Osteoporosis and Thyroid Disease

Marshall Block, MD, FACP

Osteoporosis, a condition of excessive loss of bone and mass resulting in increased incidence of fractures of the spine, hip and wrist, occurs in increased frequency in patients who have longstanding hyperthyroidism. The hyperthyroidism can be the result of having an undiagnosed thyroid condition resulting in increased thyroid hormone production or due to excessive ingestion of thyroid hormone taken for the treatment of an underactive thyroid state (hypothyroidism).

Osteoporosis is a complex disorder, the exact cause of which is still not clear. There is probably more than one form of this condition resulting in the same end result, such as bone fractures. Normally, bone mass accumulates through our early years reaching a maximum amount at about age 30 or so. From then on there is a gradual loss of bone mass which is accentuated in women at menopause. When bone mass is measured using sophisticated measurement devices, there is a range of normal values found. When patients are below these limits, they are considered to have diminished bone density. Multiple factors can influence ultimate bone mass. For instance, if there is diminished calcium intake or a process limiting calcium absorption or both from the gastrointestinal tract early in life, maximum bone mass may not be reached. This results in diminished bone density and increased risk for fracture later in life. On the other hand, normal bone mass may be reached. Excessive loss of bone mass may occur due to ongoing processes such as menopause, or the use of drugs which accelerate bone turnover (steroids used for the treatment of rheumatoid arthritis or asthma). Similarly, excessive quantities of thyroid hormone either due to overproduction from the thyroid gland or due to excessive intake in the treatment of hypothyroidism can also accelerate bone loss resulting in a decrease of bone density. This in turn may lead to increased fracture risk.

Although we hear often of bone fractures in children or young adults, in older patients fractures of the hip and spine lead to long term disability and can lead to increased mortality. Because hip fractures are slow to heal, patients require significant bed rest which in turn may lead to pulmonary problems or infections or both. It is these various complications of a hip fracture which are so disabling and lead to much morbidity.

In an attempt to avoid these problems, there has been an increased interest in finding the causes of osteoporosis so preventive measures can be undertaken early in life. Furthermore, we are just beginning to understand the factors that influence bone turnover in adulthood and measures to enhance bone
mass and bone density during normal adulthood is now being undertaken in a more systematic fashion for our entire population. For instance, a minimal level of calcium needs to be taken by all women throughout their lives. Exercise, especially weight bearing exercise, seems to be important in maintaining bone mass. Using alcohol only in moderation and the avoidance of nicotine seems also to be beneficial in diminishing bone loss. The early use of estrogen replacement therapy in patients who are entering the menopausal period of their lives has also been recommended in those patients who have diminished bone density. Likewise, the use of medications which enhance bone loss need to be closely monitored and diminished as rapidly as possible depending upon the underlying condition for which they are being utilized. In regards to thyroid disease, we now have extremely sensitive measures to monitor thyroid replacement therapy. These measures include the usual levels of peripheral thyroid hormone products, thyroxine (T4) and triiodothyronine (T3) levels. Also, very sensitive assays now exist for thyroid stimulating hormone (TSH) which allow us to carefully regulate replacement therapy to make certain that the hypothalamus and the pituitary gland, parts of the brain which control thyroid function, are fine tuned in concert with peripheral thyroid hormone levels. With careful attention to these measurements, thyroid hormone replacement therapy is safe and will allow bone density to be preserved.

With the above thoughts in mind, it is therefore essential that all patients who are being treated with thyroid medication to either control an overactive thyroid gland or to treat an underactive thyroid condition, have their physicians check their peripheral hormone levels on a regular basis. Appropriate replacement therapy is now being produced which allows us to titrate doses as needed to keep thyroid hormone levels in a range which will not adversely impact bone mass. It is also important that patients take an active role in their own health maintenance. With this total care approach – adequate nutritional calcium intake, regular weight bearing exercise (walking), limited alcohol consumption, smoking cessation, estrogen replacement therapy, and regular full range hormone level evaluation – thyroid hormone levels can be kept in a range that will enhance bone turnover and thus help to prevent the development of osteoporosis.

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