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Osteoporosis in the light of other medical conditions

In this issue of Osteoporosis Update, we are privileged to hear from experts on two specialized topics of great interest to health professionals involved in the care of individuals with osteoporosis. In the feature article, Dr. Ramsey Sabbagh, a Québec nephrologist and Adjunct Professor of Medicine at the McGill University Health Centre, informs us regarding the significant link between nephrolithiasis — a relatively common condition in the general population — and bone mineral density. This is an important topic, but not always well understood. Questions arise such as: Should patients with renal stones limit their calcium consumption? Are vitamin D supplements safe? What are the appropriate dosages of calcium and vitamin D? What therapies are effective in minimizing bone loss in people with this condition? In his well-written and clear presentation, Dr. Sabbagh provides some helpful diagnostic and management points to guide clinicians.

The case study by Dr. Cathy Craven, Clinician Scientist/Physiatrist in Toronto Rehab’s Spinal Cord Rehabilitation Program, deals with sublesional osteoporosis (SLOP), a disease unique to people who have sustained spinal cord injuries. Sublesional osteoporosis is characterized by excessive bone resorption, deterioration in lower extremity BMD and increased propensity for distal femur and proximal tibia fractures. There are currently no guidelines on treating SLOP subsequent to spinal cord injuries. Dr. Craven’s informative discussion of issues related to osteoporosis management in these patients will help to increase awareness of the debilitating effect of fractures on their functional ability and quality of life.

Also in this issue: George Ioannidis, a research methodologist from McMaster University, addresses the relationship between fractures and mortality and outlines steps to help improve outcomes in elderly patients; and Panagiota Klentrou, Professor and Chair in the Department of Physical Education & Kinesiology at Brock University and a member of the Osteoporosis Canada’s Scientific Advisory Council, looks at the timely question of bicycling — and other intense sports activities — and bone health. Finally, we have the pleasure of mentioning recent awards extended to some upcoming young researchers in the osteoporosis community.

We welcome your comments and questions. Please address correspondence to osteo@parkpub.com.
Issues in osteoporosis management related to spinal cord injury

A 35-year-old male sustained T4 paraplegia secondary to a motor vehicle accident two years ago, and is now wheelchair bound. Is osteoporosis screening indicated? Does he require treatment? Are there any special treatment considerations?

Spinal cord injury (SCI) results in diverse motor, sensory and autonomic impairments that are determined by the location and severity of the injury within the neural canal. “Tetraplegia” describes lesions of the cervical spinal cord, while “paraplegia” refers to thoracic cord lesions. A “complete injury” is defined as the absence of any motor or sensory function below the level of injury, including the sacral segments, and “incomplete injury” by preservation of motor or sensory function below the level of injury, including sacral sparing. Of an estimated 900–1000 Canadians who sustain a SCI each year, 80% are male, and 84% of injuries occur in people under age 34. The most common causes of SCI in Canada are motor vehicle accidents, falls, diving and sports accidents and other medical conditions. The trend of increased survival in the first 2 years post SCI and extended life expectancy has resulted in a shift in the emphasis of healthcare provision from assuring survival to minimizing secondary complications (including osteoporosis and fragility fracture) and enhancing quality of life.

Unique osteoporosis issues
Sublesional osteoporosis (SLOP) is a disease process unique to people with SCI. SLOP is characterized by excessive bone resorption, deterioration in lower extremity bone mineral density (BMD) and bone architecture, and an increased propensity for lower extremity fragility fracture. SLOP is distinct from osteoporosis due to aging in its rapid rate of onset, severity of BMD decline, skeletal distribution, bone micro-architecture involvement, etiology and associated regional fracture risk. Among patients with motor complete SCI, there is a 3% to 4% per month BMD decline at the hip and knee. Typically, BMD of the hips, distal femur and proximal tibia are 28%, 37%–43%, and 36%–50% below that of age-matched peers at 12–18 months post injury. There is disagreement whether this BMD decline continues or stabilizes at some point after injury.

