CORRELATION OF GENE AND RISK FACTORS WITH ETIOLOGY, EPIDEMIOLOGY OF BREAST CANCER

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ABSTRACT: Breast cancer is utmost in founding women but not rare. It is anticipated that 2.4 million new instances of Breast-cancer are reported internationally every year. That based on levels of mRNA expression of human gene, Breast-cancer might be distributed into sequence of molecular subtypes that offer visualizations into novel herbal medication techniques and affected individual stratifications that affect the control of sufferers. This study addresses the evaluation of the BC epidemiology, danger elements, and class with an emphasis on molecular types, analytical biomarkers, in addition to feasible remedy modalities. Its occurrence and loss of life prices have multiplied over the last 3 years because of the alternate in danger aspect profiles, higher cancer registration, and higher cancer detection. Approximately 80% of BC patients are over the age of 50. survival is relied upon at each level and molecular subtype. Invasive Breast-cancer contains huge spectrum of tumours that display a variety of versions regarding their scientific presentation, behaviour, and morphology. Complicated treatment includes surgery, chemotherapy, hormonal treatment, radiotherapy, or organic remedies brought in numerous sequences.

KEY WORDS: Breast cancer, genes, Epigenetics, Risk factor

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INTRODUCTION:

Beauty and inspiration can bring people to life. Breast is the mother's first sensation. Therefore, food, sensation, and energy come from the mother's breast (Lycons et al., 2020). After puberty, the female breast develops and continues to grow in size until the mature adult breast posture, with the nipples and areola growing as time goes on. Lyons and others, 2020) The size of a woman's breasts varies from person to person, depending on her physical characteristics and genetics. Ideally, the breast provides the new-born with a food factory, but few women choose not to breastfeed their children (1). When breastfeeding is stopped but the mammary glands continue to function, milk accumulation in the mammary gland can lead to a variety of health issues and diseases (2).

As a result of milk accumulation in the breast, lumps, inflammation, and pain can occur. Sometimes, milk-like fluid will spontaneously leak out of the nipple. If these issues persist for an extended period of time; they could lead to a dangerous carcionomics problem in the breast (3).

a condition in which the tissues of the female breast are the source of abnormal and mutated cells. There are primarily two types of in situ breast cancer: Nipple Paget disease and in situ ductal carcinoma (DCIS). The cells that form the ducts of breast milk, which carry milk to the female nipple, are the starting point for ductal carcinoma, which is not a rare type of breast most cancers. DCIS, also known as intraductal-carcinoma, is a type of invasive ductal carcinoma (IDC)(4). Paget's disorder of the nipple, also known as Paget's disorder of the breast, is a rare condition associated with breast cancer. It causes eczema-like changes to the skin on the nipple and in the area of darker skin (areola) surrounding the nipple. It's by and large a sign of bosom most malignant growths with inside the tissue toward the rear of the nipple. (5)

This way there might be a hereditary variety surpassed down with inside the own circle of family members that blast the possibility becoming sure cancers (6). The two most common place genes associated with hereditary breast and ovarian cancer are BRCA 1 and BRCA 2. Prostate, pancreatic, fallopian tube, peritoneal, and melanoma are just a few of the cancers that people with certain editions in these genes can increase their risk of developing (7). The acronym for "Breast Cancer Gene" is "BRCA." BRCA1 and BRCA2 are distinct genes that have been found to influence a person's risk of developing breast cancer in the future (8).
TP53, CDH1, PTEN, STK11, ATM, PALB2, BRIP1, CHEK2, and XRCC2 all play a role in breast cancer in women, as do BRCA1, BRCA2, and HER (9).
Every human has both the BRCA1 and BRCA2 genes. Breast cancer is not caused by the BRCA genes, contrary to popular belief (10). In fact, these genes are frequently linked to breast cancer prevention. Desoxyribonucleic acid breaks, which can cause cancer and uncontrollable proliferation, can be repaired with their assistance. Because of this, the BRCA genes are referred to as "tumour suppressor genes" (10). The BRCA genes are therefore referred to as tumour suppressor genes. It's possible that HER is a key component of conventional cell growth. Other types of cancer, including breast, ovarian, bladder, pancreatic, and abdomen cancers, also produce HER2/neu in more complex than usual aggregates.(11) This may cause cancer cells to grow more rapidly and spread to other parts of the body. (12)

