Primary osteoporosis revisited

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Osteoporosis is a prevalent problem in industrialized society. It is suggested that attempts to limit the development of this condition should constitute a routine health promotion and disease prevention measure in chiropractic clinical care. This paper reviews contemporary trends in the minimization and control of osteopenia as a means of preventing clinically detectable osteoporosis. Modes of intervention accessible to chiropractic clinicians are emphasised.

KEY WORDS: osteoporosis, chiropractic, calcium, nutritional, supplementation, manipulation

L'ostéoporose est un problème fréquent dans les sociétés industrialisées. Nous suggérons que des efforts visant à enrayer le développement de cette condition soient inclus dans la pratique chiropractique courante comme mesures de santé et de prévention. Le document présent est un compte rendu des orientations actuelles dans le domaine de la minimisation et du contrôle de l'ostéopénie comme moyen de prévenir cliniquement l'apparition d'une ostéoporose discernable. Les modes d'intervention accessibles aux chiropraticiens sont soulignés.

MOTS CLÉS: ostéoporose, chiropractie, calcium, supplémentation alimentaire, manipulation.

Introduction

Osteoporosis is emerging as a major public health problem in western society – in the United States of America the cost of osteoporosis is of the order of $3.8 billion annually.[1] In Australia the hospitalization costs arising out of complications attributable to osteoporosis cost about $60 million per year.[2] This condition, characterized by a progressive loss in total bone mass (osteopenia), constitutes a particular concern in chiropractic clinical practice. Chiropractors are registered health professionals working at the interface between the consumer and health care system. Their unique contribution to health care is in the sphere of the musculo-skeletal system, notably the spine. Their orientation towards health care includes a commitment to disease prevention and health promotion.[3] Osteoporosis is of concern to the chiropractic profession, not only in so far as it may constitute a contraindication to spinal manual therapy, but also because it is a prevalent condition of the skeleton, the focus of chiropractic clinical expertise.

Clinical presentation

Primary osteoporosis is a covert condition until complications precipitate clinical symptoms. Peak cortical bone mass is usually achieved at about 35 years of age; trabecular bone mass peaks a little earlier. Peak bone mass is an individual variant, although, as a generalization, it has been noted that bone mass is some 30% higher in males than females, and 10% higher in blacks than whites. Osteoporosis predisposes the individual to fractures. Regardless of cause, the likelihood of osteoporotic complications is related to bone density; the greater the peak bone mass, the greater the resistance to osteoporotic fractures, that is the larger the bone mass safety zone before fracture thresholds are reached. Once bone mass has peaked there is a gradual reduction in total bone mass. In females there is a marked acceleration of this incipient process for three to seven years following menopause. This phenomenon has resulted in classification of primary osteoporosis into two categories:[4]

Type I or post-menopausal osteoporosis affects females between the ages of 55 and 75 years. This condition is characterized primarily by trabecular bone loss. The vertebrae most affected appear to be in the lower thoracic and upper lumbar area (T-8 to L-3). Clinical identification of asymptomatic spinal osteoporosis is possible on standard spinal radiographs – after the patient has lost 20-30% of vertebral bone mass! More severe cases may be identified by the presence of vertebral wedging and microfractures on routine preadjustment X-rays. Other suggestive findings in asymptomatic patients include loss of body height (up to 10 cm may be lost in 10 years) and the development of an increased thoracic kyphosis. Symptomatic patients present with acute local back pain. This pain is related to a spinal osteoporotic crush fracture which may be precipitated by such routine activities as getting out of bed, rising from a chair, lifting or bending. Neurological sequelae are infrequent. Type I osteoporosis is believed to respond to oestrogen therapy. Hormone replacement therapy is not however, sufficient therapy in the prevention or management of osteoporosis; it is best administered either with calcium supplementation alone or in combination with calcium, vitamin D and/or fluoride. By increasing the quantity of calcium delivered to the intestinal mucosa and improving absorption by stimulating calcium binding hormone production with vitamin D, the impaired calcium absoption of perimenopausal females can, at least partially, be overcome.[5] Calcium and fluoride are incorporated into bone and participate in mineral repair processes. These minerals and vitamin D are also used in the management of senile osteoporosis.

Type II or senile osteoporosis is a condition which affects both sexes between the ages of 70 and 85 years. This condition is characterized by cortical and trabecular bone loss. Fractures particularly involve the hip and wrist; vertebrae may also be involved.

