Osteoporosis in Men Treated With Androgen Suppression Therapy for Prostate Cancer

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Androgen suppression therapy through the injection of luteinizing hormone-releasing hormone (LHRH) agonist agents has been the mainstay of therapy for patients with advanced or metastatic prostate cancer. Its use has expanded to include patients with prostate-specific antigen recurrence, also known as biochemical relapse (Moul, 1998; Zietman, 1996). The Agency for Health Care Policy and Research (1999) estimated that total 1994 Medicare expenditure for treatment of prostate cancer was almost $1.5 billion, and almost one-third of this was spent on androgen suppression therapy using LHRH agonists. Administration of LHRH agonists, such as goserelin and leuprolide, was the 13th largest category of physician reimbursement by Medicare (Agency for Health Care Policy and Research). In the United States, about 117,000 men with advanced prostate cancer were treated with LHRH agonist therapy in 1996; as the prevalence of advanced prostate cancer rises, its use and subsequent financial costs also are expected to increase (Landis, Murray, Bolden, & Wingo, 1998). Furthermore, men with biochemical relapse who generally are younger and asymptomatic now are faced with long-term use of LHRH agonist therapy. Although patients may tolerate immediate side effects, such as hot flushes, impotence, and loss of libido, healthcare professionals must address new challenges and issues surrounding long-term sequelae of LHRH agonist therapy. The effects of LHRH agonist therapy on bone metabolism, the risk of osteoporosis among men with prostate cancer, and strategies to promote quality of life for these men must be studied.

Men with advanced or metastatic prostate cancer commonly receive long-term treatment with luteinizing hormone-releasing hormone (LHRH) agonist therapy. This prolonged treatment causes a hypogonadal state of chronic testosterone deficiency. Similar to estrogen deficiency in postmenopausal women, testosterone deficiency among these men negatively affects bone metabolism through a complex self-regulating, negative feedback system and subsequent reduction in bone formation. If left undetected or untreated, the risk for osteoporosis rises. Osteoporosis increases the likelihood of fracture, especially of the hips. Researchers are studying the effects of LHRH agonist therapy on osteoporosis and other related conditions to determine whether interventions, such as pharmacologic agents (e.g., bisphosphonates), dietary supplements (e.g., calcium, vitamin D), and exercise, can slow or prevent the process and assist healthcare providers in knowing how to counsel patients. Current recommendations are found in the literature on glucocorticoid-induced and menopausal osteoporosis. Nurses need to stay abreast of current knowledge in this area, as it is expanding rapidly.

Osteoporosis

Osteoporosis is the most common bone disorder in the United States today, affecting about 15–20 million individuals (Mundy, 1995). A metabolic disorder characterized by decreased bone mass and mechanical support of the skeleton, osteoporosis occurs when the rate of bone resorption greatly exceeds the rate of bone formation. The primary bones affected are the hips, pelvis, wrists, and vertebral column. Although osteoporosis occurs more commonly in postmenopausal women, more than two million men have been diagnosed with the disease (National Osteoporosis Foundation, 1995). Approximately 18 million adults have decreased levels of bone mass, which places them at risk of developing osteoporosis (Lindsay, 2001). The incidence of osteoporosis increases significantly with aging, particularly among men and women over the age of 70 (Zilkoski & Morrow, 1987). Osteoporosis is a major risk factor in fractures (National Institutes of Health [NIH], 2000). When it results in fractures,