Correspondence

Vitamin C and Osteoporosis: Is there a connection?

As I have become older and more personally familiar with the chronic conditions that come with age, I have become more interested in their causes since the standard medical treatment for them is usually only symptomatic, expensive, and doesn't address the root cause. Collagen is the most abundant protein in our bodies making up 25% of the total protein and is the major component of bone, teeth, skin, cartilage, and tendon. It is also the major component of the elastin in lung tissue and is responsible for holding cells together in all tissues. vitamin C is absolutely necessary for collagen production and maintenance. Vitamin C also has a big responsibility in the immune system and concentrates in the adrenal glands to be released in response to stress. It is incorrectly labelled a vitamin and in most animals, (except for man and a few others), is made in the liver and/or kidneys at a rate of about 50 mg per kg of body weight per day. In man the gene for L-gulonolactone oxidase is present but for some reason not expressed and results in the inability to produce vitamin C from glucose. In nature animals make grams of vitamin C daily and don't suffer the chronic diseases of man.

I am writing to propose a connection between vitamin C and osteoporosis. In searching the literature on vitamin C I cannot find any sources that emphasize its use as supportive or preventative therapy for osteoporosis. I am going to review the physiology of bone formation to show how vitamin C deficiency may be the overlooked primary cause of this disease.

Bone is a living tissue that is in constant turnover. It is composed of an organic matrix; osteoid, impregnated with the mineral salts, calcium and phosphorus. During life old bone is continuously being reabsorbed by osteoclasts while new replacement bone is being laid down by osteoblasts. Normally this process is in equilibrium so the density and amount of bone remains constant. In order for osteoblasts to make osteoid which is 90% collagen they must have adequate vitamin C. Calcium and phosphorus are used to mineralize the osteoid but are not necessary for its production. Of its dry weight one third of bone is osteoid and two thirds is mineral salts.

Osteoporosis is a loss of bone mass or density in a given volume. The remaining bone is normal, there is just less of it per unit volume. It could be compared to what termites do to wood. The wood remaining is normal, just less of it. In the growing skeleton, when osteoid production is halted we call it scurvy and the treatment is vitamin C. Radiology texts point out that the bony changes in scurvy are due to the same mechanism as osteoporosis and even have called scurvy, "infantile osteoporosis", to emphasize that a lack of osteoid is common to both conditions.[1] In senile osteoporosis we don't know whether the porous bones result from an increase in osteoclastic activity or a decrease in osteoblastic activity. Anything that tilts the balance towards the osteoclastic side of the equation will produce osteoporotic bone.

Osteomalacia, on the other hand, is a failure to mineralize the osteoid. It is due to lack of Calcium, Phosporus, or vitamin D. Osteomalacic bone is soft and is the cause of bowlegs in rickets. Rickets is nothing more than osteomalacia occurring in the growing skeleton. In adults, inward protrusion of the acetabuli, exaggerated disc impressions and flattening of the vertebral bodies occurs due to a softening of the bone. In osteoporosis the vertebra flatten due to fractures and bones don't bend, they break.

Osteopenia is a radiological term of recent origin that describes a decrease in density of bone on x-ray. Both osteomalacia and osteoporosis result in decreased bone density on x-ray and both have specific radiological findings but it is impossible to be sure whether one or the other or

both are the cause of the decreased bone density. Only a bone biopsy can tell for sure, so in order not to imply a specific disease the term osteopenia was coined.

In my opinion supplemental doses of vitamin C in the quantities produced by animals in nature should be part of the foundation of any nutritional program. This is not only true in osteoporosis but also in all of the connective tissue related diseases–emphysema, diverticulosis, hernias, arthritis, thin skin, braising, heart disease, immune deficiencies, periodontal disease, and probably many others. The fact that smoking depletes vitamin C levels, and is associated with an increased incidence of all of the above diseases, is further evidence for my opinion.

I hope this letter encourages someone with the proper resources to conduct a study that correlates bone density to vitamin C intake. I predict that benefits will be minimal until the oral supplementation is sufficient to produce what would be normal physiological levels were we able to activate our gene for L-gulonolactone oxidase. This level should be attainable with three or more grams per day.

References

1. Paul; Juhl: Essentials of Radiologic Imaging. Philadelphia. J. B. Lippincott Co. 1987; 212.

–W. M. Wassell, M.D.
New Orleans & Charity hospital &
Confederate Memorial Hospital,
Shreveport, LA.