BREAST CANCER ETIOLOGY AND EPIDEMIOLOGY

Distribution of Breast Cancer in all over the world

According to a WHO survey, the greatest global burden of disease for females is represented by malignant neoplasms, estimated at 108 million DALYs, of which female breast cancer accounts for 19.7 million DALYs. By 2020, breast cancer will account for 2.27 million new cases worldwide, making it the most common cancer in women to be diagnosed. In the United States, it is anticipated that female breast cancer will account for 30% of all other cancers that affect women (13). The Human Development Index (HDI) is strongly and unquestionably associated with the standardized age prevalence costs of breast cancer, as shown by GLOBOCAN statistics from 2018 (14). According to data from 2020, the ASIR performed better than the competition in coffee HDI regions (with a score of 36.1 out of 100,000), medium HDI regions (with a score of 27.8 out of 100,000), and very high HDI regions (with a score of 75.6 out of 100,000).
Not only is breast cancer the most common type, but it is also the leading cause of death among women worldwide. Breast cancer is estimated to be the cause of 684,997 deaths worldwide at an age-adjusted rate of 13.7 per 100,000 people (15). Although prevalence rates have been highest in advanced regions, Asian and African nations shared 64% of all deaths in the previous year (15). The majority of women who develop breast cancer while earning a lot of money will survive; the other is exact for females in most extreme low wages and a lot of centre salaries worldwide localities (16).
In 2020, mortality predominance proportion as an expert mark of endurance costs that was 5 a year changed into 0.30 universally (15). Taking into thought the clinical measure of diseases, in places with some high level wellness upkeep in which Hong Kong, and Turkey are incorporated the 5 a year's endurance changed into 89% for neighbourhood and 75.4% for adjacent most malignant growths. The survival rates for limited and nearby other cancers were 76% and 47.5 percent, respectively, in less

unconventional international sites that include the Philippines, Pakistan, Saudi Arabia, India, and Thailand (17).

Breast cancer and genetics:

The modern definition of epigenetics is the heritable or brief changes in gene expression that do not involve a specific change in the DNA sequence. An explosion of research into epigenetics and cancer has led to a significant increase in our comprehension of the various mechanisms and outcomes that underlie these non-structural genome modifications (18). The possibility of reversing the effects with treatments that might cause gene re-expression is the exciting aspect of discovering numerous epigenetic changes. DNA methylation and histone modification cause nucleosome reworking, which silences essential tumor-suppressor and growth-regulatory genes in breast most cancers. Preclinical research has focused largely on using epigenetic treatments to re-explicit the silenced gene in molecular traces (19). The scientific applicability of what we see in the laboratory and the scientific efficacy of this strategy have been the more difficult issues. Preclinical research, for instance, clearly demonstrates that the maspin gene can be re-expressed in breast cancer molecular traces by employing demethylating agents and histone deacetylase inhibitors (HDACi). However, the most important question is whether or not this kind of re-expression in a patient's tumor would not affect scientific findings (19). The capacity function of such treatments in breast cancer research has advanced despite the challenging aspects of the scientific application of epigenetic therapy (20).

Breast cancer and skin eczema

Eczema are frequently linked to changes in multiple systems caused by paraneoplastic manifestations. dermis sclerus, diffuse sclerosis, acquired lanuginose, and choriotomyositis There are enlargement cells in the skin and pores in maximum pore enlargement and related skin manifestations as well as erythroderma (21).

An interaction between cytokines (interleukin 12 8) and adhesion cells (VCAM1, E-selectin, ICAM1, and Pselectin) may also play a significant role in the development of paraneoplastic rashes. This interaction results in the binding, migration, and penetration of lymphocytes and mononuclear cells, which ultimately replace the essential epidermis. Cell adhesion fragments are prevalent, and carcinoma is another well-known release of this protein (22).

