

Digital Dermal Patterns in Carcinoma of Breast

P.E. Natekar*, F. M. DeSouza*, D.D. Motghare** and A.K. Pandey***

* *Department of Anatomy*, ** *Department of P.S.M.*, *** *Department of Radiotherapy.*
Goa Medical College, Bambolim, Goa, India

KEYWORDS Finger prints, Dermatoglyphics, Neoplastic disease, Breast cancer.

ABSTRACT Finger print patterns of 100 female patients of cancer of breast were compared with 100 matched controls accordingly. A pattern of six or more digital loops were identified more frequently in women with breast cancer than those without the disease ($p < 0.01$). Women with more than six loops in their fingerprint pattern were 46 times more at risk of having carcinoma of breast as compared to controls, the OR ratio was 46.58, 95% C.I.=15.78-137.49. The presence of >6 whorls are also significantly associated negatively with carcinoma of breast ($\chi^2 = 16.61$, d f=1, $p < 0.01$). It appears that >6 whorls have a protective effect for carcinoma of breast. (OR=0.1319, 95% C.I. = 0.035-0.49). These findings suggest that the digital dermatoglyphics may have a future role in identifying women at increased risk for breast cancer.

INTRODUCTION

The study of dermatoglyphics plays an important role in the diagnosis of chromosomal disorder. Abnormal dermatoglyphic patterns have been observed in several non chromosomal and other diseases whose etiology may be influenced directly or indirectly by genetic inheritance. A study by Holt (1970) reveals its significance in Down syndrome. Holt and Lindstein (1964) show the importance of dermatoglyphics in Turners syndrome, Schauman and Alter (1976), prove its involvement in Trisomy 18. Genetic predisposition is one of the most intriguing factors associated with increased risk for breast cancer. The growing knowledge base about the fundamental changes in gene structure and expression involved in tumorigenesis suggest that patterns of risk can be precisely defined on a person to person basis. Easton et al. (1993) state that the genetic predisposition is reflected in approximately 20% of breast cancer patients who have a positive family history of breast cancer, and identified more specifically in the 5% of patients in whom a specific germ line mutation has been identified. Parker et al. (1996, 1997) and Sakorafas (1999), state that breast cancer is the commonest neoplastic disease in women in the western world, with a lifetime risk of 11-12 % in the general population. Sakorafas et al. (2000) further state that hereditary breast cancers

account for 5-10 % of all breast cancer cases wherein about 90 % of hereditary breast cancers involve mutation of the BRCA1 and/or BRCA2 genes. Studies by Brody et al. (1998) reveal that the BRCA1 and BRCA2 genes are associated with an inherited susceptibility to breast and ovarian cancers. Other cancer related genes (including myc, c-erb B2, Tsg 101 and Mdg1) are involved in breast carcinogenesis, but they do not give rise to familial breast cancer syndromes. Earlier reports by Bierman et al. (1988); Gamel (1989); Huang et al. (1987) and Lynch et al. (1987) have all shown that finger print patterns were also affected in carcinoma of breast.

MATERIALS AND METHODS

This study was carried out in 100 histopathologically diagnosed female patients of carcinoma of breast attending the Radiotherapy department of Goa Medical College, Bambolim, Goa. The cases of carcinoma of breast and the normal controls were selected from the Goan population. The Goan population comprises of around 55% Hindus and 45% Christian (Roman Catholic) population. Both the cases of carcinoma of breast and normal controls were selected randomly for inclusion in this study. These patients were divided into two groups. Group I consisted of carcinoma of breast who had no history of any other genetic disorder or hereditary diseases. They were matched with 100 controls (Group II) having no family history of cancer breast or any other inheritable diseases. Finger prints were recorded with cyclostyling ink and rolled prints were taken of both hands. The finger prints were studied to classify the pattern of

Address for correspondence: Dr. Prashant E. Natekar
Professor & Head, Department of Anatomy, Goa Medical
College, Bambolim 403 202, Goa, India
Phone: (O) 0832-2458701-09 Ext.2250;
(R) 0832-2458787 (M) 09326150001
E-mail: drpenatekar@hotmail.com

whorls, loops and arches. The digital patterns of both hands of cancer of breast patients were compared separately with those of controls. The results were analyzed by using the Chi-square test for testing significance of difference, Odds ratio and 95% C.I. on OR by Woolfe's method.

