BRIEF COMMUNICATION

ARM EDEMA AFTER TREATMENT FOR BREAST CANCER

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Ipsilateral arm edema after treatment for breast cancer remains a troublesome morbid condition. Yet, only a variable fraction (2-25%) of treated patients later develop arm edema. Because the pathomechanism therefore is not entirely clear, some have postulated an occult but compensated deficiency of lymphatics (1) which becomes unmasked (decompensated) after axillary dissection and/or radiotherapy (2-7). From our experience between 1984-1992 with 142 patients with arm edema after treatment of breast cancer, we attempted to determine the basis for this complication in terms of the type of treatment before the onset of arm edema, the interval between original therapy and the development of arm swelling and the factors responsible for aggravating arm edema. Of the 142 patients with arm edema, 13 or 9.2% received only operative treatment, 121 or 85.2% received both surgery and radiotherapy and 8 or 5.6% received only radiotherapy. Twenty-six patients or 23.2% developed lymphangitis as the precipitating factor in the onset of arm edema. Seventyfour patients or 52.1% had at least one subsequent episode of lymphangitis with uniform worsening of the arm edema. Twelve patients or 8.5% had concomitant axillary vein thrombosis corroborated by venography. In each, the arm edema suddenly worsened without cellulitis-lymphangitis or evidence of cancer recurrence. Thirty-four patients or

23.9% had significant functional impairment of the ipsilateral arm after operative treatment and complemental radiotherapy or after radiotherapy alone. This functional disability was gradual and progressive and these individuals had noticeable worsening of arm edema which was typically intractable to physiotherapy. Thirty patients or 22.5% had worsening of arm edema without lymphangitis, cellulitis, or functional impairment of the arm. These patients after 6 months manifested recurrence of mammary cancer. Nine patients or 6.3% demonstrated diffuse cancer infiltration of the skin of the unilateral arm (cutaneous permeation) with rapid development of fibrous edema. One hundred three patients or 75.5% developed arm edema within one year of the original treatment and 39 or 27.5% thereafter. No patient developed a (lymph)angiosarcoma (Stewart-Treves syndrome).

From this experience, we conclude that the key precipitating factors in the development of arm edema after treatment of breast cancer include in the order of importance: lymphangitis-cellulitis, severe functional impairment of the arm, recurrence of the cancer, axillary vein thrombosis. Most patients with arm edema had undergone both operative and radiation treatment and the majority displayed arm edema within the first year after definitive therapy.

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