Consequences of leprosy in breast cancer

Effects of leprosy on breast cancer Another aspect of leprosy that is very relevant is its episodes of reactional. However, there have been no earlier references with inside the examination study concerning articulation of mRNA in responses of disease. In accordance with type I reaction, we discovered that 14 mRNAs were differentially expressed primarily in those samples. In the literature, many of these mRNAs have been linked to conditions like lung, chest, and renal cancer (23). Upregulation of HsamiR-500a and HsamiR-34a is linked to improved neuroblastoma and a destructive response to chemotherapy in large molecular pulmonary carcinoma (24). The mRNA-378 gene's relevance to the comparison of R1 to R1, particularly in terms of scientific correspondence, is limited. The lipid metabolism is linked to miRNA-378, according to the literature (25).

Six miRNAs have been exclusively expressed in type 2 responses in those models. According to previous research, downregulation of HsamiR-214 and Hsa-miR-125b-2 is associated with miscarriages, the development of elderly gastric adenocarcinoma, glioma-molecular

proliferation, and germ molecular tumor growth inside the testis (26). In addition, periodontitis and gastric cancer are immediately linked to hsamiR-223 upregulation (27). How these miRNAs participate in the initiation and/or preservation of type I and type II reactions is currently unknown. The clinical, histological, and pathological characteristics of reactions and scientific records are distinct. Interstitial cell phenotype, granuloma composition, and angiogenesis are just a few of the molecular types that undergo significant change. As a result, the scientific and histopathological characteristics that are unique to the various documents and responses of the disease could be defined using differentially expressed mRNAs. It is unknown where these mRNAs are located in leprosy (27).

In patients with these kinds of cancer, its ability to target arylamine N-acetyltransferase 1 is linked to a longer survival time (28). Hsa-miR-139-5p expression is significantly lower in disease samples than in healthy tissues, suggesting that it plays a crucial role in breast and respiratory organ cancers. Abuse that objectives oncogenic c-Met supports its restraint of sub-atomic expansion, metastasis, and necrobiosis (29).

FACTORS OF RISK:

Non-Adaptable Factors

Chromosome instability

Factors that Cannot Be Changed Chromosome Instability The role that genetic changes play in neoplasia has been the subject of much debate for the past one hundred years. In 1890, David Hansemann conducted the first comprehensive study of the molecular structure of malignant tumors (30). Later, in 1914, Theodor Boveri (31) made the first suggestion that malignancy may actually result from a disruption in the everyday chromosome balance, which is necessary for daily molecular function (31). The somatic mutation principle, or the idea that neoplasia arises in a single molecular way from a obtained genetic change, remains the predominant view of the pathogenesis of most cancers. There is a plethora of experimental evidence to back up this viewpoint. Spontaneous chromosome instability has been linked to the majority of cancer risk factors. The majority of genetic modifications in cancer, including the nonrandom kind like the Ph1 chromosome in chronic myelogenous leukemia, are obtained within the target cells following the formation of the zygotes and the differentiation of the tissues. Therefore, a genetic predisposition to the majority of cancers may be caused by a variety of mechanisms (32). One possibility is genetic instability, which occasionally manifests as chromosome instability. Additionally, individuals with genetic instability are more likely to produce more cells containing mutations or chromosomal aberrations than those with robust genomes. A genetic charter similar to the initial stage of carcinogenesis may also emerge from one of these abnormal cells in the target tissue (33).

Feminine behavior

Due to enhanced secretion stimulation, female gender is one of the primary factors associated with a degree redoubled risk of carcinoma. Female breast cells are particularly vulnerable to sex hormones, particularly estrogen and progesterone, as well as slight imbalances in their balance, unlike male breast cells, which give off insufficient estrogen intensities. Increased cancer risk is inextricably linked to existing hormones (34). Changes in the physiological stages of endogenous hormones are to blame for the subsequent cancer risk in biologically timed women; These interpretations were also supported by the hormones and carcinoma cooperative group (35).

