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Breast Cancer Screening and Operable Breast Cancer: Management, Techniques for Early Diagnosis, Prevention

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ABSTRACT

Background: Breast disease is the most well-known threat in ladies. It is normal described by an absence of early manifestations, which brings about late recognition of the infection. Identification at cutting edge phases of the expire infers the treatment is more troublesome and questionable. The fitting screening programs have been directed inside the coordinated preventive assessments and have made huge commitments to the early bosom disease location.

Objective: It is important to improve the screening interaction to diminish the level of female populace that isn't covered by screening projects and increment the quantity of early-recognized bosom malignancies. The improvement of the screening system might be reflected in the accompanying: more proficient assurance of the rundown of the ones who need to go through preventive assessment, presentation of screening program in thermography as an analytic technique applied in pre-screening stage, more productive investigation of mammograms and nonstop development of patients.

Techniques: The recognizable proof of target populace for bosom malignant growth screening program has been founded on the period of ladies. The improvement of the early bosom disease finding measure proposed in this paper is reflected in more effective assurance of the gathering of ladies who need to go through preventive assessment dependent on the components influencing the event of bosom malignant growth. Incorporation of the pre-screening stage in which warm imaging could be applied and programming backing to mammographic location of tumor are proposed.

Results: This paper depicts the bosom malignancy, current evaluating project and procedures for beginning phase bosom disease identification, the examination of hazard factors influencing the event of bosom malignancy, mammography and part of warm imaging during the time spent early bosom disease location. It's anything but an outline on significant accomplishments in PC helped recognition and determination of bosom disease in mammography and thermography.

Conclusion: Based on the acquired outcomes, elements of preventive assessments for specific gatherings of ladies that is not the same as the standard two-year assessments, can be effectively characterized. It tends to be inferred that the utilization of a PC framework for tumor analysis in mammogram dependent on different techniques for picture handling can help specialists in dynamic, while the utilization of warm imaging in the pre-screening stage would essentially diminish the rundown of ladies for screening mammograms.

1. INTRODUCTION

Breast cancer is the most common invasive cancer in women and the second leading cause of cancer death in women after lung cancer. Advances in screening and treatment for breast cancer have improved survival rates [1-9]. The last decade has seen rapid advances in understanding of breast cancer, facilitated by numerous clinical trials, and an ever expanding knowledge of the molecular changes associated with malignancy. Tumours can now be classified on the basis of their molecular signatures, which are being used to determine prognosis and treatment. There is an increasing array of drugs available for treatment and newer surgical techniques to enable breast preservation [10-12]. This increased range of options has resulted in an individualized approach to the treatment of breast cancer [13].

2. EPIDEMIOLOGY AND ETIOLOGY

Breast cancer is common in the West where women have a lifetime risk of 1:8. In India, the lifetime risk is lower although the incidence is rising. In 2008, the Indian Cancer Society population based registry recorded 100,000 new cases, and 52,000 deaths due to breast cancer

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[7,14-21]. The lifetime risk in the city of Mumbai has increased from 1:27 a few years ago, to 1:22. The urban-rural divide persists with cervical cancer being the commonest female cancer in rural areas. However, in the cities of Mumbai, Delhi, Chennai, and Bangalore breast cancer has overtaken cervical cancer to become the commonest cancer in women [13,22]. The cause of this change remains unknown, and has loosely been attributed to a 'Western lifestyle' [23-30]. However, it may be attributed to increasing awareness, access to mammography and an economically increasing middle-class, with access to medical care [32].

3. RISK FACTORS

The established risk factors of breast cancer include the following:

Age: Breast cancer is not common below the age of 35, the incidence increasing rapidly between the ages of 35 and 50. A bimodal trend in the age distribution has been observed with a dip in incidence at the time of menopause [33].

Family History: The risk is high in those with a positive family history of breast cancer, especially if a mother or sister developed breast cancer when premenopausal [34,35].

Parity: The risk of breast cancer is directly related to. The age at which women bear the first child. An early first, full-term pregnancy seems to have a protective effect [36]. Those whose first pregnancy is delayed to their late thirties are at a higher risk than multiparous women. Unmarried women tend to have more breast tumours than married single women, and nulliparous women had the same risk [37].