RESULTS

Out of the 1000 finger prints studied, the cancer breast patients had 33% whorls, 66.6% loops and 0.4% arches whereas the control group had 63.8% whorls, 35.5% loops and 0.7% arches (Table 1, Fig. 1). The number of patients for both carcinoma of breast and the control had very few arches, hence they were excluded from further analysis. The finger print pattern has also been depicted by means of multiple bar diagram in cancer breast patients and the control group.

When the association of presence of more than six loops and carcinoma of breast was tested (Table 2) it was found to be significantly associated ($\chi^2 = 84.48$, $df = 1$, $p < 0.01$ HS). When the risk associated with presence of more than

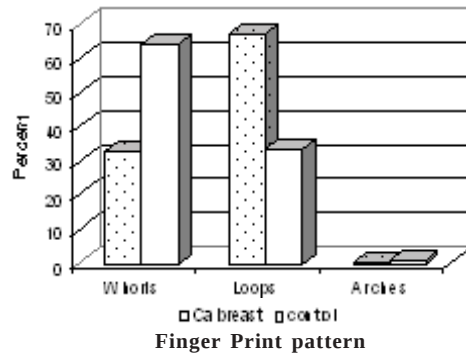


Fig. 1. Multiple bar diagram showing percentage of whorls, loops and arches in the fingerprints of cabreast and control

six loops in carcinoma of breast was evaluated it revealed that the women with more than six loops in their fingerprint pattern were 46 times more at risk of having carcinoma of breast as compared to controls, the OR ratio was 46.58, 95% C.I.=15.78-137.49.

Table 3 shows the association between presence of more than six whorls and carcinoma

Table 1: Percentage distribution of whorls, loops and arches in the fingerprints of carcinoma of breast and the control group and significance of difference of proportion.

Finger print pattern	Carcinoma of breast (I)			Control (II)			t value	p value
	Total fingers	Number of fingers	%	Total fingers	Number of fingers	%		
Whorls	1000	330	33	1000	638	63.8	14.48	p<0.01
Loops	1000	666	66.6	1000	355	35.5	15.67	p<0.01
Arches	1000	4	0.4	1000	7	0.7	0.9	p>0.05

I = Carcinoma of breast

II = Control

Table 2: Association between presence of more than six loops and carcinoma of breast.

Finger print pattern	Carcinoma of breast (I)	Control (II)	Total	χ^2	p value	OR
Loops > 6	66	4	70	84.48	<0.01HS	46.58
Loops < 6	34	96	130			
Total	100	100	200			

OR = 46.58, 95% C.I. =15.78-137.49.

Table 3: Association between presence of more than six whorls and carcinoma of breast.

Finger print pattern	Carcinoma of breast (I)	Control (II)	Total	χ^2	p value	OR
Whorls > 6	4	24	28	16.61	<0.01	0.1319
Whorls < 6	96	76	172			
Total	100	100	200			

OR=0.1319, 95% C.I. = 0.035-0.49

of breast. Analysis shows that presence of >6 whorls is also significantly associated negatively with carcinoma of breast ($\chi^2=16.61$, $df=1$, $p<0.01$). It appears that >6 whorls have a protective effect for carcinoma of breast. (OR=0.1319, 95% C.I. = 0.035-0.49)

DISCUSSION

The specific breast cancer predisposing genes are BRCA1, BRCA2 and p53. Studies by Bowcock (1997), Easton et al. (1993), Shattuck et al. (1995) and Petty et al. (1997), all corroborate the finding that mutations in BRCA1 account for breast cancer in 50 % of families. According to Wooster et al. (1994) BRCA2, the second breast cancer susceptibility gene, was mapped to chromosome 13q12-q13. Sakorafas et al. (1993) state that the human p53 gene located on the short arm of chromosome 17 is known to be a tumour suppressor gene that can be inactivated by point mutations. Recently Begg (2002) reported that though most BRCA mutation carriers were ascertained by membership in families with a high incidence of breast and ovarian cancers, the actual effects of the gene are likely to be confounded by environmental factors or by contributory activity of other genes. Serova et al. (1997) state that at least one (and possibly several) other major susceptibility genes are likely, since only a fraction of high risk families have been demonstrated to have mutations in BRCA1 or BRCA2.