Less than 1% of all cases of breast cancer involve men. Contrary to popular belief, men are more likely than women to develop chest cancer, which is a rare condition. Males were about 67 years old when the discovery was made. The most significant factors that increase a man's risk of developing breast cancer are age, elevated estrogen levels, alterations in the BRCA2/BRCA1 gene, Klinefelter syndrome, a family history of cancer, and prior exposure to radioactivity therapy (36).

Age: Menopausal

About 80% of cancer patients are over 50 years old at the same time, and more than 40% of cancer patients are over sixty-five years old at the same time (37). The following factors will increase the risk of developing breast cancer: 1.5% by age 40, 3% by age 50, and over 4% by age 70. A correlation between a patient's age and a specific molecular cancer subtype was discovered, which was encouraging. Patients under the age of forty are more likely to develop the competitive resilient threefold undesirable carcinomas subtype, whereas patients over 70 are more likely to develop the luminal A subtype (37). The majority of older cancers are now more than just breast cancers, in general; Carcinogenesis rises over time as a result of exposure to cancer agents and the accumulation of numerous cell mutations.

Inheritance of breast cancer

Breast most cancers records in one's own family are a significant predictor of a long-term risk of developing the disease. Inheritance of breast cancer The same state affected between 13 and 20% of breast cancer patients with a primary diploma. Furthermore, as the quantity of top of the line mates impacted builds, the gamble of bosom malignant growth will altogether rise; The odds are probably even better when the spouse and children are younger than 50 (38). Women of any age who have breast cancer in their family are significantly less likely to develop the disease. Changes in the epigenome drive this connection, as well as factors in the environment that appear to be ability triggers. A family history of ovarian cancer, particularly those characterized by BRCA1 and BRCA2 gene mutations, may also increase the risk of developing cancer (39).

Genetic information mutation:

A number of genetic mutations have been linked to an increased risk of breast cancer in a strong way. There are two main DNA segments with a lot of penetration: the first gene, BRCA1, which is on the 17th chromosome, and the second gene, BRCA2, which is on the 13th chromosome. The majority of them are connected to an increased risk of breast cancer (40). the mutations that were found in the previous study. The majority of DNAs are passed down through dominant autosomal inheritance;(41) However, sporadic changes are also frequently mentioned (Shahbandi and others, another gene that causes cancer has a wide range of penetration, and its name is TP53. 2020)(42). When they are not in a location with a higher risk of breast cancer, carriers do not face any additional challenges. People with these genetic mutations are likely to also have advanced ovarian cancer(43). A significant number of ATM, PALB2, CHEK2, and BRIP1 genes are models of DNA renovation genes that can interrelate with gene BRCA and were found to be convoluted in the induction of breast carcinogenesis, (44) despite having a lower penetrance (moderate degree) than BRCA1 or BRCA2(45). According to the findings of a recent study, mutations in the genetic material of XRCC2 may also raise the risk of developing cancer (46).

Effect of socioeconomic and racial background

Breast cancer patients continue to typically exhibit quality and racial disparities; The

underlying mechanisms of this development are still poorly understood. The overall prevalence of breast cancer remains highest among white, non-Hispanic women (47). However, the malignancy mortality rate is particularly high among black females; Similar characteristics define this establishment, including its low survival rate strategy (48).

History of family generation

Numerous studies demonstrated a strong link between exposure to estrogen, an endogenous hormone, and the risk of female cancer. (49) As a result, the development of malignant neoplastic disease in the breast microenvironment is significantly influenced by specific factors like physiological state, breastfeeding, menarche, and climacteric at the same time as their period, as well as collateral secretion imbalance(50). Primary full-term pregnancies at a young age (especially in the early 20s) are linked to a lower risk of breast cancer and a later growing diversity of births. Albrektsen, others, and in addition, cancer can be avoided by the physiological state itself. However, protection for pregnancies with a gestational age of less than 33 weeks was not demonstrated and was only established around the 34th week of pregnancy (51). Women who had toxemia during pregnancy or delivered from a preeclampsia pregnancy have a lower risk of developing cancer. Up until this point, there was no clear connection between abortion and an increased risk of breast cancer (52). High levels of insulin, insulin-like growth factor-1, cortisol, androgens, human sac gonadotropin, corticotrophin-releasing issue, and Associate in Nursing IGF-1 binding super molecule deviating from physiological series, as well as dysregulated endocrine levels that occur during preeclampsia have a defensive effect and prevent breast carcinogenesis (53).