Age at Menarche and Menopause: Early menarche and late menopause are established risk factors. The risk is reduced for those with a surgically induced menopause [38,39].

Hormonal Factors: The association of breast cancer with early menarche and late menopause suggests that ovary appears to play a crucial role in the development of breast cancer. Evidence suggests that both elevated oestrogen as well as progesterone are important factors in increasing breast cancer risk. In short, hormones appear to hold the key to the understanding of breast cancer [40].

Prior Breast Biopsy: Prior breast biopsy for benign breast disease is associated with an increased risk of breast cancer.

Diet: Current aetiological hypotheses suggest that cancer of the breast is linked with a high fat diet and obesity. It is not known how dietary fat influences breast cancer risk at a cellular level.

Socio-economic status: Breast cancer is common in higher socio-economic groups. This is explained by the risk factor of higher age at first birth.

Others:

(i) Radiation: An increased incidence of breast cancer has been observed in women exposed to radiation.

(ii) Oral contraceptives: Oral contraceptive appears to have little overall effect on breast cancer, although prolonged use of oral pills before the first pregnancy or before the age of 25 may increase the risk in younger women.

Risk Factors for Breast Cancer Development

Factors	Relative Risk
BRCA mutation	3.0 to 7.0
Breast density	1.8 to 6.0
Atypical ductal hyperplasia on prior biopsy	3.7
Bone density	2.7 to 3.5
Age at first birth	1.9 to 3.5
Mother or sister with breast cancer	2.6
Age at menopause	2.0
Postmenopausal BMI	1.7
Age at menarche	1.4
Hormone replacement therapy	1.2 to 1.4
Oral contraceptive pills	1.2 to 1.4

