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Endocrine Therapy in Breast Cancer: The Neoadjuvant, Adjuvant, and Metastatic Approach

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Objectives: *To review the rationale for endocrine therapy in the neoadjuvant, adjuvant, and metastatic breast cancer setting and to highlight clinical considerations unique to this treatment.*

Data Sources: *Contemporary literature, clinical guidelines, and national statistics.*

Conclusion: *Endocrine therapy represents an important strategy in the management of both early and advanced hormone positive breast cancer. Additional research is required to better define the role of neoadjuvant therapy and the optimal duration of treatment.*

Implications for Nursing Practice: *Nurses play a pivotal role in the identification and management of endocrine therapy-associated symptoms.*

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Prompt symptom intervention may improve therapy adherence and ultimately, may improve long-term disease outcomes.

KEY WORDS: *Endocrine therapy, breast cancer, aromatase inhibitors, tamoxifen*

Breast cancer is the second most common cancer overall and the most common cancer among women in the United States, with an estimated 232,670 new cases in 2014.[1] The overall 5-year survival rate is high at approximately 89%, but this rate is reduced to 25% in the presence of metastatic disease.[1] Breast cancers may arise from the milk ducts, known as ductal breast cancer, or from the milk-producing lobules of the breast, known as lobular breast cancer. Ductal and lobular carcinomas can be further classified as in situ or invasive (Fig. 1). In situ tumors are those that do not spread from their origin and represent only 20% of all breast cancers. Ductal carcinoma in situ is both the earliest stage of breast cancer and the most common type of non-invasive breast cancer, representing 83% of all in situ carcinomas.[2] Lobular carcinoma in situ, also known as lobular neoplasia, is not considered malignant, but patients with lobular carcinoma in situ are at risk for the future development of a separate invasive breast cancer. In contrast, invasive breast cancers occur much more frequently, spread to the surrounding tissue, and have the potential to metastasize to other areas of the body.

Breast cancer can be further defined by its growth response in the presence or absence of the hormone receptors (HR), estrogen and/or progesterone. These receptors can be targeted by anti-endocrine therapies, which represent the mainstay of treatment for estrogen receptor (ER)/progesterone receptor-positive (+) breast cancer. In addition to endocrine receptor status, breast cancer is also differentiated by the presence or absence of a mutation in the *human epidermal growth factor receptor 2 (HER2)* gene, which encodes for proteins that promote the growth of cells, including malignant cells.[3]

ENDOCRINE THERAPY

In HR + breast cancer, exposure to endogenous estrogen, which includes estrone, estradiol, and estriol, results in dimerization of the ER and promotes estrogen-regulated gene transcription. In premenopausal women, the ovaries are the main source of estrogen production. In postmenopausal women, androgens released by the adrenal glands are converted into estrogen by aromatase primarily in the adipose tissue and the muscle. Endocrine therapy functions by blocking the effect of estrogen at the receptor level or by inhibiting estrogen production. Several classes of endocrine therapy have been developed to date and these include selective estrogen receptor modulators, selective estrogen receptor downregulators, aromatase inhibitors (AIs), luteinizing hormone releasing agonists, high-dose estrogens, and targeted therapies (Table 1).[4]

Tamoxifen (TAM) is a selective estrogen receptor modulator because of its tissue specific activity and is indicated in pre- and post-menopausal women. TAM exerts anti-estrogenic activity in the breast and vaginal mucosa through partial inhibition of ER dimerization. However, TAM also exerts an estrogenic effect on the endometrium (promoting endometrial hyperplasia), the coagulation system (promoting thromboembolic events), bones (preventing osteoporosis), lipids (preventing hyperlipidemia), and the liver (promoting hepatotoxicity). In contrast, fulvestrant completely inhibits ER dimerization and ultimately results in downregulation of ER expression. Fulvestrant is anti-estrogenic in all tissue; it is categorized as a selective estrogen receptor downregulators.[4-8]

AIs block the synthesis of estrogen through inhibition of aromatase. Anastrozole and letrozole are non-steroidal AIs with reversible inhibition of aromatase, whereas exemestane is a steroidal AI with irreversible inhibition of aromatase requiring production of new aromatase to overcome inhibition. AIs are indicated in postmenopausal women; in

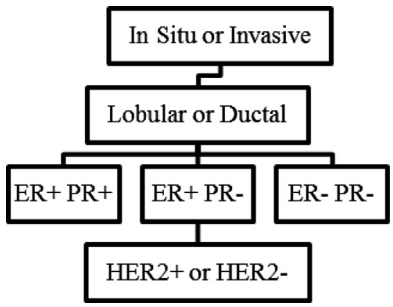

FIGURE 1. **Breast cancer classification.**

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