Breast Tissue Anatomy

The breast muscle's uneven thickness persists throughout the period; However, a number of groups, such as those with low mass, high density, and heavy breasts, had been identified in medical education. Senior women are more likely to use hormonal replacement therapy at some point, have a lower body mass index (BMI), are more likely to be pregnant or breastfeeding, and have a higher density (54). Greater breast muscle density is typically associated with a higher risk of breast cancer; Postmenopausal women typically sport this look (55). During routine medical examinations of females with a higher risk of most cancers, it was anticipated that demonstrating breast tissue mass would also be an efficient, minimally invasive method (56).

Chronicle past of Breast Cancer

A personal history of carcinoma is associated with an increased likelihood of breast cancer lesions recurring (57). The risk is also significantly increased if you have a history of other breast changes that aren't cancer, like atypical hyperplasia, situ cancer, or a number of other proliferative lesions. The histological classification of benign tumors and breast cancer cases has a strong correlation with cancer risk (58).

History of Breast Radiology

The possibility of minor tumors following radiotherapy treatment persists. Human gene material that is influenced by the characteristics of a cancer patient is a fairly common occurrence that causes numerous medical issues. The generation of cancer cells during radiation therapy is strongly correlated with an individual's age; cancer Patients who underwent radiation therapy before the age of 30 are more likely to develop breast cancer (59).

MODIFIABLE FACTORS

Selected Drugs

According to a rough analysis of the data, children who take stilbestrol while in good health may be more likely to get carcinoma;

Nevertheless, research studies continue to be erratic. Diethylstilboestrol consumption during pregnancy has been linked to an increased risk of breast cancer in both the mother and her unborn child (60). Because neither progestin receptors nor estrogen hormone appear in the same cancer's microscopic molecular anatomy, this connection is evaluated (61). Age will increase the risk; Women over the age of 140 are nearly 1.8 times more likely to be at risk than women under the age of 40. In addition, taking a lot of diethylstilboestrol will make it more likely that you will develop carcinoma. According to a variety of studies, women who use HRT, particularly those over the age of seven, also face a higher risk (62). Numerous studies (63) have found a link between taking antidepressants and selective monoamine neurotransmitter uptake inhibitors and an increased risk of breast cancer (64,65). One study (Friedman et al.,) states: segregating antibiotic medications commonly increment risk. 2006). The possibility of a link between cancer risk and non-steroidal anti-inflammatory treatments and hypertensive medications was looked into (66). However, this data still exhibits a great deal of inconsistency (Coogan et al., 1999) (67).

Exercise

It is believed that regular exercise lowers the risk of developing breast cancer, although the mechanism is still unknown. On the other hand, regular exercise reduced the risk of cancer in women with a history of breast cancer. However, this was only available to women who had entered menopause (68). According to Hoffman-Goetz (1998), physical activity may support the protective effect of exercise on the incidence of carcinoma by lowering endogenous sex hormone exposure, adjusting system responses, or increasing insulin-like growth factor-1 levels(69,70).

Body fitness:

Medical studies show that being overweight makes it more likely that you will get breast cancer. This kind of cancer is more common in larger postmenopausal women who are also more likely to develop estrogen-receptor-positive breast cancer. However, poor clinical outcomes in obese women are caused by a number of factors, including menopause (71). Cancer was more common in over-50 women with a higher BMI than in those with a lower BMI (72). Additionally, a higher body mass index (BMI) was found to be linked to larger lymph nodes, a higher risk of lymph node metastasis, and biological growth options that were more aggressive. Fat may contribute to higher mortality rates and the likelihood of cancer recurrence, particularly in postmenopausal women (73). By altering hormone levels and escalating inflammation, body fat may facilitate pro-carcinogenic processes (74). A higher BMI-related carcinoma risk is also linked to a history of breast cancer (75).