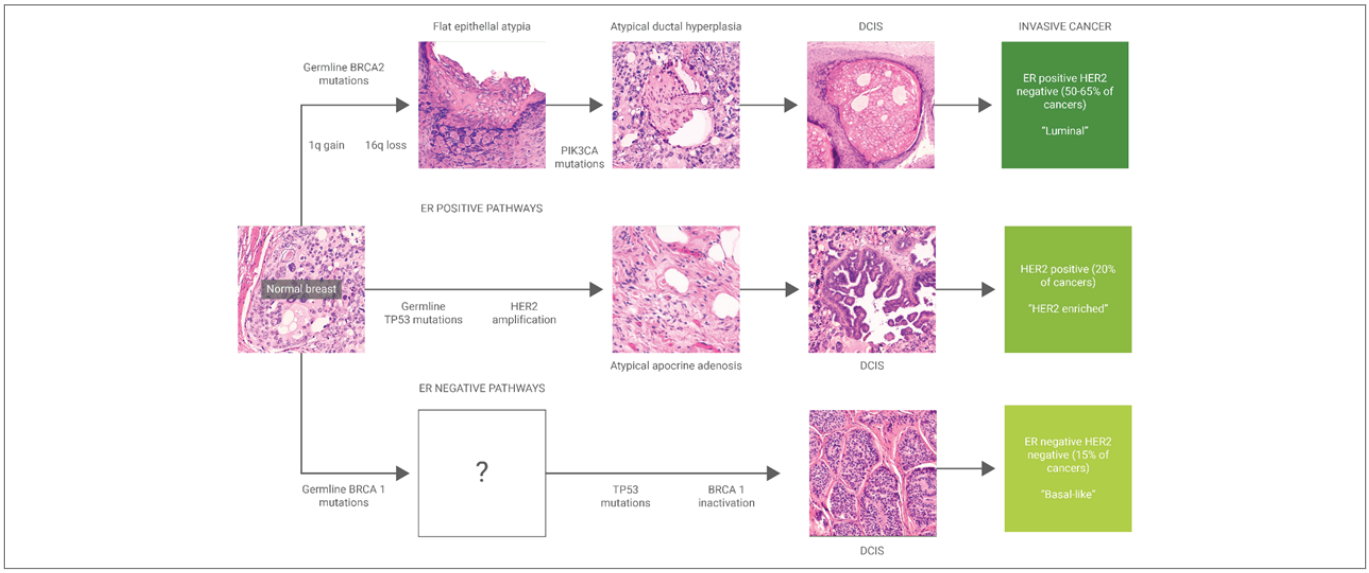
4. PATHOLOGY OF BREAST CANCERS

The majority of breast cancers start from the ductal epithelium, which extends from the nipple to the terminal duct unit situated in the breast lobule. The major histological subtypes are ductal and lobular carcinoma. The classification is shown in table below. All invasive carcinomas with the exception of medullary carcinoma are graded using the Nottingham combined histologic grade (Elston-Ellis modification of Scarff-Bloom- Richardson grading system) based on morphological features (tubule formation, nuclear pleomorphism, and mitotic count). The grade of the tumour is determined by, assigning a value of 1 (favourable) to 3 (unfavourable) for each feature, and adding together the scores for all the three categories [41,42].

5. HISTOPATHOLOGIC TYPES AND GRADES

5.1 Major Pathways of Breast Cancer Development

Three main pathways have been identified. The most common pathway (yellow arrow) leads to ER-positive carcinomas. Recognizable precursor lesions include flat epithelial atypia and atypical hyperplasia. A less common pathway (blue arrow) leads to carcinomas that are negative for ER and HER2 [42-45]. The box with the question mark indicates that no precursor lesions have been identified—perhaps because lesions progress quickly to carcinoma. The third pathway (green arrow) consists of HER2-positive cancers, which may be ER-positive or ER-negative. Amplification of the HER2 gene is also present in a subset of atypical apocrine lesions, which may represent a precursor lesion. Each molecular subtype has a characteristic gene expression profile termed luminal, HER2 enriched, and basal-like, respectively. More recently a classification based on molecular signatures has been proposed. Gene expression profiling studies have identified four major subtypes, viz. luminal A, luminal B, HER2, and basal-like. These subtypes reflect clinical features, response to therapy and treatment outcome. Luminal A and B express hormone receptors, account for 70% of the breast cancers and



WHO classification of breast cancer	
Non-invasive carcinoma	Ductal carcinoma in situ Lobular carcinoma in situ
Invasive carcinoma	Invasive ductal carcinoma Invasive lobular carcinoma Mucinous carcinoma Medullary carcinoma Papillary carcinoma Tubular carcinoma Adenoid cystic carcinoma Secretory (juvenile) carcinoma Apocrine carcinoma Carcinoma with metaplasia (metaplastic carcinoma) Inflammatory carcinoma
Paget's disease of the nipple	
The Nottingham combine histopathologic grade (G)	
GX	Grade cannot be assessed
G1	Low combined histologic grade (favourable)
G2	Intermediate combined histologic grade (moderately favourable)
G3	High combined histologic grade (unfavourable)

have a good prognosis, although luminal B are usually higher grade and may express human epidermal growth factor receptor 2 (HER2). They respond to hormone treatment (A better than B) and have a variable response to chemotherapy (B better than A). HER2 cancers (15%) have a high HER2 receptor expression and are generally ER/PR negative. They tend to respond to trastuzumab and anthracycline based chemotherapy and are associated with a poor prognosis. Basal-like breast cancers are associated with the worst prognosis. These demonstrate a high expression of basal epithelial genes, basal cytokeratins, and low expression of estrogen receptor, progesterone receptor, and HER2. These constitute 15% of the breast tumours, are often referred to as 'triple negative' tumours, have a poor prognosis and do not respond to either hormonal treatment or trastuzumab. The basal-like phenotype is commonly seen with BRCA-1 associated breast cancers. Platinum-based chemotherapy is being increasingly used to treat this subset of cancers. Although classifying breast tumours based on gene expression is of interest, a more practical classification is based on the presence of immunohistochemical markers, viz. oestrogen receptor, progesterone receptor, and HER2. These are used as surrogate markers to reflect the molecular subtypes on gene expression. Luminal (A and B) are ER/PR positive and HER2 negative, HER2 cancers are ER or PR negative and HER2 positive, while basal-like are ER, PR and HER2 negative [44].

