Women's Health Update

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Essential Fatty Acids and Osteoporosis

Introduction and general observations

Osteoporosis is a growing concern with post-menopausal women due to its prevalence in 75 year olds and older and the disability it can cause. Although essential fatty acids (EFAs) have not been talked about much in relationship to this disease, there is a growing body of evidence and research to warrant our attention. Most of the research and focus on osteoporosis has been around the loss of calcium from bone before and during osteoporosis, reduced bone strength and increased risk of fractures. What has received less attention is that osteoporosis may be a marker for other serious potential health problems, apart from fractures. Not only must we consider demineralization of bone, but ectopic calcification and the possible connection between the balance between ectopic calcium deposits, particularly the arteries and the kidneys, and bone calcification. Specifically, low bone density may be related to other vascular problems and essential fatty acids and their regulation of calcium metabolism may be a key player in influencing the sites at which calcification occurs. The role of essential fatty acids has largely been ignored in relation to osteoporosis despite animal and human studies that have indicated that EFAs enhance calcium absorption, enhancing the effects of vitamin D, reduce urinary calcium excretion, increase bone calcium, reducing ectopic calcification elsewhere and increase bone protein synthesis and bone strength.

The first published paper that clearly showed the relevance of EFAs on calcium showed that in EFA-deficient animals, the kidneys became highly calcified, apparently because of a shift of calcium from the bones.[1] Other early studies, although about 20 years later, demonstrated that EFA deficiency in animals was associated with loss of normal collagen synthesis and of normal connective tissue in bone, loss of normal cartilage, demineralization of bone and bone weakness.[2-4]

After this early body of research, it had been sufficiently established that EFA deficiency led to servere osteoporosis in animals and that the osteoporosis was associated with significant ectopic calcification. Not until the 1990s did new observations lead to renewed interest in EFAs and calcium. These series of observations included that prostaglandin (PG) formation could stimulate bone growth, that renal calcium stones were rare among the Inuit (Eskimos) of the Arctic (seemingly due to their high intake of EFAS from fish oils), and that EFA metabolism might form a common etiologic basis for the associations between osteoporosis and coronary artery disease, peripheral vascular disease and stroke.

EFA Biochemistry

It's important to understand the basics of EFA biochemistry. There are two families of EFAs, the n-6 series and the n-3 series. Linoleic acid(LA) is the parent compound of the n-6 series and alpha-linolenic acid (ALA) is the parent compound of the n-3 series. Each of these is metabolized by a series of enzymatic reactions in which their metabolites play key roles within the body. The most important metabolites are probably dihomo-gamma-linoleic acid (DGLA) and arachidonic acid (AA) of the n-6 series and eicosapentaenoic acid (EPA) and docosahexaenoic acid (DHA) of the n-3 series.

The EFAs are normal and required constituents of every membrane within the body. They are required for the normal functioning of calcium release from storage and for the normal function of every membrane. EFAs are also part of most of the signaling systems within every cell.

Calcium Excretion

How calcium is excreted and how much is excreted is a major factor in the metabolism of bone, the development of kidney stones, as well as overall calcium balance in the body. Probably the major factor controlling calcium excretion is calcium intake, but we also know that prostaglandins are involved in calcium reabsorption and excretion. Significantly elevated levels of urinary prostaglandin E2 are positively correlated with urinary calcium excretion.[5-7] Calcium stones forming in the urine was also correlated with increased PGE2 and increased calcium excretion. Although we do not understand the precise role of EFAs and prostaglandins in hypercalciuria, the balance of research demonstrates that excessive production of PGE2 from arachidonic acid is a factor.

If we could reduce urinary calcium excretion, this could not only have a bone preserving effect but also reduce the formation of stones. Research in the early 1990s explored enhancing dietary EPA with fish oils to protect against stone disease. What was found was that the fish oils were able to reduce urinary calcium excretion. This work was preliminary and fundamental to understanding how EFAs and prostaglandin metabolism could affect calcium excretion and reabsorption. Although the work was largely in an effort to prevent kidney disease and kidney failure, it was key in supporting the idea that EFAs affect calcium metabolism and could be used to improve bone health and bone density.