Habitual of Alcohol

Habitual Alcoholic Numerous studies have demonstrated a link between an elevated risk of epithelial duct tract cancer and excessive alcohol consumption; However, it is also common knowledge that it increases the risk of breast cancer. More specifically, alcohol consumption quantity rather than type has the greatest impact on cancer risk. The connection between alcohol use and carcinogenesis in female reproductive organs is explained by elevated estrogen levels and an imbalance in secretory function (76). Additionally, excessive fat gain and a higher BMI, both of which contribute to alcohol consumption frequently, will raise the risk. Alternate hypotheses regarding alcohol include malnutrition and the effects of alcohol metabolites on cancer, both directly and indirectly. It was discovered that estrogen-positive breast cancer risk was increased by drinking alcohol, particularly after heavy lifting (77). It contributes to the

development of morphological changes in breast tissue prior to the first pregnancy, making it more susceptible to carcinogenic processes (78).

Consumption of Tobacco

Tobacco-derived carcinogens are transported to breast tissue, enhancing the quality of mutations in oncogenes and suppressor genes (particularly p53). As a result, the onset of pro-carcinogenic events is significantly facilitated by both active and passive smoking (79). A longer smoking history and smoking prior to the first mature pregnancy are additional risk factors that are more prevalent in females with a cancer history (Catsburg et al., 2015) (80).

Vitamin deficiency

Vitamins have antitumor properties, which may assist in the prevention of breast cancer and other forms of cancer (81). On the other hand, the mechanism is still poorly understood. The effects of eating (vitamins like B-complex, C, E, folic acid, etc.) are frequently the focus of study. on the possibility of bosom disease. The majority of studies on ergocalciferol supplementation in breast cancer currently focus on confirming its possible protective effects (82). However, the information is still inconsistent and insufficient to verify the findings and derive reliable data. Blood levels of 25-hydroxyvitamin D are linked to a lower risk of breast cancer over biological time and in postmenopausal women, according to El-Sharkawy & Malki (2020) (83). A lower level of ergocalciferol receptor expression has been linked to a higher risk of death from breast cancer. However, any investigation is required because the information available in this instance is inconclusive (84).

Exposure to Invisible light

In recent years, artificial light-weight in the dark (ALAN) exposure has been linked to an increased risk of cancer. A fluctuating endocrine cadence and the subsequent epigenetic changes might be the possible effort (85). According to the research that has been conducted up to this point, people who are more open to ALAN have a significantly higher risk of developing breast cancer than people who are less exposed (86). However, there is insufficient information regarding the connection between an increased risk of breast cancer and excessive use of electronic devices with light-emitting diodes, and some of the results are contradictory (86).

Nutrition

The World Health Organization (WHO) identified highly processed meat as a single substance associated with an increased risk of both individual and collective carcinoma. Similar findings were made regarding an excessive intake of saturated fats (87). Food that has been extremely processed contains a lot of sugar, sodium, and fat, all of which have been linked to an increased risk of breast cancer (88). For every 10 percent increase in ultra-processed food in a diet, there is a 11 percent increase in the risk of breast cancer (88). A diet high in fruits, vegetables, legumes, whole grains, and lean organic compounds, on the other hand, has been shown to lower B.C. risk (89). Similar properties were found in sulforaphane (SFN), a curcuminoid still derived from turmeric (90).

Not breast-feeding

Some research suggests that breast-feeding may also only marginally reduce the risk of breast most cancers, particularly if it is continued for 1.5 to 2 years. Other studies found no effect on most breast cancers (91).

