

BONE PAIN



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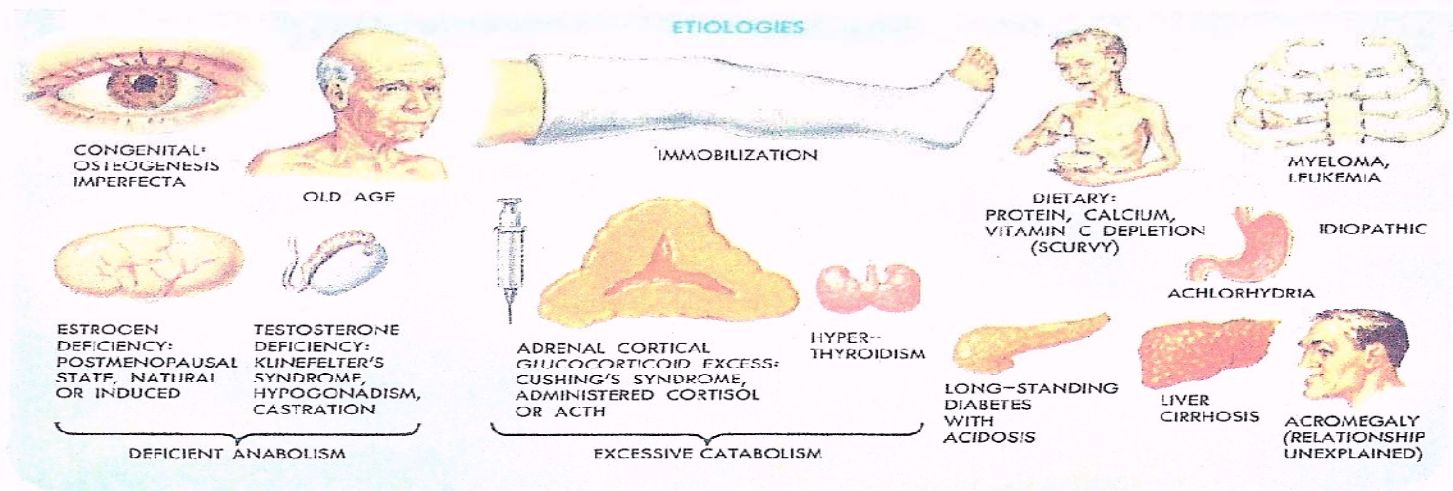
LEARNING OBJECTIVES

- REVIEW BRIEFLY THE CAUSES OF BONE PAIN
- REVIEW PATHOPHYSIOLOGY
- REVIEW MECHANISMS
- REVIEW TREATMENTS
- LOOK TO THE FUTURE

CAUSES OF BONE PAIN

- ARTHRITIC CONDITIONS
- PAGET'S DISEASE
- SICKLE CELL DISEASE
- BONE CANCER





Bone remodeling replaces approximately 20% of bone tissue annually. This remodeling occurs in 5 cycles with each cycle lasting 4-8 months. All of the above conditions impact this process in one or more cycles.

PATHOPHYSIOLOGY

- BONE PAIN ORIGINATES FROM THE PERIOSTEUM LINING BONES AS WELL AS FROM THE BONE MARROW THAT RELAY NOCICEPTIVE SIGNALS TO THE BRAIN CREATING THE SENSATION OF PAIN.
- BONE TISSUE IS INNERVATED BY BOTH MYELINATED AND UNMYELINATED SENSORY NEURONS.

MECHANISMS OF PAIN

- BONE CANCER IS ONE OF THE MOST DREADED SERIOUS FORMS OF PAIN CREATED BY DESTRUCTION OF BONE TISSUE BY MALIGNANT CELLS AS WELL AS MANY CHEMICAL REACTIONS INDUCING INFLAMMATORY RESPONSES AND TRIGGERING SIGNALS VIA THE SPINAL CORD. INFLAMMATORY LIPIDS (PROSTAGLANDINS) ACTIVATE NOCICEPTORS WITHIN THE BONE.

RANK LIGAND

- THE RANK LIGAND PLAYS A ROLE IN BONE LOSS DUE TO OSTEOPOROSIS AND DESTRUCTIVE CHANGES DUE TO CANCER BY AFFECTING THE WAY IN WHICH OSTEOPROTEGERIN (OPG) BALANCES BONE GROWTH AND LOSS

ORIGINS OF BONE CANCER PAIN

- SENSORY AND SYMPATHETIC INNERVATION OF THE BONES WAS PREVIOUSLY NOT WELL DELINEATED DUE TO HARSH PROCESSING TECHNIQUES INVOLVING FORMALDEHYDE. In the 1960s we began using more sophisticated preservative agents such as gluteraldehyde that preserved the delicate neuron structures within the bone and bone marrow enabling us to begin to look at mechanisms of pain.

- INITIALLY LOOKING AT ANIMAL MODELS IDENTIFIED NEURAL FIBERS TRAVELING WITH BLOOD VESSELS VIA THE HAVERSIAN AND VOLKMANNS' CANALS AND LOCATING THE PATTERNS OF INNERVATION IN THE PERIOSTEUM, MINERALIZED BONE AND BONE MARROW CAVITY.
- Mach, et al, Sensory and sympathetic innervation of the mouse femur, Neuroscience,113, p155-166

MOLECULAR MECHANISMS OF PAIN

- Pain is the first presenting sign of cancer in over 50% of patients. The mechanisms can be due to pathologic fractures, hypercalcemia, and direct invasion with malignant cells.
- Glial cells in the CNS such as astrocytes can increase responsiveness of pain transmission to neurons in the spinal cord and increase pain sensation-see diagram

CANNABOID RECEPTORS

- Localization of cannabinoid receptors within the superficial dorsal horn of the spinal cord has led to evaluations of alleviation of bone cancer pain via the CB1 activation. Pain can be evoked by movement or just spontaneous. Activation of the cannabinoid receptors reduced bone cancer-related behaviors that included movement related and not. Minimizing the inflammatory response was crucial.

NEWER CONCEPT IN PAIN MANAGEMENT

- We as osteopathic physicians have always been sensitive to musculoskeletal responses to pain and its effect on controlling pain. Microcurrent electrotherapy has been available for over 50 years, but recently studies have identified benefits in pain management that would complement manipulation and effectively control pain via reduction of inflammatory substances and breaking the spasm cycle

- References published in 2004 and 2010 by Carolyn McMakin, et al. in Journal of Bodywork and Movement Therapies have brought attention to the decrease in inflammatory cytokines associated with pain and improvement in sensations of pain that merit a second look at how we approach pain safely and effectively as we move into the next generation of pain management

QUESTIONS AND SUMMARY