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clude the induction of RANKL, which is a powerful osteoclast activator. When RANKL is present, it blocks the apoptosis of mature osteoclasts—basically it just keeps on chewing away on bone. OPG is a decoy receptor for RANKL. It's the shut-off mechanism. When it is present and able to bind to RANKL, the cell-to-cell signaling between marrow stromal cells and osteoclast precursors is inhibited, and thus osteoclasts are inhibited. We hypothesized that the relationship between the induction and inhibition of osteoclasts [in JDM], as measured by the serum RANKL:OPG ratio would be off balance. Our data confirm this: The RANKL in the JDM children was elevated and the decoy protein levels were decreased compared with the healthy children—the osteoclasts were being pushed to damage their bone.

**RN:** What are the clinical implications of these findings?
**Dr. Pachman:** The lack of normal bone mineral accretion leads to a reduction in lumbar spine BMD *z* scores. The longer the duration of untreated disease, the more likely the lumbar spine BMD is going to be reduced, suggesting that early diagnosis and effective treatment is critically important for maintaining bone health. The effects of BMD are hard to reverse, so the strategy has to be 'hit fast and hit hard' with early intervention.

**RN:** How does treatment for JDM alter the RANKL:OPG ratio?
**Dr. Pachman:** Treatment for the disease is focused on suppressing inflammation. Our current challenge is to identify reliable indicators of continued immune activation for all stages of JDM to guide this immunosuppressive therapy. In turn, the RANKL:OPG ratio drops, suggesting a normal balance in the osteoclast induction and inhibition process. The bone is no longer being destroyed, but the existing damage is not being reversed.

**RN:** In terms of optimizing bone health, what early intervention strategies should be employed?
**Dr. Pachman:** We are very enthusiastic about giving exogenous calcium and vitamin D to help prevent fractures in these children. At diagnosis, we routinely start smaller kids on 600 mg of calcium and 400 IU of vitamin D and we start larger children on 1,200 mg of calcium and 800 IU of vitamin D. This is more vitamin D than is found in some of the calcium/vitamin D combination preparations. This is important because the drugs we use to treat the disease, particularly steroids, prevent the absorption of calcium and the vitamin D acts as a magnet. Since we've been starting children on calcium and vitamin D, we've seen a substantial drop in the fracture rate among our JDM patients. If the duration of untreated disease has been longer than 4 months, they are more likely to have a bone density greater than –1.5 standard deviations from normal and therapy with the bisphosphonate pamidronate or other agents that suppress bone resorption should be considered.

**RN:** Is routine bone scanning warranted in JDM, and if so, how often should it happen?
**Dr. Pachman:** DXA [dual x-ray absorptiometry] should be arranged for all children with JDM at the time of diagnosis and should be repeated annually, especially if decreased bone matrix is identified. For children with untreated disease duration of more than 4 months who have severe bone loss, especially those who have started specific intervention therapy for decreased bone mass, DXA should probably be conducted twice per year to document that the proper goal is being achieved. ■

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*By Diana Mahoney, New England Bureau*

## FYI

### Pediatric Rheumatic Diseases

An informational CD-ROM for physicians who treat children with rheumatic diseases is being offered free of charge from the National Institute of Arthritis and Musculoskeletal and Skin Diseases (a division of the National Institutes of Health) in partnership with the Arthritis Foundation. For a copy, call 877-226-4267.

### Pain Management Guide

The American Pain Society, in collaboration with the Greater Philadelphia Pain Society, is offering "Pain Control in the Primary Care Setting," a new guide for the management of chronic noncancer pain in adults and children. The handbook contains evidence-based and practical strategies for diagnosing and treating pain. For more information, visit the American Pain Society at www.ampainsoc.org.

IN THE TREATMENT OF OSTEOARTHRITIS (OA) KNEE PAIN

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SYNVISC® HYLAN G-F 20 A STEP AHEAD

SYNVISC® (hylan G-F 20) is indicated for the treatment of pain in osteoarthritis (OA) of the knee in patients who have failed to respond adequately to conservative nonpharmacologic therapy and simple analgesics, eg, acetaminophen.

In clinical trials, the most commonly reported adverse events were transient local pain, swelling, and/or effusion in the injected knee. In some cases, these symptoms have been extensive. Other side effects such as rash have been reported rarely. SYNVISC is contraindicated in patients with known hypersensitivity to hyaluronan products or patients with infections in or around the knee. Use caution when using SYNVISC in patients allergic to avian proteins, feathers, or egg products; who have evidence of venous or lymphatic stasis in the leg to be treated; or who have severe inflammation in the knee joint to be treated. Patients should be advised to avoid strenuous or prolonged weight-bearing activities after treatment. Strict adherence to aseptic technique must be followed to avoid joint infection. The safety and effectiveness of SYNVISC in children and in pregnant or lactating women have not been established. It is unknown whether SYNVISC is excreted in human milk.

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