her2neu - 18% were triple negative and 16% triple positive. Most common chromosomal aberrations were - 1q (chromatid break, chromosomal break, chromatid gap). Statistically, there was no correlation between hormonal study and chromosomal aberrations.

Key Words: Carcinoma Breast, Histopathology, Hormonal Evaluation, Chromosomal Aberration

INTRODUCTION

Carcinoma breast is most common cancer urban indian women accounting for >30% of all female cancers; National cancer registry (2001). Worldwide, Weiss *et al.*, (2005)² it is the second most common after lung cancer and the fifth most common cause of cancer death. Clinically, Norman *et al.*, (2008) presentation varies between breast lump, tenderness, skin irritation, dimpling and nipple discharge/pain/ulceration or retraction. Pathologically, Townsend *et al.*, (2008) chief forms of carcinoma breast are classified as: (a) Non invasive epithelial - DCIS, LCIS (b) Invasive epithelial - lobular, ductal (c) Mixed connective and epithelial tumours. Genetics play an important role in development of carcinoma breast. Mutations, Venkitaraman (2002) in either BRCA1, or BRCA2 confers a life time risk between 60 – 85%. However, these mutations account for 2% - 3% of all breast cancer.

MATERIALS AND METHODS

The study was conducted on 50 patients of carcinoma breast at Sri Guru Ram Das Institute of Medical Sciences and Research in conjunction with Guru Nanak Dev University from June 2009 – Dec 2011. All patients with cytological and/or histopathological proof were included in the study. Benign breast disease, recurrent carcinoma and patients with distant metastasis were excluded from the study. Patients were subjected to routine hematological investigation, LFT's, mammography and FNAC. The tissue sample for hormonal study was taken from excised specimen and sent for ER, PR and Her2neu evaluation by Immuno - histochemical assay using computer based image analyzer in paraffin embedded tissue. For chromosomal aberrations study, Peripheral blood lymphocyte culture was sent in Rosewell Park memorial Institute media.

RESULTS

Carcinoma breast was found commonly in 46 - 55 yrs of age group. Incidence is more in post menopausal women (56.25%) than in premenopausal. Only in two patients (4%) family history was present. Presenting symptom of 44 (88%) patients was breast lump, whereas 4 had breast pain and 2 had nipple

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discharge. Left breast (upper outer quadrant) had a higher preponderance (54%) than right breast. Invasive ductal carcinoma was the most common (98%) histopathological type. Majority of the patients (64%) were in stage II followed by stage I (20%). In hormonal study, majority of the cases (48%) were double positive i.e ER+, PR+, Her2neu - . 18% were triple negative and 16% triple positive.

Hormonal Status	E+P+H+	E+P+H -	E - P - H -	E+P - H -	E - P - H+	E - P+H+	E+P - H+	E - P+H -
No. of Patients	8	24	9	7	0	0	2	0
%	16	48	18	14	0	0	4	0

Various chromosomal aberrations were found as premature centromere division, chromatid break, acentric fragment, chromosomal break, terminal deletion, chromatid gap, dicentric chromosome, complete centromere separation. Most common chromosomal aberrations were - 1q (chromatid break, chromosomal break, chromatid gap); 2q (chromosomal break, terminal deletion, chromatid gap); 1p, 12q (chromosomal break); 3q (terminal deletion) and 6p (chromatid gap). It was not possible to grow chromosomal study culture in 20 patients. Out of the remainder 30 patients, 9 patients belonged to uncommon hormonal groups, therefore comparison between the most common hormonal groups and chromosomal aberrations was done in 21 patients.

Table 1: 15 patients had chromosomal aberrations in form of premature centromere division (pcd)

ER/PR/Her2neu	PCD Involved	PCD Not Involved	Total
+/+/-	4	7	11
-/-/-	3	2	5
+/+/+	2	3	5
Total	9	12	21

p Value=0.668

Table 2: 17 patients had chromosomal aberrations in form of chromatid break

ER/PR/Her2neu	Chromatid Break (chtb) Involved	Chtb Not Involved	Total
+/+/-	7	4	11
-/-/-	3	2	5
+/+/+	3	2	5
Total	13	8	21

p value=0.761

Table 3: 4 patients had chromosomal aberrations in form of chromosomal break

ER/PR/Her2neu	Chromosomal Break (chrb) Involved	Chrb Not Involved	Total
+/+/-	2	9	11
-/-/-	0	5	5
+/+/+	2	3	5
Total	4	17	21

p value=0.272

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Table 4: 20 patients had chromosomal aberrations in form of terminal deletion

ER/PR/Her2neu	Terminal Deletion (ter del) Involved	(ter del) Not Involved	Total
+ / + / -	5	6	11
- / - / -	4	1	5
+ / + / +	4	1	5
Total	13	8	21

p value=0.266

Table 5: 19 patients had chromosomal aberrations in form of chromatid gap

ER/PR/Her2neu	Chromatid gap (chtg) Involved	Chromatid gap (chtg) not involved	Total
+ / + / -	6	5	11
- / - / -	3	2	5
+ / + / +	3	2	5
Total	12	9	21

p value=0.969

Therefore, statistically there was no correlation between hormonal study and chromosomal aberrations.