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In the clinical diagnosis of osteoporosis, laboratory investigations tend to be unhelpful – plasma calcium and phosphate levels are within normal limits; as is serum alkaline phosphatase. Urinary calcium is unpredictable being either normal or elevated. Other means such as assessment of bone mass and strength are needed. Older techniques, radiological assessment of vertebral body height or cortical thickness of long bones, have been superseded by photon absorptiometry, computerized tomography or neutron activation analysis (measures total body calcium).[6] Early clinical recognition of osteoporosis, while no longer constituting a diagnostic dilemma, now constitutes a financial problem. Modern techniques, while facilitating earlier recognition of osteoporosis, still do not provide therapeutic answers. These must be sought by considering the pathogenesis of primary osteoporosis.

Pathogenesis:

The pathogenesis of osteoporosis remains controversial. While it is generally accepted that osteoporosis is manifested by a reduction in bone density and that overall bone density gradually decreases with age, the reason(s) for a reduction in bone density remain obscure. Certainly bone is a living tissue undergoing constant remodelling and the individual who attains a low peak bone mass is more at risk of osteoporosis than one who achieves a larger bone mass. Whether osteoporosis results from accelerated resorption of this bone mass, reduced ongoing bone deposition or a disparity between the two processes, remains uncertain.

If post-menopausal osteoporosis is indeed attributable to an increase in bone resorption, the mechanism remains unclear.[7] It has been suggested that bone loss may be attributable to elimination of the antagonistic effect that oestrogen has on parathormone and therefore bone resorption, alternatively enhanced metabolism of vitamin D has also been postulated.[8] The oestrogen hypothesis is somewhat supported by the clinical observation that obesity protects against osteoporosis – obesity is known to be associated with increased levels of circulating oestrogen. On the other hand, weight bearing is recognised to enhance maintenance of, and may increase, bone mass. Calcium dietary deficiency has been implicated in osteopenia. It has been documented that females in the United States consistently ingest less calcium than the RDA and it is furthermore suspected that RDA levels of calcium for females are inadequate.[9] Despite this correlation certain nutritional experts are hesitant to identify human osteoporosis as a consequence of calcium deficiency.[10] While adequate calcium absorption is deemed highly desirable, calcium deficiency alone is probably too simplistic an explanation for the pathogenesis of osteoporosis. This however, does not justify failure to attempt to reverse a patient's negative calcium balance by withholding calcium supplementation at currently recommended preventative and therapeutic doses.[11] Only persons with a history of urinary lithiasis are presumed to be at risk when placed on calcium supplementation. An alternative approach to intervention in the pathogenesis of osteoporosis is stimulation of bone formation. Androgens and fluoride have, with reservation, both been shown to have clinically detectable effects. Fluoride stimulates osteoblastic activity with resultant production of osteoid tissue; adequate mineralization of this osteoid tissue requires complementary calcium supplementation.[12] In addition to stimulating bone formation the fluoride ion is incorporated into hydroxyapatite's crystal lattice, thereby enhancing the bones resistance to resorption. Fluoride intervention requires long-term therapy and it is not without side effects. Fluoride is however the only substance shown to increase trabecular bone volume and mineral content.[13] The pathogenesis of osteoporosis and its complications have been postulated to involve mechanical microdamage.[14] Exercise may play a role both in minimizing microtrauma and in maximizing peak bone mass by modifying remodelling architecture.

Risk factor analysis

Diagnosis of osteoporosis is late; prevention of any reduction in bone mass or microtrauma prior to clinically overt skeletal disease being detectable is desirable. Clinical recognition of osteoporosis implies identification of a patient at risk of fractures. Therapeutic intervention at this stage requires reversal of bone loss; mere arresting of further bone depletion fails to address the patient's already significant risk of fractures. Realistic intervention therefore depends on disease prevention and health promotion. Clinical intervention may thus be initiated by risk factor analysis.

Non-modifiable risk factors which alert the clinician to the individual at increased risk of primary osteoporosis include consideration of:

- sex: females are at more risk than males.
- race: whites are at more risk than blacks.
- age: elderly persons are at increased risk of osteoporosis.
- menopause: bone mass is more rapidly depleted when women cease to menstruate.[15] A woman who has undergone an early menopause is likely to reach the threshold for bone fracture at an earlier age than she would had she experienced a later menopause. This is true regardless of whether the menopause was natural or surgically induced (following oophorectomy). It is interesting to note that amenorrhoea induced by excess exercise in athletes is also associated with a reduction in bone mass.
- physique: thin people are at increased risk; obesity appears to carry some protective effect.
- genetic predisposition: persons with a family history of osteoporotic fractures are at risk. Usually certain of the above variables are present.

