Advances in the Treatment of Bone Metastases

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Bone metastases are common in many cancers. They are the most common cause of cancer-related pain (Mercadante, 1997) and a major cause of morbidity in patients with cancer (Rubens, 1998). Malignancies that most frequently metastasize to the bone are breast, prostate, and lung cancers. Multiple myeloma, osteosarcoma, and Ewing’s sarcoma are primary skeletal tumors but nonetheless have the same destructive effect on bone as metastatic disease (Mercadante). Although metastases are the focus of this article, from a standpoint of therapeutic interventions, similar principles of treatment apply to primary skeletal malignancies.

Nurses play a vital role in the assessment and management of metastatic bone disease. Early identification of skeletal lesions not only aids in early intervention and better pain control but also can prevent complications, such as hypercalcemia, myelosuppression, pathologic fractures, and spinal cord compression.

This article reviews bone physiology, the pathophysiology associated with bone metastases, strategies of medical management, and nursing implications for managing patients with metastatic bone lesions. Areas of future research in bone metastases also are described.

Physiology of Bone

The human skeletal system consists of bones and articulations that provide structural support and allow movement. Bone also is essential in maintaining hematopoiesis and mineral homeostasis (Fisher, Mayer, & Struthers, 1997). Bone is composed of minerals (cortical portion) and collagen (cancellous portion). Cortical bone is very dense and compact and constitutes about 85% of the total skeletal mass. Cancellous bone (15%) is more porous and spongy, and thereby is more susceptible to disease processes such as osteoporosis and malignant metastases (Mundy, 1995). It is found in the metaphysis of long bones such as the femur and inside of flat bones such as the ilium, cranium, and ribs.

Once the skeleton has reached maturity, regeneration (referred to as remodeling) occurs on a continuous basis (Body, 2000). At the cellular level, bone remodeling is regulated by an intricate balance of varying levels of simultaneous osteoblastic and osteoclastic activity (see Figure 1). Osteoblasts (bone-forming cells) are responsible for the production of collagen and other proteins to synthesize the bone matrix. Osteoclasts (bone-eroding cells) mediate resorption of the bone matrix by binding to the bone surface, secreting citric and lactic acids, then dissolving and digesting bone minerals and collagen (Body). Osteocytes, another type of bone cell, are transformed osteoblasts that are believed to participate in supplying nutrients to the bone matrix (McCance & Huether, 1998).

Many hormones and cytokines are involved in the modulation of bone formation and resorption. These modulators work at either a local or systemic level and may increase or decrease the activity of osteoclasts (resorption) and osteoblasts (formation).

Pathophysiology of Bone Metastases

The cortical (calcified) matrix stores many growth factors. These can nourish cancer cells that have settled in the bone, thus providing a fertile microenvironment for the continued growth of cancer cells. Because cancellous

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