Young Anorexics Risk Long-Term Bone Damage

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TAMPA — Anorexia nervosa reduces bone mass and puts young women at risk for early onset of osteoporosis, just at the time when they should be building peak bone mass, Dr. Steven Crawford said at the annual meeting of the International Society for Clinical Densitometry.

Anorexia nervosa is one of the most common psychiatric diagnoses in adolescent women, with the age of onset showing bimodal peaks at ages 14 and 18 years. In addition to loss of bone density, patients can suffer cardiovascular problems, muscle loss and weakness, severe dehydration, anemia, and leukopenia. Female anorexic patients are amenorrheic. Anorexia nervosa leads to a sevenfold increase in fracture risk. Of adult women with anorexia nervosa, 38% have osteoporosis, and 50% have a bone mineral density (BMD) level below the fracture threshold.

The extent of bone damage is directly affected by the severity of malnutrition and the disease duration. Consequences are more severe when disease onset occurs during the time of peak bone development. About 60% of total bone mass is attained in the growth spurt that normally occurs in adolescence, and skeletal growth essentially is complete by age 18.

"Bone mineral density is lower when anorexia nervosa begins in adolescence than when it occurs in adult life, even when the duration of illness is comparable," said Dr. Crawford, a clinical psychiatrist at the Center for Eating Disorders, Sheppard Pratt Health System, Baltimore.

Pathophysiologically, low bone density in anorexia nervosa results from multiple factors, including undernutrition, hypogonadism, altered levels of bone-essential hormones and growth factors, excessive exercise, and hypercortisolism. Undernutrition in anorexia nervosa leads to decreased levels of the sex hormones that are critical for bone development.

Levels of insulinlike growth factor-I (IGF-I) and growth hormone normally increase during puberty, and stimulate bone anabolism. In anorexic patients, IGF-I levels decrease, and patients acquire growth hormone resistance. Lack of calcium may prevent bone remodeling normally stimulated by exercise, and hypogonadism may impair the function of osteocytes that normally are activated by exercise.

Low BMD occurs at all skeletal sites in anorexic patients, affecting both trabecular and cortical bone. The spine is more likely to be affected than the hip. In addition to decreased BMD, another factor that contributes to bone fragility is decreased bone size. Patients with anorexia nervosa develop smaller bones in the vertebral body and femoral neck, compared with normal patients. Osteoporosis contributes to stress fractures in weight-bearing bones and vertebral fractures, leading to chronic back pain and reduced height.

Dr. Crawford recommends a routine bone density scan in all patients with anorexia nervosa at disease onset and at least every 2 years thereafter. Restoration of normal weight can improve BMD in anorexic patients, but bone loss may continue, with bone restoration taking at least 21 months.

Bisphosphonates are not approved for treatment of premenopausal women. Moreover, the increased bone turnover that occurs normally during adolescence makes the use of bisphosphonates especially controversial for patients in that age group.

Although adequate calcium and vitamin D intake should be provided to patients with anorexia nervosa, such supplementation does not enhance BMD in anorexic patients. Some evidence suggests that a combination of twice daily IGF-I administration and estrogen-progesterone treatment may be effective in increasing BMD in anorexic women. Androgen-replacement studies have yielded conflicting results. Usually, patients with osteoporosis are advised to engage in weight-bearing exercise. But for anorexic patients, the potential benefits might be offset by the risk of fractures, delayed weight gain, and exercise-induced amenorrhea.