5.2 Molecular Subtypes of Invasive Breast Cancer

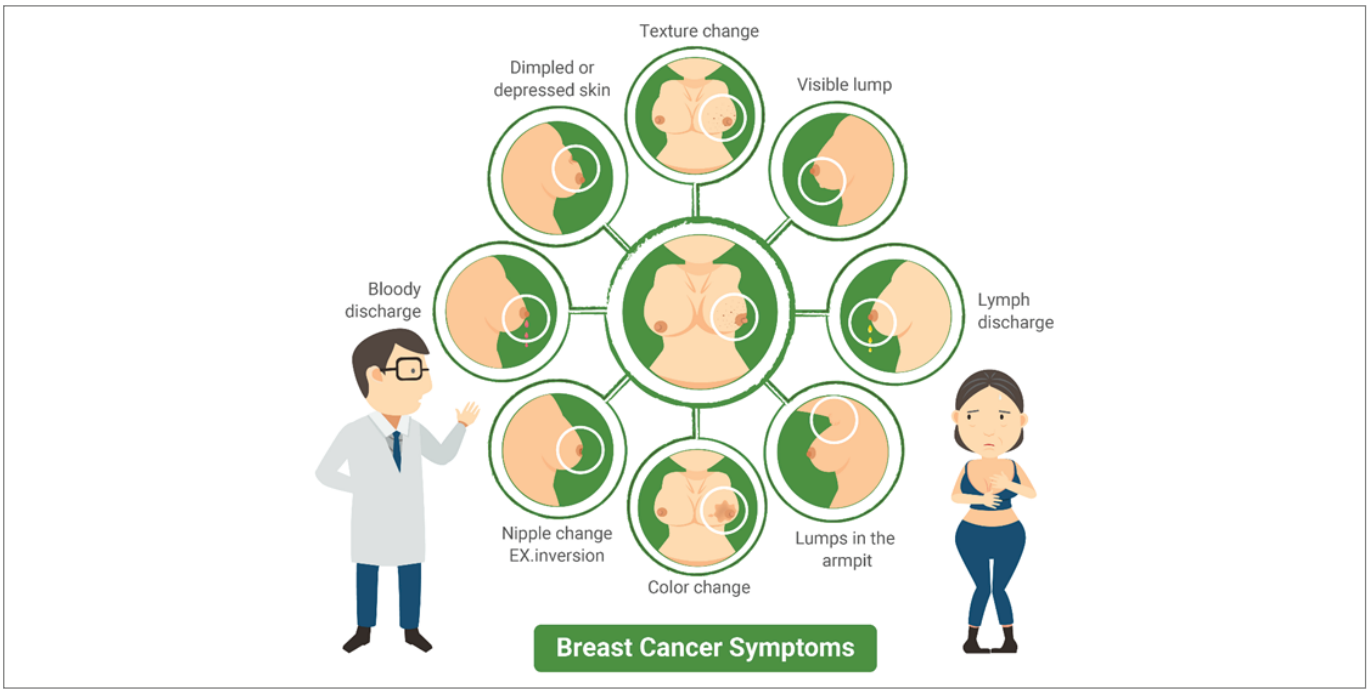
	Luminal A	Luminal B	Her-2/neu	Basal-like
Gene expression pattern	Expression (LMW) cytokeratins, and high expression of HR's and associated genes	Expression (LMW) cytokeratins, and moderate to HR's and associated genes.	**High expression of Her-2/neu.** Low expression of ER and associated genes.	High expression of basal epithelial genes, basal cytokeratins. **Low expression of ER and Her-2/neu** associated genes.
Clinical	~ **50%** of invasice brest cancer	~ **20%** of invasice brest cancer	~ 15% of invasice brest cancer	~ 15% of invasice brest cancer
ER/PR status	**ER/PR positive**	**ER/PR positive**	ER/PR negative	Most ER/PR negative
Her-2/nenu status	Her-2/neu negative	Her-2/neu expression variable (+/-)	Her-2/nenu positive (by definition)	Her-2/neu negative **("triple negative")**
Biological features	Her-2/neu negative	High proliferation thanluminal A	High proliferation	High proliferation