Calcium Absorption

The influence of dietary fats on mineral absorption is complex and only understood in part. Several key observations have been made although many of the factors which influence absorption are still unknown. For example, increasing linoleic acid in the diet significantly reduces calcium in the stool, indicating that omega-6 EFAs stimulate calcium absorption.[8] Calcium absorption will also significantly increase when the diet is supplemented with either fish oil, evening primrose oil, a mixture of both or sunflower oil, daily.[9]

Animal studies have revealed many mechanisms related to EFAs and calcium absorption. Probably the largest body of work

established that there is a significant relationship between EFAs, the actions of vitamin D, the transport of calcium across the membrane and an increase in membrane fluidity followed by an increase in calcium absorption.

Bone

Bone density is a continuous process of bone building and bone loss. In our younger years, the bone building cells outpace the bone-loss cells. In our older years, the bone-loss cells exceed the bone building. If we have too little bone building in the early years, and an excessive amount of bone loss in the later years, low bone density and/or osteoporosis result.

Deficiencies of EFAs modify bone fatty acid levels and have profound effects on the degree of mineralization of the bone. Animals fed EFA deficient diets, develop osteoporosis. Evidence is also building that prostaglandins have an influence on bone metabolism. Prudent use of EFAs may reduce the degradation of bone matrix collagen, while also increasing bone mineral content. Using different ratios of evening primrose oil (high in gamma linolenic acid), fish oil (rich in EPA and DHA), sunflower oil and flax seed oil, animal studies suggest that supplementation with high evening primrose oil and fish oil is more effective in inhibiting bone loss than linoleic and alpha-linolenic acids.[10]

Osteoporosis and Cardiovascular Disease

The relationship between osteoporosis and cardiovascular disease has several correlations. One is that those individuals with osteoporosis and a subsequent hip fracture, have an increased risk of mortality due to strokes. There is also a problem in that calcium is not simply lost from the bone in osteoporosis, but that some of the calcium lost from bone is deposited in the arteries and kidneys. Individuals with osteoporosis frequently have ectopic calcification in other tissues, especially the discs between the vertebrae, the arteries and the kidneys. The calcification process in atherosclerosis is very similar to what occurs in bone. It may be that metabolic issues that regulate calcification are common to both diseases. Loss of bone calcium with concomitant calcification in the kidneys was observed as far back as 1931 in the study by Borland and Jackson where an induced EFA deficiency elicited both problems.[1] More recently, supplementation with EPA and GLA prevented ectopic calcification. This is better understood by looking at the role of EFAs in membrane health, calcium absorption, calcium excretion and bone mineralization. It is also worth speculating that since deficits of long chain EFAs are important in cardiovascular disease, the associations between osteoporosis and heart disease may be dependent on a commonality of impaired EFA metabolism and poor sources of dietary fat.

Summary

Osteoporosis affects 25 million people in the US and causes 1.5 million fractures annually. EFAs are often overlooked in the equation of what causes osteoporosis and what determines increased risk for osteoporosis and fractures. Laboratory, animal and human research have shown increased calcium absorption from the gut, reduced urinary excretion of calcium, increased calcium that is deposited in the bone and improved the strength of bone. Patients with osteoporosis who are given fish oil show an increase in calcium levels. In animals, EFA deficiency leads to the development of severe osteoporosis along with increased calcifications in the kidney and the walls of the arteries. This is similar to what we see in elderly patients with osteoporosis, i.e. loss of bone calcium and increased calcifications in soft tissues, particularly the arteries and the kidneys. These calcifications outside the bone may be more dangerous than the osteoporosis itself, since the great majority of osteoporosis related deaths is due to a vascular problem such as a blood clot formation.

Supplementing with oils high in gamma linolenic acid such as evening primrose oil, black currant oil and borage oil, and fish oils, rich in DHA and EPA may improve the absorption of calcium, enhance the calcium content in the bone, and improve the blood levels of calcium.

While preventive measures of a well balanced diet, avoiding smoking and excess alcohol, regular weight bearing exercise, and proper mineral intake are hallmarks in the prevention of osteoporosis, the information and data on essential fatty acids should motivate us to not only improve sources of dietary fat, it also suggests that EFA supplements are a viable method of decreasing the risk of osteoporosis.

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