Exposure to Chemical Hazards

In addition to triggering pro-carcinogenic processes, chronic chemical exposure can also exacerbate breast carcinogenesis by altering epigenetics and interacting with the growth microenvironment (92). Women who have been exposed to chemicals for a significant amount of time have a significantly increased risk of developing a

cancerous neoplastic disease, and this risk is directly correlated with the amount of exposure they have received (93). Numerous chemicals have been associated with breast cancer; (94) Pesticide (DDT) and polychlorinated biphenyl (PCB) have been the subjects of the majority of research regarding carcinoma to date due to the fact that early exposure to these chemicals has an effect on the development of the mammary gland (Eve et al., 2020) (95). In the event of increasing exposure to pesticides, oil mist, polycyclic aromatic hydrocarbons (PAHs), artificial fibers, organic solvents, and PAHs, a possible link was also discovered (96).

Side effect Medication

Prescription medications (such as metal element channel blockers and angiotonin II-converting accelerator inhibitors), antibiotics, antidepressants, statins,(97) and cholesterol-lowering medications are additional potential risk factors for carcinoma (Bjarnadottir et al., 2013) (98).

BREAST CANCER TYPES

According to site

Non-Invasive Breast Cancer:

The website states that non-invasive breast cancer (99) DCIS, or ductal carcinoma in situ, is the most common type of non-invasive breast cancer, affecting only the ducts and not the connective and fatty tissues that surround the breast (90 percent). A less common type of breast cancer known as lobular-carcinoma in situ (LCIS) is a sign of an increased risk (100).

Invasive carcinoma

Carcinoma that spreads: cells that harm the fatty and connective tissues of the breast as well as the walls of the duct and lobe. Cancer will spread to individual organs or humor nodes, but it will not be pathogenic (invasive) (101).

FREQUENTLY GOING ON BREAST MOST CANCERS

Lobular carcinoma in situ:

Frequently occurring Breast most cancers In situ lobular carcinoma: The majority of cancers are referred to as "in situ" when they have not spread beyond the initial location. LCIS is a sharp development with inside the wide assortment of cells in the milk organs (lobules) of the bosom.

In situ ductal carcinoma: DCIS is confined to the breast ducts and is the most prevalent type of non-invasive breast cancer. Take, for instance, ductal comedocarcinoma.

Typical Structure related to ductal carcinoma

Lobular carcinoma that has invaded:

The typical structure of lobular carcinoma that has invaded ductal carcinoma: ILC is a different term for invasive lobular carcinoma. Although ILC typically begins in the lobules of breast milk glands, it frequently metastasizes to other parts of the body. 10 to 15% of all cases of breast cancer are caused by it.

Infiltrating ductal carcinoma (IDC): IDC is also known as invasive ductal carcinoma. Through the duct wall, IDC enters the breast fatty tissue and possibly other parts of the body from the milk ducts. IDC accounts for 80% of all cases of breast cancer and is the most common type (102).

Carcinoma of the medulla, a less common type: A type of invasive breast cancer known as medullary carcinoma has a distinct border between the tumor and normal tissue. Only 5% of breast cancers are medullary carcinomas.

Carcinoma mutant: Mutinous carcinoma, also known as mixed carcinoma, is a rare form of breast cancer that develops from cancer cells that produce mucus. Women who have mutinous carcinoma typically have a much better prognosis than women who have more common forms of invasive cancer.

Cancer of the tubules: Tubular carcinomas are invasive (infiltrating) breast cancers. The prognosis of women with tubular carcinoma

is better than that of women with other invasive cancers. About 2% of all carcinoma diagnoses are hollow carcinomas.

Inflammatory Breast carcinoma

Known as inflammatory breast most cancers, infected breasts have a crimson and warm appearance with dimples and/or thick ridges as a result of cancer cells interfering with the liquid frame substance vessels or channels in the mend the breast. Albeit provocative bosom malignant growth is uncommon (representing under 1% of all bosom tumors), it is very forceful.

Paget's disease of the nipple is a rare form of breast cancer that starts in the milk ducts and eventually spreads to the nipple and areola skin. It is only responsible for about 1% of all breast cancers.