Modifiable risk factors associated with attainment of a small peak bone mass and or impaired bone mass maintenance include:

- sedentary life style, immobilization and/or prolonged bed rest. Weight bearing physical activity reduces bone loss and increases bone mass.

- inadequate calcium absorption. This may be attributable to dietary deficiency or absorption defects.
- steroid medication.

Consideration of these risk factors prior to obtaining radiological evidence of vertebral osteopenia in chiropractic practice is desirable. The predictable universal phenomenon of age-related bone loss cannot be eliminated. An attempt can however be made to prevent any single individual from reaching the fracture threshold. In women, the average rate of trabecular and cortical bone loss after about 35 years of age is 1% of peak adult bone mass per annum; in males the cortical loss is 0.3% of peak bone mass per annum. These losses represent a negative calcium balance of 0.25mmol/day of calcium in men and 0.5mmol/day in women, with peak losses following menopause of 1-3mmol/day.[16] Chiropractic intervention can be aimed at:

- minimizing future bone loss
- maximizing peak bone mass (in younger patients)

Prevention of primary osteoporosis in the chiropractic clinic

Women who present with clinically overt osteoporosis should be referred to medical practitioners capable of prescribing oestrogen therapy. Hormonal replacement therapy is the only undisputed intervention which prevents accelerated postmenopausal bone loss. Oestrogens are therefore recommended for all postmenopausal women who suffer from crush fractures and for women who enter menopause with a strong family history of osteoporosis and a low bone mass.[17] Therapy should be continued for 10 to 15 years. When hormonal replacement is discontinued accelerated bone loss follows. Despite the increased risk of hypertension, cholelithiasis and endometrial carcinoma in woman on long term oestrogen replacement therapy, the beneficial skeletal effects are believed, with adequate monitoring, to outweigh any consequences.[18] Patients who present with osteoporosis risk factors in the absence of overt clinical symptons or signs are suitable for chiropractically accessible intervention measures. These include:

- Calcium supplementation with or without dietary modification.
- Weight bearing exercise
- Fluoride supplementation

Calcium supplementation:

The success of calcium supplementation in osteoporosis is controversial.[19] Woolf and Dixon quote various workers who have demonstrated that intakes of calcium greater than 1500mg/day reduce cortical bone loss. Others have shown that calcium supplements almost halve the incidence of post-menopausal vertebral crush fractures. In contrast, other trials have failed to demonstrate any significant reduction in bone loss in patients on calcium supplements. Certainly there is little support for the notion that calcium supplementation may cause a gain in bone mass.

As 99% of the body's calcium is located in the skeleton any prolonged discrepancy between calcium intake and calcium excretion must be reflected in the skeleton. Calcium intake is determined by dietary calcium and intestinal absorption. In general, about 30% of ingested calcium is absorbed. Calcium, at low intake levels, is absorbed by an active transport mechanism; at higher concentrations it diffuses across the intestinal wall.[20] Vitamin D3 (1,25 dihydroxycholecalciferol) the active form of vitamin D, facilitates calcium absorption from the intestine; it also facilitates calcium reabsorption at the renal tubule and enhances calcium resorption from bone. Vitamin D3 facilitates active intestinal absorption of calcium by stimulating the synthesis of a specific calcium binding protein. Passive calcium absorption is dependent on the concentration of soluble or available calcium in the intestine. The major sources of dietary calcium are dairy products and to a lesser extent vegetables, fruit and grain products. The protein and phosphorus in dairy products are believed to have no effect on calcium absorption while lactose is thought to enhance the diffusional component of calcium transport. Phytate, oxalate and fiber (particularly uronic acids), in contrast, have been shown to impair calcium absorption. This is of particular importance in view of the current trend towards high fiber foods, whole grains and vegetable rather than animal food sources. Oxalate is found in significant concentrations in green leafy vegetables eg. spinach and tea; phytates are found in legumes and unleavened bread. When complexed with phytate or oxalate, calcium is unavailable for absorption eg. the calcium in spinach. Calcium absorption decreases with age; individuals should increase their calcium intake as they get older.

