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Opinion Article

Pathophysiology of Skeletal Metastases and Its Diagnosis in Combination of Clinical and Radiological Findings

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DESCRIPTION

Bone metastases are a common complication of advanced cancer, occurring in up to 70% of patients with breast and prostate cancer, and in up to 30% of patients with lung, renal, or gastrointestinal tumors. Skeletal metastases have a profound impact on patients' quality of life, causing bone pain, pathological fractures, spinal cord compression, and hypercalcemia.

Pathophysiology

Skeletal metastases result from the hematogenous spread of cancer cells to the bone marrow, where they can establish a microenvironment that supports their growth and survival. The process of bone metastasis involves a complex interplay between cancer cells, osteoblasts, osteoclasts, and the extracellular matrix.

Osteoblasts are bone-forming cells that produce collagen and other proteins that form the bone matrix. They also secrete factors such as Osteo Prote Gerin (OPG) and Receptor Activator of Nuclear Factor-Kappa B Ligand (RANKL), which regulate the activity of osteoclasts. Osteoclasts are bone-resorbing cells that break down the bone matrix, releasing calcium and other growth factors that promote tumor growth.

Cancer cells can alter the balance between osteoblasts and osteoclasts by producing factors such as ParaThyroid Hormone-related Protein (PTHrP), which stimulates osteoclast activity and inhibits osteoblast function. Other factors produced by cancer cells, such as Transforming Growth Factor-beta (TGF-beta), interleukin-6 (IL-6), and Vascular Endothelial Growth Factor (VEGF), can also promote osteoclast activity and bone resorption. The net effect of these interactions is the destruction of bone tissue and the formation of osteolytic or osteoblastic lesions. Osteolytic lesions are characterized by the destruction of bone tissue and the release of calcium into the bloodstream, leading to hypercalcemia. Osteoblastic lesions are characterized by the deposition of new bone tissue, leading to bone sclerosis and fractures.

Diagnosis

The diagnosis of skeletal metastases is based on a combination of clinical and radiological findings. The most common presenting symptom of bone metastases is pain, which can be localized or diffuse and may be worse at night or with activity. Other symptoms include pathological fractures, spinal cord compression, and hypercalcemia.

Imaging studies such as X-ray, Computed Tomography (CT), Magnetic Resonance Imaging (MRI), and bone scan are used to detect bone metastases. X-ray is the simplest and most widely available imaging modality, but it has low sensitivity for detecting early bone metastases. CT is more sensitive than X-ray for detecting bone metastases and can also provide information about the extent of bone involvement. MRI is the most sensitive imaging modality for detecting bone metastases and can also provide information about the spinal cord and surrounding soft tissue structures. Bone scan is a nuclear medicine test that uses a radioactive tracer to detect areas of increased bone turnover, which can indicate the presence of bone metastases.

Biopsy of bone lesions is not always necessary for the diagnosis of bone metastases, but it may be necessary to differentiate between benign and malignant bone lesions, to identify the primary tumor, or to guide treatment decisions.

Management

The management of skeletal metastases is multidisciplinary and involves a combination of systemic therapy, local treatment, and supportive care.

Systemic therapy

Systemic therapy for bone metastases includes chemotherapy, hormone therapy, targeted therapy, and immunotherapy. The choice of systemic therapy depends on the type of cancer, the extent of bone involvement, and the patient's overall health status.

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