6. NATURAL HISTORY OF BREAST CANCER

Breast cancers may grow at different rates. However, on an average, these are considered to be slow growing. It is estimated that by the time a breast cancer lesion assumes the size of 1 cm it has been in the body for a period of 7 to 10 years [46]. It spreads by direct extension to surrounding tissue, via lymphatics and by the blood stream. The ipsilateral axillary lymph nodes are the commonest site of involvement. Other sites of involvement include the internal mammary lymph nodes especially in medial quadrant tumours and the supraclavicular nodes. Distant organs commonly involved are the lungs, liver, brain, and bones.

7. CLINICAL FEATURES

The commonest presentation is that of a lump in the breast, frequently situated in the upper outer quadrant of the breast. It may be associated with skin changes, which are subtle to begin with and become more pronounced as the lesion grows. In late disease, the classical appearance of 'peau de orange' is noted. Other symptoms include a vague lumpy area, breast pain, nipple discharge, nipple retraction, nipple eczema, swelling of the breast and a lump in the axilla [43,47]. On occasion, patients may present with symptoms suggestive of disseminated disease, such as weight loss, bone pains, ascites or headache and vomiting.



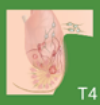
8. DIAGNOSIS

The diagnosis of breast cancer rests on a detailed history and examination. Confirmation is by pathological evaluation. This may be easily achieved by fine needle aspiration cytology (FNAC) [42]. FNAC provides a cytological diagnosis but is unableto differentiate between non-invasive and invasive cancer. A core needle biopsy or an open biopsy (incisional/excisional) provides adequate tissue for a definitive histopathological diagnosis. Imaging modalities, viz. mammography, ultrasonography and magnetic resonance imaging (MRI) provide complementary information. Mammography is a soft tissue X-ray of the breast in two directions (mediolateral and craniocaudal). It can detect microcalcifications, which may be the earliest sign of breast cancer and is the modality of choice for breast cancer screening. However, it is known to miss 10% to 15% of all breast cancers. Ultrasonography is used as an adjunct to mammography. It is especially useful in dense breasts and in young women, where mammography is of limited use. However, it is less sensitive than mammography and not recommended as a screening modality. Magnetic resonance imaging is the most sensitive technique to detect breast cancer. It is useful in ruling out local recurrence after breast conserving treatment (BCT) and in evaluation of indeterminate masses. It is the preferred modality for screening high-risk women and is used to rule out multicentric disease in patients considered for BCT [48,49].

Triple Diagnosis: To minimise mistakes, the concept of 'triple diagnosis' is used in the diagnosis of breast cancer. Based on a review of clinical findings, fine needle aspiration and imaging studies confirmed that the diagnosis of cancer is only made if all three modalities are definitely indicative of the presence of breast cancer. If there is a lack of complete agreement between all the three components, then histological proof (core biopsy/incisional biopsy/excisional biopsy) should be obtained before a definitive diagnosis of breast cancer can be made.

Staging: Following diagnosis, the stage of the disease is determined by investigations, such as serum alkaline phosphatase, liver enzymes, an ultrasound evaluation of the liver, and a chest X-ray. A CT scan of thorax and abdomen and an isotope bone scan are indicated in selected patients. A PET scan may also be performed in individualised cases, however, its routine use is not recommended at the moment. Current recommendations suggest clinical staging with mammography and liver function tests, based on which patients are classified into low risk and high risk for occult metastases and locally advanced breast cancer. The Manchester system and the TNM system (Tumour, Node and Metastases) staging system [American Joint Committee on Cancer (AJCC) classification] are used to stage the disease.