Calcium balance is the result of both calcium absorption and calcium excretion. Calcium is lost in urine, sweat and faeces. Faecal calcium is the sum of unabsorbed dietary calcium and unresorbed calcium secreted in intestinal juices. Plasma and urinary calcium rise in response to a calcium load; a change of 0.02mg/100ml of plasma calcium may double or halve the urinary calcium. There is however an obligatory urinary calcium excretion; when absorbed calcium is less than about 150mg/day negative calcium balance results.[21] Dietary protein has been shown to influence urinary calcium excretion. At any given calcium intake, the amount of calcium excreted in the urine increases as protein ingestion increases. It has been postulated that the acid end products of protein catabolism are responsible for this phenomenon. Ingestion of small amounts of sodium bicarbonate have been shown to have some effect on reducing protein induced calciuria.[22] Protein induced calciuria should not be interpreted as a contraindication to encouraging patients to drink milk; in fact milk and dairy products are viewed as desirable sources of protein.[23] For maximum benefit, persons supplementing their calcium intake with milk and milk products should limit their protein intake from other sources.

Hormones secreted in response to calcium balance are also modulated by serum phosphate levels. Vitamin D3 enhances phosphate resorption from bone and absorption from the intestine; differential calcium/phosphate reabsorption and ex-

cretion at the kidney tubule are achieved in response to parathormone and calcitonin. Phosphate, furthermore, is linked with calcium in the hydroxyapatite lattice of bone. Optimal structural integrity of the skeleton is believed to be achieved when the dietary intake ratio of calcium to phosphorus is 1:1. Modern dietary excesses may upset this ratio. These include phosphate loading by over indulgence in processed foods using phosphate as a buffer eg. carbonated drinks. This modern dietary trend is postulated to cause nutritional hyperparathyroidism to manifest clinically as alveolar bone resorption and osteoporosis.[24] High serum phosphate and relatively low serum calcium levels are associated with raised serum parathyroid hormone levels, increased bone resorption and enhanced urinary phosphate excretion.

In addition to absorptive and excretory variables, which may influence calcium balance, calcium requirement may be modified by physiological states. Increased calcium is required during the last trimester of pregnancy, lactation and puberty. Recommendations for calcium intake take cognisance of these increased requirements. Calcium recommendations do, however, tend to fall short of current thinking as it relates to prevention of osteoporosis.[25] It has been suggested that men and premenopausal females should have a daily calcium intake of at least 1000mg; this should be increased to 1500mg postmenopausally.[26] In order to achieve these levels nutritional supplementation may be indicated.

In recommending calcium supplements both the time of administration and the type of supplement are worthy of consideration. Parathyroid hormone secretion appears to peak at night. This noctural rise of parathormone may be suppressed by taking one gram of calcium prior to going to bed.[27] The form in which this supplement may be taken could be as milk or as an over-the-counter preparation. Tablet, chewable, syrup and antacid forms of calcium supplementation are available. Care should be taken to note the concentration of elemental calcium as this is somewhat less than the total concentration of the tablet which reflects the concentration of the complex. Calcium complexed with gluconate, carbonate or lactate is cheaper but less well absorbed than calcium aspartate or orotate. Microcrystalline hydroxyapatite crystals derived from whole bone extract may have particular promise in the prevention and treatment of osteoporosis in view of their physiological mineral complement.[28] Patients should not attempt to derive their total daily calcium requirement from nutritional supplements. In general, an individual may ingest at least 400mg/day of calcium in his/her diet. One cup of milk or yogurt contains 300mg of calcium, one slice of cheese contributes 200mg and one cup of ice cream 180mg. Half a cup of broccoli or baked beans will contribute a further 65mg, while 3 oz of sardines (bones included) contribute 375mg.[29] In view of the absorption and excretory control of calcium it is unlikely that calcium intake up to 2000mg per day will cause hypercalcaemia. Patients with a predisposition to urinary lithiasis may however be at risk. Persons with a history of renal stones should increase their dietary calcium levels with caution and ensure adequate magnesium ingestion (300mg/day of magnesium oxide) with or without pyridoxine (10mg/day) supplementation.[30] Adequate fluid ingestion is also imperative.

