

Mini-Review**STAGES, ALCOHOLISM AND GENETIC BASIS
OF BREAST CANCER**

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ABSTRACT

Breast cancer (malignant breast neoplasm) initiate from breast tissue, mostly from the inner lining of milk ducts or the lobules that supply the ducts with milk. Two abnormal genes that involve are: BRCA1 (breast cancer gene one) and BRCA2 (breast cancer gene two). The occupation of the BRCA genes is to renovate cell damage and keep breast cells growing normally. But breast cancer risk increases when abnormal or mutated genes pass from generation to generation.

Keywords: Breast cancer, Stages, genes, BRCA1, BRCA2

INTRODUCTION

A form of cancer discovered in Egypt. Breast It is a major public health issue in less developed countries that leads to death in women. Breast cancers are responsive to hormones such as estrogen and progesterone, so it is feasible to treat it by blocking the effects of these hormones in the objective tissues (Zhu et al., 2011; Dowlatshahi at al., 1997). The prognosis of estrogen and progesterone positive tumor are much better and less forceful treated as compare to hormone negative cancer. It is the number one cause of cancer deaths in female (DeBoer et al., 2010; Weaver, 2003; Krag et al., 2007).

STAGES OF BREAST CANCER

Different phases are described as under

Phase 0

It is non-insidious breast cancers, in it no indication of cancer occurs but Paget's disease of nipple can be observed (Weaver et al., 2000; Weaver et al., 2009).

Phase 1

It is unrelenting (breaking of cancer cells that invade to the neighbouring cells) in which cancer capable to extend 2 centimeter in surrounding tissue (Weaver, 2009).

Phase 2

Cancer in this phase swells up not above 4 cm in greatest span (Greene et al., 2002).

Phase 3

It is further divided in two sub phases (Faderl et al., 1999).

Phase 3A

In this sub phase the greatest dimension of cancer not more than 5 cm and not less than 2 cm (Lugo et al., 1990).

- with homolateral region but some time lacking.

Phase 3B

Describes invasive breast cancer in which

- cancer size of up to and more than 5 cm in diameter
- cancer may have broaden to lymph nodes near the breastbone
- this stage is considered as inflammatory breast cancer

Phase 4

It is also invasive in which

Cancer of any size may spread to any organ of body. This stage is considered as metastatic cancer (Baccarani et al., 2006; Baccarani et al., 2009).

ALCOHOLISM AND BREAST CANCER

Alcohol enhances the level of hormones such as estrogen in our body. A high level of estrogen causes breast cancer. In our body, acetaldehyde is formed by the conversion of alcohol. Acetaldehyde is a toxin that causes cancer by damaging DNA and averting it from being mended. Acetaldehyde is found in large amount in the saliva of people who drink heavily and smoke. It is found by various observations that every alcohol unit drunk a day enhances the possibility of breast cancer about 7-11%. The chances of breast cancer in teenage girls is 25% elevated than as compare to women because of early menarche (Hochhaus et al., 2008; Shah et al., 2008).

GENES INVOLVED

Breast cancer genes (BRCA)

BRCA1 and BRCA2 genes are found in human chromosomes. Breast cancer is developed by its altered form. But several studies have shown that breast cancer is not developed by only an altered form BRCA-1 gene. It is also observed that 10% of such cancer originated by abnormal form of these genes (Shah et al., 2010).

p53

The p53 involves in protein construction which use in the growth of cell. Abnormal p53 gene causes Li-Fraumeni syndrome (soft tissue cancer), it enhances the threat feature of breast cancer (Druker et al., 2006; de Lavallade et al., 2008).

Other genes

ATM: Dented DNA are repaired by the ATM gene But brain development is affected by inherit two abnormal copies of ATM gene. In some families, inherit abnormal ATM gene enhances the risk of breast cancer because it stops renovation of cells of breast tissue.

CONCLUSION

It is serious inflammatory disease which leads to death in women. Various risk factors are responsible for it. Different gene that control the breast tissue's cell cycle, when they are in abnormal form they may cause breast cancer.

REFERENCES

- Baccarani et al., (2006). Evolving concepts in the management of chronic myeloid leukemia: recommendations from an expert panel on behalf of the European LeukemiaNet. *Blood*, 108:1809-1820.
- Baccarani et al., (2009). Chronic myeloid leukemia: an update of concepts and management recommendations of European LeukemiaNet. *J Clin Oncol*, 27:6041-6051.
- de Lavallade et al., (2008). Imatinib for newly diagnosed patients with chronic myeloid leukemia: incidence of sustained responses in an intention-to-treat analysis. *J Clin Oncol*, 26:3358-3363.
- DeBoer et al., (2010). Breast cancer prognosis and occult lymph node metastases, isolated tumor cells, and micrometastases. *J Natl Cancer Inst*, 102:410-425.
- Dowlathshahi et al., (1997). Lymph node micrometastases from breast carcinoma: reviewing the dilemma. *Cancer*, 80:1188-1197.
- Druker et al., (2006). Five-year follow-up of patients receiving imatinib for chronic myeloid leukemia. *N Engl J Med*, 355:2408-2417.
- Faderl et al., (1999). The biology of chronic myeloid leukemia. *N Engl J Med*, 341:164-172.
- Greene et al., (2002). AJCC cancer staging manual. 6th ed. New York: Springer.
- Hochhaus et al., (2008). Dasatinib induces durable cytogenetic responses in patients with chronic myelogenous leukemia in chronic phase with resistance or intolerance to imatinib. *Leukemia*, 22:1200-1206.
- Krag et al., (2007). Technical outcomes of sentinel-lymph-node resection and conventional axillary-lymph-node dissection in patients with clinically node-negative breast cancer. *Lancet Oncol*, 8:881-888.
- Lugo et al., (1990). Tyrosine kinase activity and transformation potency of bcr-abl oncogene products. *Science*, 247:1079-1082.
- Shah et al., (2008). Intermittent target inhibition with dasatinib 100 mg once daily preserves efficacy and improves tolerability in imatinib-resistant and -intolerant chronic-phase chronic myeloid leukemia. *J Clin Oncol*, 26:3204-3212.
- Shah et al., (2010). Potent, transient inhibition of BCR-ABL with dasatinib 100 mg daily achieves rapid and durable cytogenetic responses and high transformation-free survival rates in chronic phase chronic myeloid leukemia patients with resistance, suboptimal response or intolerance to imatinib. *Haematologica*, 95:232-240.
- Weaver et al., (2000). Pathologic analysis of sentinel and nonsentinel lymph nodes in breast carcinoma: a multicenter study. *Cancer*, 88:1099-1107.
- Weaver et al., (2009). Metastasis detection in sentinel lymph nodes: comparison of a limited widely spaced (NSABP protocol B-32) and a comprehensive narrowly spaced paraffin block sectioning strategy. *Am J Surg Pathol*, 33:1583-1589.
- Weaver, D. L. (2003). Sentinel lymph nodes and breast carcinoma: which micrometastases are clinically significant. *Am J Surg Pathol*, 27:842-845.
- Zhu et al., (2011). Perspectives of breast cancer etiology: synergistic interaction between smoking and exogenous hormone use. *Chin J Cancer*, 30(7):433-41.