8.1 Staging of Breast Cancer (Manchester and TNM Classification):

Tumor size **T**	Tumor size <2 cm  T1	Tumor size 2-5 cm  T2	Tumor size >5 cm  T3	Tumor extends to skin or chest wall  T4
Lymph Nodes **N**	**N0** No lymph node metastasis	**N1** Metastasis to ipsilateral, movable, axillary LNs	**N2** Metastasis to ipsilateral, fixed axillary, or IM LNs	**N3** Metastasis to infraclavicular/ supraclavicular LN, or to axillary, and IM LNs
Metastasis **M**	**M0** No distant metastasis	**M1** Distant metastasis	LNs=Lymph Nodes; IM=Internal Mammary	

		Stage	Primary tumour (T)*	Regional lymph node status (L)	Distant metastasis (M)
T- Tumour		**0**	Tis	No	Mo
T1	Tumour ≤ 2 cm	**I**	T1	No	Mo
T2	Tumour ≥ 2 cm but ≥ 5 cm		T0	N1	Mo
T3	Tumour ≥ 5 cm	**IIA**	T1	N1	Mo
T4	Tumour of any size with direct extension to chest wall or skin		T2	N0	Mo
N-Lymph node		**IIB**	T2	N1	Mo
N0	No cancer in regional node		T3	N0	Mo
N1	Regional movable metastasis	**III A**	T0	N2	Mo
N2	Non-movable regional metastases		T1	N2	Mo
N3	Cancer in the internal mammary lymph nodes		T2	N2	Mo
M- Metastasis			T3	N1/N2	Mo
MO	No distant metastases	**III B**	T4	Any N	Mo
M1	Distant metastases	**IV**	Any T	N3	Mo
		IV	Any T	Any N	M1

Criteria for staging breast tumours according to the UICCICD - 10 TNM classififcation.
*Size measurements are for the tumour's greatest dimension.

9. MANAGEMENT

Management of breast cancer is based on the stage of the disease. The overall scheme is determined on whether the disease is early and localised to the breast and axillary lymph nodes (locoregional disease/ operable cancer), extensive local disease (locally advanced breast cancer [LABC]) or associated with visceral/bony metastases. Treatment is multi-disciplinary and based on a combination of local treatment (surgery and radiotherapy) and systemic treatment (chemo-, hormone, and targeted therapy).

10. EARLY BREAST CANCER/ OPERABLE BREAST CANCER

The aim of treatment is to cure, control the disease in the breast and axilla, and conserve form and function. In early disease, surgery with or without adjuvant radiation therapy is adequate for local control of the disease. If the primary tumour size is small there is a choice of surgical procedures, viz. BCT or modified radical mastectomy (MRM). The extent of surgery whether BCT or MRM is based on a detailed discussion of the advantages and disadvantages with the patient. Both approaches result in comparable results in terms of local and systemic control resulting in equal long-term survival. Data from breast conservation trials have confirmed BCT to be safe in selected patients.

Surgery: Modified radical mastectomy consists of removal of the entire breast with lymph nodes in the axilla. The extent of lymph node dissection may vary from complete clearance to less extensive procedures. More recently, the concept of sentinel lymph node biopsy has been introduced. However, this should only be undertaken in specialised centres. Breast conservative treatment (BCT) consists of a wide excision of the breast lump with negative margins and an axillary dissection (in continuity or through a separate incision). Every patient undergoing BCT is advised adjuvant radiation therapy to the breast, which is usually administered for a period of 6 weeks. Shorter durations of radiation therapy, as well as intraoperative radiation therapy are being evaluated and should be considered experimental at the moment. Oncoplastic surgery refers to the integration of plastic surgery techniques with excisional surgery.It encompasses minor techniques to maintain aesthetic breast shape and size following lump excisions, to reconstructions following mastectomy.

Radiation Therapy: This is administered in daily fractions to the breast + regional lymph node basin. The usual dose is 50 Gy in the form of external radiation (with or without a boost to the local tumour bed). Indications for post-operative radiotherapy include all patients undergoing BCT and certain patients undergoing MRM, viz. large primary lesions greater than 5 cm, multiple involved axillary lymph nodes and those with poor prognostic features in the primary lesion, viz. lymphovascular invasion, high-grade lesions [50].