Phylloides neoplasm Phylloides tumors, also known as "phyllodes," can be benign (meaning they are not cancerous) or malignant (meaning they are cancerous). Phylloides tumors spread through the breast's connective tissues and can be treated surgically. Tumors of Phylloides are extremely uncommon; In the United States, this type of cancer kills approximately ten women annually (103).

CONCLUSION: The current prevalence and incidence of breast cancer, risk factors, and classification were the primary focus of this review, with a strong emphasis on summarizing and bringing up-to-date previous knowledge. Concerns are raised by the significant rise in breast cancer mortality and morbidity rates over the past few decades.

It is imperative that preventative measures be flexible and of the highest possible efficacy.

Breast cancer incidence may be reduced significantly by risk factors. The screening procedures that are currently carried out the most frequently enable early detection and are mammography and sonography. breast cancer detection The physical, mental, and social aspects of a woman's life are all affected by this illness. On the other hand, the illness's negative effects can be mitigated with the assistance of social and family support. The administration and clinical results of bosom malignant growth patients have essentially worked on because of the continuous quest for prognostic biomarkers and focuses for possible natural treatments.

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Table 1: Breast cancer risk factors, both modifiable & non-modifiable

Modifiable Factors	**Non-Modifiable Factors**
Selected Drugs	Chromosome instability
Excersice	Feminine behavior
Fitness of the body	Meno-pausal Age
Habitual of Alcohol	Inheritance of breast cancer
Use of Tobacco	Mutation of Genetic information
Deficiency of Vitamin	Ethnic background
Exposure to Un-visible light	History of family generation
Nutrition	Anatomy of Breast Tissue
Not breast feeding	Chronicle past of Breast Cancer
Airing of Hazards Chemical	History of Breast Radiology
Side effect Medication	

Table 2: The gene that causes breast cancer

Dispersion	**Gene**	**Chromosome Site**	**Related Syndromes**	**Foremost Roles**	**Cancer Hazard**	**Ref.**
High	BRCA1	17q21.31	Ovarian cancer Pancreatic cancer Fanconi anemia Breast cancer	Cell cycle control DNA repair	45–87%	(104)
	BRCA2	13q13.1	Biliary cancer Pancreatic cancer Prostate cancer Melanoma Ovarian cancer Fallopian tube cancer Breast cancer Medulloblastoma Fanconi anemia Glioblastoma Wilms tumor	Cell cycle control DNA repair	50–85%	(105)
	TP53	17p13.1	Pancreatic cancer Hepatocellular carcinoma Breast cancer Colorectal cancer Nasopharyngeal carcinoma Li-Fraumeni syndrome Adrenocortical carcinoma Osteosarcoma	DNA repair Cell cycle control Induction of senescence Induction of apoptosis Maintenance of cellular metabolism	20–40% (Even up to 85%)	(106)

	CDH1	16q22.1	Ovarian cancer Prostate cancer Breast cancer Gastric cancer Endometrial carcinoma	adhesions Control of the epithelial cells Regulation of cellular	63–83%	(107)
	PTEN	10q23.31	Prostate cancer Lhermitte-Duclos syndrome Breast cancer Autism syndrome Cowden syndrome 1	Cell cycle control	50–85%	(108)
	STK11	19p13.3	Breast cancer Pancreatic cancer Peutz-Jeghers syndrome Testicular tumor	Maintenance of energy Homeostasis Cell cycle control	32–54%	(109)
Moderate	ATM	11q22.3	Breast cancer Lymphoma Ataxia-telangiectasia T-cell prolymphocytic leukemia	DNA repair Cell cycle control	20–60%	(110)
	PALB2	16p12.2	Pancreatic cancer Breast cancer Fanconi anemia	DNA repair	33–58%	(111)
	BRIP1	17q23.2	Fanconi anemia Breast cancer	Involvement in the BRCA1 activity	ND	(112)
	CHEK2	22q12.1	Prostate cancer Breast cancer Osteosarcoma Li-Fraumeni syndrome	Cell cycle control	20–25%	(113)
	XRCC2	7q36.1	Fanconi anemia Spermatogenic failure Premature ovarian failure	DNA repair	ND	(114)