Prospective nursing study of breast cancer lymphedema: Exploring possible relationships with tamoxifen therapy
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Lymphedema (LE) is a lifetime risk for breast cancer survivors. This predisposes individuals to infection, possibly life-threatening, leads to difficulties in clothing fit and activities of daily living, and also affects self-esteem, self-identity, and quality of life. Tamoxifen, considered a first-choice adjuvant therapy drug following breast cancer treatment, has been shown to halve the cancer recurrence risk. Tamoxifen is known to influence fluid and electrolyte balance with fluid retention (32%) being one of the most common adverse effects of the drug. Theoretically, it may act at the cellular level by increasing capillary membrane permeability, thus increasing the interstitial fluid movement and workload of a lymphatic system already compromised due to surgery and, often, radiation. Current literature has not reported a definitive association between tamoxifen use and LE occurrence. The research goal is to explore tamoxifen-related variables in LE occurrence and whether or not LE occurrence is higher in breast cancer survivors who take tamoxifen. A secondary analysis of data from an established National-Institutes-of-Health-funded parent study will be performed. The NIH study includes more than 200 persons newly-diagnosed with breast cancer who were consented, enrolled, and assessed at pre-op, post-op, and followed for 30 months. In the absence of a “gold standard,” the study defines four measurements of LE in exploring approaches to assessing and diagnosing post-breast cancer LE. In this proposal, data from a self-report of symptoms and tamoxifen use (≥ 6 months) are derived from the nurse interview using a validated measurement tool and medical record review. Data from limb volume estimation are derived from reliable and valid perometry measurement. Relationship indicators between tamoxifen and LE occurrence would provide an early identifier for at-risk individuals, lay groundwork for targeted interventions, and justify future research examining the underlying physiological cellular mechanism associated with LE emergence in the presence of tamoxifen.