Several models for assessing breast cancer risk from multiple factors have been developed:

Gail Model

The most widely accepted model for general risk assessment. Gail et al (1989) assessed a variety of potential risk factors using unstratified logistic regression analysis. The major determinants of risk in this population of women were: 1) family history in a first degree relative, (2) late age at child birth, (3) early menarche, and (4) multiple previous benign breast biopsies.

Claus Model

Claus et al (1994) developed a model to address some of the deficits in Gail model such as second degree relatives, family history of contralateral cancer, or age at which relatives

developed breast cancer. In this model, risk assessment is based on the number and type of the relatives affected and on the ages at which they become affected. This model is appropriate only for a particular high risk subset of patients with breast cancer, who have at least one family relative also diagnosed with breast cancer.

Other Models

The BRCAPRO model by Berry et al. (1997) calculates the probability that a particular set of family history criteria is related to a mutation in a BRCA gene, taking into account the ages of all affected first and second degree relatives, and whether they have been diagnosed with unilateral breast cancer, bilateral breast cancer, or ovarian cancer. Studies by Brodian et al. (1996) calculate the risk of invasive or in situ breast cancer based on the occurrence of LCIS (Lobular in situ carcinoma) and the age at which LCIS was diagnosed. This study provides a comprehensive coverage of breast cancer patients. The pattern of dermal ridges and furrows are formed very early in the fetal life. Once formed, they remain unchanged throughout life and vary between the individuals.

Earlier studies in breast cancer patients were centered on the dermatoglyphics patterns of the fingers in individuals suffering from breast cancer. Studies by Seltzer et al. (1990) reveal that a pattern of six or more digital whorls was identified more frequently in women with breast cancer than in those without the disease. According to Bierman et al. (1988), four significantly associated finger patterns were observed with breast cancer: accidentals, transitionals, angled ulnar loops and horizontal ulnar loops. Further, Huang et al. (1987) reported significant excess of radial loops on the left hand, whereas in premenopausal women with breast cancer there was increased frequency of ulnar loops on the left hand and excess of radial loops on the left hand in postmenopausal women with breast cancer. In our study a pattern of six or more digital loops were significantly identified more frequently in 66 women with breast cancer than those without the disease. ($\chi^2 = 84.48$). Although studies by Seltzer et al. (1990) reveal that a pattern of six or more digital whorls was identified more frequently in women with breast cancer, a pattern of six or more loops is also a significant predisposing factor for development of carcinoma of breast. The positive predicted

value associated with six or more digital loops concluded that digital dermatoglyphics may have a future role in identifying women either with or at increased risk for breast cancer such that either risk reduction measures or earlier therapy may be instituted.

CONCLUSION

The present study concludes that there is a possible genetic influence on the digital ridge patterns in carcinoma of breast patients in whom the digital ridge patterns is otherwise significantly affected. Though a high risk population is epidemiologically identified, these studies will allow us to detect possibility of breast cancer so as to enable us to take preventive prophylactic measures concerning the environmental factors, and in particular hormonal factors. These relatively non invasive techniques could reasonably be used on selected non symptomatic women (e.g., those with a positive family history) as part of definitive risk assessment strategy. An ability to detect the earliest changes associated with breast tumorigenesis, years or decades before the appearance of measurable tumour may allow the introduction of more effective chemopreventive strategies. More precise tools based on techniques of molecular biology such as microarray analysis, will be needed to assess individual risk for breast cancer. Women who are at high risk for breast cancer can have a variety of options available to them, including watchful waiting, prophylactic surgery and chemoprevention so as to accurately assess a patient's risk.