Systemic Treatment: Systemic therapy is decided on the risk of developing recurrent disease. The risk is calculated on the basis of age, menopausal status, tumour characteristics (size, grade, lymphatic invasion/ vascular invasion, margins of excision), lymph node status (number of positive nodes, extra-nodal spread), hormone receptor status of the primary lesion (oestrogen, ER and progesterone, PgR receptor status) as well as the epidermal growth factor receptor Cerb B2 (Her2 neu) status. All patients who have a primary tumour which is greater than 1 cm in size and a tumour grade greater than 1 are advised some form of systemic treatment. Systemic therapy may be in the form of chemotherapy, hormone therapy, targeted therapy or a combination of these. 'Oncotype Dx' and 'Mammaprint' are molecular profiling tests which classify patients into high risk, intermediate risk, and low risk of recurrence categories with the aim of individualising treatment decisions.

Chemotherapy: Chemotherapy is administered at 3 weekly intervals. It is usually multi-drug and multi-cycle. The common adjuvant regimen used is FEC (5-fluorouracil, epirubicin, and cyclophosphamide). Taxol is increasingly used for aggressive disease. Chemotherapy usually consists of 6 cycles; however, there are alternative regimens that utilise 4 and 8 cycles. Other dose dense and dose intensive regimens are also available [44].

Chemotherapy Regimens are as Follows:

- **TAC:** Docetaxel (Taxotere) 75 mg/m^2 IV on day 1 plus doxorubicin (Adriamycin) 50 mg/m^2 IV on day 1 plus cyclophosphamide 500 mg/m^2 IV on day 1 every 3 wk for six cycles or
- **Dose-dense ACP:** Doxorubicin 60 mg/m^2 IV plus cyclophosphamide 600 mg/m^2 every 2 wk for four cycles; followed by paclitaxel 175 mg/m^2 every 2 wk with colony-stimulating factor (CSF) support (more effective than the 3-wk schedule in ER-negative or progesterone receptor (PR)-negative disease) or
- **Dose-dense ACP:** Doxorubicin 60 mg/m^2 IV plus cyclophosphamide 600 mg/m^2 every 2 wk for four cycles; followed by paclitaxel 175 mg/m^{2}every 2 wk with colony-stimulating factor (CSF) support
- **AC:** Doxorubicin 60 mg/m^2 IV plus cyclophosphamide 600 mg/m^2 IV on day 1 every 3 wk for four cycles (comparable to CMF [cyclophosphamide, methotrexate, fluorouracil]) or
- **TC:** Docetaxel 75 mg/m^2 IV on day 1 plus cyclophosphamide 600 mg/m^2 IV on day 1 every 3 wk for four cycles

For HER2-positive tumors, the following neoadjuvant regimen is administered every 3 wk for three to six cycles:

- **Pertuzumab:** 840 mg IV infusion over 60 min, then 420 mg IV infusion over 30-60 min plus
- **Trastuzumab:** 8 mg/kg IV infusion over 90 min initially, then 6 mg/kg IV infusion over 30-90 min plus
- **Docetaxel:** 75 mg/m^2 IV infusion initially; may increase to 100 mg/m^2 IV infusion if initial dose is well tolerated

Hormone Therapy: Hormone therapy is considered in hormone receptor positive tumours. Pre-menopausal women are advised tamoxifen 20 mg per day and post-menopausal women either tamoxifen (20 mg) or one of the aromatase inhibitors, e.g. anastrozole 1 mg per day, letrozole 2.5 mg per day, exemestane 25 mg per day. Hormone therapy is advised for a period of 5 years. Surgical or medical oophorectomy using [luteinising hormonereleasing hormone (LHRH) agents] provides another avenue of treatment in premenopausal women who are hormone receptor positive. An oophorectomy may be combined with tamoxifen or an aromatase inhibitor to provide 'complete receptor blockade'.

Targeted Therapy: Targeted therapy in the form of monoclonal antibody to the epidermal growth factor receptor (EGFR, trastuzumab) may also be added as adjuvant treatment in patients who overexpress the Cerb B2/HER2 neu receptor. The treatment is recommended for a period of 1 to 2 years at 3 weekly intervals. During chemotherapy and targeted therapy, it is essential to monitor patients carefully, as there is significant toxicity associated with these agents.