DISCUSSION

Post menopausal females were more commonly affected in our study. Similar results were there in other studies, Verkasolo *et al.*, (2001) Corbould *et al.*, (1998). Our patients were mostly in stage II>I>III as against Bhattacharya *et al.*, (2006) in which maximum patients were in stage IIIB>IIIA>IV. Similar to other studies, Bhattacharya *et al.*, (2006) Newton *et al.*, (1999) most common complaint of our patients was lump breast and most common site was left upper outer quadrant in consistent with other studies, Sandhu *et al.*, (2010) Cheung *et al.*, (2011). As in other studies, Malik *et al.*, (2010) Kari *et al.*, (2011) the most common pathological variant was infiltrating ductal carcinoma. Most common chromosome affected by structural aberration in our study was chr no. 1 followed by 2, 3, 6, 12. Majority of the studies, Thompson *et al.*, (1993) Cervantes *et al.*, (1996) Malamou *et al.*, (1999) also found involvement of chr no 1, 3, 6. We found no statistical correlation between triple positive/triple negative/double positive and chromosomal aberrations.

CONCLUSION

In our study the most common chromosomal aberrations are found on chromosome no. 1, 2, 3, 6, 12 and there is no statistical correlation between triple positive/triple negative/double positive (ER+, PR+, Her2neu -) and chromosomal aberrations. In literature, we have not come across any study comparing the two.

REFERENCES

Bhattacharya S and Adhikary S (2006). Evaluation of risk factors, diagnosis and treatment in carcinoma breast - A retrospective study. *Kathmandu University Medical Journal* 4(1) 54-60.

Research Article

Cervantes M and Glassman AB (1996). Breast cancer cytogenetics: a review and proposal for clinical application. *Annals of Clinical and Laboratory Science* **26**(3) 208-214.

Cheung K, Leung F and Khoo U (2011). Palpable asymmetrical thickening of breast: a clinical, radiological, and pathological study. *The British Journal of Radiology* **74**(2) 402-406.

Corbould AM, Judd SJ and Rodgers RJ (1998). Expression of types 1, 2 and 317-beta-hydroxysteroid dehydrogenase in subcutaneous abdominal and intra-abdominal tissue of women. *Journal of Clinical Endocrinology and Metabolism* **83**(1) 187-194.

National Cancer Registry ten year consolidated report of Hospital based cancer registries 1984-1993, an account of burden and care of cancer patients (2001). New Delhi: Indian Council of Medical Research.

Kari D and Jill S. What are the different types of breast cancer. *Breast and Ovarian Cancer*. (Online) Available: <http://www.genetic health.com> [Accessed 5 September 2011].

Malamou-Mitsi VD, Syrrou M, Georgiou I, Pagoulatos G and Agnantis NJ (1999). Analysis of chromosomal aberrations in breast cancer by comparative genomic hybridization. Correlation with histoprognostic variables and c-erbB-2 immunoexpression. *Journal of Experimental and Clinical Cancer Research* **18**(3) 357-361.

Malik AM, Pathan R, Shaikh NA, Quereshi JN and Talpur KA (2010). Pattern of presentation and management of ca breast in developing countries. *Journal of Pakistan Medical Association* **60**(9) 718-21.

Newton P, Hannay DR and Laver R (1999). The presentation and management of female breast symptoms in general practice in Sheffield. *Family Practice* **16**(4) 360-366.

Norman SW and Christopher JK (2008). Bailey and love's short practice of surgery 25th edition, edited by Hodder Education 837-847.

Sandhu DS, Sandhu S, Karwasra RK and Marwah S (2010). Profile of breast cancer patients at a tertiary care hospital in north india. *Indian Journal of Cancer* **47**(1) 16-22.

Townsend CM, Beauchamp RD, Evers BM and Mattox KL (2008). Sabiston Textbook of Surgery. Vol 2, 18th edition, edited by Saunders Elsevier 842-845.

Thompson F, Emerson J, Dalton W, Yang JM, McGee D and Bhattacharya (1993). Clonal chromosome abnormalities in human breast carcinomas, twenty eight cases with primary disease. *Genes, Chromosome, Cancer* **7**(4) 185-193.

Verkasolo PK, Thomas HV, Appleby PN, Davey GK and Key TJ (2001). Circulating levels of sex hormones and their relation to risk factors for breast cancer: a cross-sectional study in 1092 pre and post - menopausal women (United Kingdom). *Cancer Causes and Control* **12**(1) 47-59.

Venkitaraman AR (2002). Cancer susceptibility and the functions of BRCA1 and BRCA2 Cell. *Cell Press Journal* **108**(2) 171-182.

Weiss JR, Moysich KB and Swede H (2005). Epidemiology of male breast cancer. *Cancer Epidemiological Biomarkers Preview* **14**(1) 20-26.