REFERENCES

- Begg, C.B.: On the use of familial aggregation in population based case probands for calculating penetrance. *J. Natl. Cancer Inst.*, **94**: 1221-1226 (2002).
- Berry, D.A., Parmigiani, G., Sanchez, J.: Probability of carrying a mutation of breast ovarian cancer gene BRCA1 based on family history. *J. Natl. Cancer Inst.*, **89**: 227-238 (1997).
- Bierman, H.R., Faith, M.R. and Stewart, M.E.: Digital dermatoglyphics in mammary cancer. *Cancer Invest.*, **6**(1): 15-27 (1988).
- Bodian, C.A., Perzin, K.H. and Lattes, R.: Lobular neoplasia: Long term risk of breast cancer and other relation to other factors. *Cancer*, **78**: 1024-1034 (1996).
- Bowcock, A.M.: Breast cancer genes. *Breast J.*, **3**(Suppl 3): 1-6 (1997).
- Brody, L., Biesecker, B.B.: Breast cancer susceptibility genes: BRCA1 and BRCA2. *Medicine*, **77**: 208-226 (1998).
- Claus, E.B., Risch, N. and Thompson, W.D.: Autosomal dominant inheritance of early onset breast cancer. *Cancer*, **73**: 643-651 (1994).
- Easton, D.F., Bishop, D.T., Ford, D. and Crockford, G.P.: Genetic linkage analysis in familial breast and ovarian cancer: Results from 214 families. The breast cancer linkage consortium. *Am. J. Hum. Genet.*, **52**: 678-701 (1993).
- Gamel, G.W.: Digital dermatoglyphics in mammary cancer. *Cancer Invest.*, **6**(1): 15-27 (1989).
- Gail, M.H., Brinton, L.A. and Byar, D.P.: Projecting individualized probabilities of developing breast cancer for white females who are being examined annually. *J. Natl. Cancer Inst.*, **81**: 1879-1886 (1989).
- Holt, S.B.: Dermatoglyphics in Mongolism. *Ann. Acad. Sci.*, **171**: 602 (1970).
- Holt, S.B. and Lindstein, J.: Dermatoglyphics in Turners syndrome. *Ann. Hum. Genet.*, **28**: 87 (1964).
- Huang, C.M., Mi, M.P.: Digital dermal patterns in breast cancer. *Proc. Natl. Sci. Counc. Repub. China B.*, **11**(2): 133-6 (1987).
- Lynch, H.T., Albano, W.A., Heieck, J.J., Mulcahy, G.M., Lynch, J. F., Layton, M.A. and Danes, B.S.: Genetics, biomarkers, and control of breast cancer: A review. *Cancer Genet. Cytogenet.*, **13** (1): 43-92 (1987).
- Parker, S.L., Tong, T., Bolden, S. and Wingo, P.A.: Cancer statistics. *CA Cancer J. Clin.*, **46**: 5-27 (1996).
- Parker, S.L., Tong, T., Bolden, S. and Wingo, P.A.: Cancer statistics. *CA Cancer J. Clin.*, **47**: 5-27 (1997).
- Petty, E.M. and Killeen, A.A.: BRCA1 mutation testing: Controversies and challenges. *Clin. Chem.*, **43**: 6-8 (1997).
- Sakorafas, G.H. and Glinatsis, M.T.: *Oncogenes: Their Clinical Significance*. Athens: Infomedia (1993).
- Sakorafas, G.H.: *Breast Cancer*. Lagos Medical Publications, Athens (1999).
- Sakorafas, G.H. and Tsiotou, A.G.: Genetic predisposition to breast cancer: A surgical perspective. *British J. Surg.*, **87**: 149-162 (2000).
- Serova, O.M., Mazoyer, S. and Puget, N.: Mutations in BRCA1 and BRCA2 in breast cancer families: Are there more breast cancer susceptibility genes? *Am. J. Hum. Genet.*, **60**: 486-495 (1997).
- Schauman, B. and Alter, M.: *Dermatoglyphics in Medical Disorders*, pp146-203, Springer Verlag Publication, New York (1976).
- Seltzer, M. H., Plato, C.C. and Fox, K.M.: Dermatoglyphics in the identification of women either with or at risk for breast cancer. *Am. J. Med. Genet.*, **4**: 482-8 (1990).
- Shattuck-Eidens, D., Mc Clure, M., Simard, J., Labrie, F., Narod, S., and Couch, F.: A collaborative survey of 80 mutations in the BRCA1 breast and ovarian cancer susceptibility gene. Implications for presymptomatic testing and screening. *JAMA*, **273**: 535-41 (1995).
- Wooster, R., Neuhausen, S.L., Mangion, J., Quirk, Y., Ford, D. and Collins, N.: Localisation of a breast cancer susceptibility gene, BRCA2, to chromosome 13q12-13. *Science*, **265**: 2088-90 (1994).