11. LOCALLY ADVANCED BREAST CANCER

The aim of treatment is to control micrometastatic disease and to downstage the size of the primary lesion to increase cure rates and to decrease the extent of local surgical procedures. This is achieved by systemic treatment. Both chemotherapy and primary hormone treatment are associated with excellent response rates. Following downstaging of the disease, surgery is undertaken to optimise local control. This is followed by further systemic treatment (chemo-/hormonal/targeted therapy) and radiotherapy. An important development has been the feasibility of breast conservative surgery in patients who respond well to systemic therapy. Numerous studies have demonstrated that it is safe to offer breast conservation to patients whose tumours have adequately decreased in size following primary systemic therapy. However, the option should be carefully discussed with the patient, and if there is any doubt, it is probably better to proceed with a mastectomy.

12. METASTATIC BREAST CANCER

In the presence of metastasis (visceral or bone involvement) the primary modality of treatment is systemic (chemotherapy or hormonal therapy) as the aim is to control disseminated disease. The accepted practice is to start with systemic chemotherapy. Recent data indicate that hormonal treatment may be effective in carefully selected postmenopausal women who are strongly hormone receptor positive. Once the metastatic disease is adequately controlled, attention is directed towards control of the disease in the breast. Treatment options

include mastectomy or breast conservation in selected cases, viz. small primary lesion presenting with metastases. If the mastectomy is associated with a loss of skin cover, it may be accompanied by reconstructive measures. Radiation therapy is administered to patients with extensive local disease and all patients who undergo breast conservation.Palliative radiation therapy to sites of bony metastases is useful in controlling the disease. Bisphosphonates, viz. zoledronic acid infusions are also administered at regular intervals for control of skeletal symptoms and to prevent fractures.Surgery may be indicated for stabilisation of pathological fractures or vertebral collapse.

13. PROGNOSIS

The prognosis of breast cancer does not depend on its chronological age but on its metastatic potential. A range of factors including tumour size grade, receptor status, proliferation markers and oncogene product measurements are assessed. Microarrays are used to prognosticate breast tumours into high, intermediate and low risk, based on their genetic profiles. The best clinical indicators of prognosis are lymph nodal status and tumour size. Survival ranges from 80% to 90% at 10 years for stage I disease, 60% to 70% for stage II, 40% to 55% for stage III, and 10% to 20% for stage IV disease [52].

14. HIGH-RISK STATES AND BREAST CANCER PREVENTION

Women who are carriers of BRCA 1 and 2 mutations, or who have a family history consistent with genetically transmitted breast cancer are considered to be at a higher risk than the general population. Presence of lobular carcinoma in situ or atypical hyperplasia in a breast biopsy is also considered as an indicator of a high-risk state. Assessment of such patients involves a detailed evaluation of the level of risk using certain "risk models" (e.g. Gail, Brcapro, etc.). Based on the level of risk, the patient is counselled. Management options include increased surveillance, medical interventions (viz. anti-oestrogen therapy) or surgical interventions such as a prophylactic oophorectomy or bilateral prophylactic mastectomy.

15. BREAST CANCER SCREENING AND METHODS FOR EARLY DIAGNOSIS

The main methods for the early diagnosis of breast cancer include screening mammography of the breasts, clinical breast examination, and breast self-examination (BSE). Current recommendations of the American Cancer Society for early diagnosis include BSE once a month starting at the age of 20, clinical breast examinations about once every 3 years for women in their 20s and 30s and yearly for those over 40 years and yearly mammograms after the age of 40. Magnetic resonance imaging screening is recommended for women with a 20% to 25% or greater life-time risk of breast cancer.

16. CONCLUSION

An overall thought should consolidate quality ensured screening, capable logical imaging, histopathological assessments, mind blowing cautious methodology, proficient radiotherapy, pharmacological treatment including the main cutting edge treatment procedures, and master advancement over the entire treatment length and past. Unsurprising execution of the standard proposition makes it possible to upgrade quality along the treatment tie and can incite a reduction in the terribleness and mortality of chest dangerous development. It is major for execution to be amidst interdisciplinary and cross-sectoral appraisal inside the setting of prosperity organizations research.

Conflict of Interest

Not Declare

Ethical Consideration

None

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