Weight bearing exercise:

Exercise in the presence of adequate skeletal nutrition can increase bone mass.[31] The type of exercise for preserving or increasing bone mass requires active weight bearing or vigorous muscle pulling on bone. This is consistent with the concept that weightbearing causes laying down of bone with skeletal strength being directly proportional to the load.[32] This is congruent with the findings that obese woman are less at risk of osteoporosis, and that resumption of normal physical activities may gradually restore bone lost during periods of immobilization or weightlessness (astronauts).[33] A controlled trial has shown that exercise can inhibit or reverse involutional bone loss from the vertebrae of normal women.[34]

The mechanism whereby exercise modifies bone mass remains obscure. Muscular activity may have a direct neural influence on bone; blood perfusion may be altered during exercise and/or the mechanical stress of weight bearing may create electric fields with benefit being derived from endogenous piezo-electric effects.[35] Certainly muscle mass correlates with bone mass, and habitual exercise and/or increased physical activity are related to increased bone mass. It is therefore desirable that everybody refrain from prolonged bed rest and indulge in weight bearing exercise eg. walking or jogging. A basal requirement seems to be of the order of 30-60 minutes, three times a week. Quiet walking for 4 hours despite immobilization for 20 hours of a day may, according to Korcok's interpretation of Whedon's work, prevent a negative calcium balance.[31]

In postmenopausal females who have evidence of spinal osteoporosis, caution must be demonstrated in providing exercise advice. Therapeutic exercise programs should suggest extension or isometric back and abdominal strengthening exercises and avoid flexion postures. Exercises that exert flexion forces on osteoporotic vertebrae have been shown to increase the incidence of vertebral fractures.[36] Any patient with evidence of osteoporosis should avoid twisting, explosive or staccato movements.

Fluoride supplementation:

Adverse reactions to fluoride therapy at therapeutic doses include joint pain, swelling or painful plantar fascial syndrome, nausea and vomiting, peptic ulceration and microcytic anaemia.[13] Despite the risk of patients developing these reactions and having to discontinue their sodium fluoride, this drug remains a useful adjunct to therapy in patients with clinically overt osteoporosis. Fluoride, in the presence of calcium supplementation, can increase bone mass in postmenopausal osteoporosis and prevent spinal crush or wedge fracture.[37] Effective doses are 1mg/kg/day sodium fluoride accompanied

by a daily calcium supplement of 1500-1800mg. Best results are obtained when the patient is also placed on hormonal replacement.[38] At least one year of fluoride treatment may be required before a substantial increase in axial bone mass is detected by neutron activation analysis. In some patients no increase in bone mass may be detected, and it has been postulated that a subgroup of osteoporotic patients with an intrinsic abnormality of osteoblast function are nonresponsive to fluoride intervention.

Therapeutic recommendations:

Chiropractic clinics are attended by individuals who will benefit from measures aimed at reducing the risk of osteoporosis. In the 1983 Australian Health Survey 79,900 interviewees in the 25-44 year age group, and 49,000 in the 45-64 year age group had visited a chiropractor during the two weeks prior to the interview. Of these 42,100 were women between the ages of 25 and 44, and 28,300 were women between 45 and 64 years of age.[39] Men, premenopausal women and those postmenopausal women without clinical evidence of osteoporosis who present with a large frame with good muscle mass and no history of steroid ingestion, should be assessed for the following:

1 Calcium intake: calcium determination resulting from the patient's food diary should be supplemented by calcium tablets so that the daily calcium intake equals 1000mg in males and premenopausal females; and in postmenopausal women exceeds 1500mg, but not 2000mg. Adequate calcium intake is recommended for all persons and not only for postmenopausal women.[40] Calcium overdose may manifest as anorexia, nausea, constipation and thirst. Check for a history of renal stones prior to implementing calcium supplementation.
2 Dietary patterns: excess protein should be reduced. Increased dietary calcium derived from milk and dairy products should be counterbalanced by a reduction in other protein sources. Alkali supplementation with high protein meals may be considered. Excess phosphate intake eg. consumption of carbonated drinks and certain canned foods should be controlled.
3 An exercise routine: the individual should spend at least 30 minutes three times a week involved in weight bearing exercise. Walking, running and bicycling are good exercise modes.
4 Drug usage: limitation of alcohol and tobacco use may disputedly be helpful. Aluminium ingestion in any form, including as an antacid, enhances calciuria and is therefore contraindicated.

Women who undergo a premature menopause and postmenopausal women with clinical evidence of osteoporosis should be referred for hormonal replacement therapy, calcium supplementation and exercise prescription with or without fluoride intervention.

Conclusions

Chiropractors have a contribution to make in the minimization of osteoporosis and its clinical complications. Their major contribution is in the early recognition of risk factors. Correction of inadequate calcium consumption coupled with appropriate weight bearing exercise can facilitate maximization and maintenance of bone mass. Once osteopenia has potentiated the evolution of clinically apparent osteoporosis, additional therapy including vitamin D, fluoride and hormones such as oestrogens and androgens should be considered.

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