Distal femur and proximal tibia fractures prevail; 25% to 46% of chronic SCI patients develop fragility fractures, frequently caused by torsional stresses on the legs during a transfer or compressive forces at the knee during a low-velocity fall from a wheelchair. A single fragility fracture often leads to a cascade of events that increase patients’ morbidity due to complications of fracture treatment and immobilization (e.g. heel or ankle ulcer from a cast or deep venous thrombosis) and decrease their functional abilities (i.e. immobilization devices or increased need for attendant care services during healing). Fragility fractures after SCI frequently result in delayed union, non-union or mal-union, and amputation in extreme cases.

Diagnosis and fracture risk assessment
Identification of patients with SLOP and high fracture risk entails BMD testing and a review of fracture risk factors. There are several established methods for measuring knee region BMD. Regardless which is chosen, assessment of knee region BMD is crucial as it is the best predictor of knee fracture risk after SCI. Risk factors for fragility fracture after SCI include: SCI before age 16, paraplegia vs tetraplegia, complete vs incomplete SCI, female vs male gender, duration of SCI > 10 years, BMI ≤ 25 kg/m², knee region BMD below the fracture threshold, an alcohol intake of > 5 servings per day, prior history of fracture and maternal history of fracture.

Lazo has shown that BMD T-scores are predictive of risk fracture in men with SCI, noting a 2.8 times relative increased risk of fracture for every one standard deviation decrease in femoral neck T-score. Conventional measures of spine and hip region BMD in patients with SCI are often inaccurate or difficult to interpret longitudinally due to the presence of hardware, laminctomy, posterior element degenerative changes at the spine and/or heterotopic ossification (abnormal formation of true bone within extraskeletal soft tissues), contracture or dislocation at the hip. Based on hip or knee BMD results (Table 1), clinicians may identify patients with SLOP according to their gender and age at the time of the scan.

Not all causes of low BMD after SCI are attributable to SLOP; routine serum and urine screening among others.
patients with SCI identifies other contributing factors 30% of the time: hyperthyroidism, vitamin D deficiency and secondary hyperparathyroidism, renal insufficiency; alcoholism, hypogonadism (men) and amenorrhea (women). 29

Treatment strategies

To date, no SLOP treatment trial has been adequately powered to address fracture reduction among patients with SCI. Most osteoporosis intervention trials have selected increases in lower extremity BMD as a proxy for fracture reduction. Treatments for SLOP after SCI include: rehabilitation interventions, oral bisphosphonates, calcium and/or vitamin D supplements and lifestyle modifications (weight-bearing exercise, smoking cessation, counselling on restriction of alcohol and caffeine intake). Prior to initiating therapy, an assessment of the patient’s diet is necessary to ensure sufficient — but not excessive — calcium and vitamin D intakes. Excess calcium (above 1500–1750 mg/day) may precipitate bladder or kidney stones, while too much vitamin D may lead to heterotopic ossification among SCI patients. At Toronto Rehab, we routinely recommend daily intakes of 1000 mg calcium and 1000 IU vitamin D, for patients with SCI. 29

Recent systematic reviews summarize drug and rehabilitation interventions for SLOP treatment. 30,31 Of the 17 studies reviewed (3 drug and 14 rehab), no intervention led to sustained increases in hip or knee region BMD in subjects with chronic SCI and SLOP. FES (Functional Electrical Stimulation) cycle ergometry or passive standing may be offered provided patients understand that these are lifetime prescriptions, as the therapeutic benefit abates with cessation of therapy.

Level I evidence supports alendronate for treatment of patients with motor complete paraplegia. Using a randomized open-label design, Zehnder et al evaluated the effectiveness of alendronate 10 mg daily and elemental calcium 500 mg daily vs elemental calcium 500 mg daily (alone) for 24 months. The study cohort consisted of 55 men with motor complete SCI. Injury duration ranged from 1 month to 29 years post SCI, with group means of 10 years post injury. The key findings included an 8.0% decline in tibia epiphysis BMD (the primary outcome) in the control group and relative maintenance of tibia epiphysis BMD (~2.0%) in the treatment group (p < 0.001). Patients with SLOP and motor complete SCI, injury may be treated with alendronate (70 mg weekly) and calcium (1000 mg daily in divided doses) and vitamin D supplements, to ensure serum values in the therapeutic range (75–150 nmol/L). 32

Although many oral and intravenous bisphosphonates are available on the market, only alendronate has been shown to maintain hip and knee region BMD after SCI, although fracture outcome studies have not been done. There are no clinical trials evaluating drug treatments of SLOP among patients with motor incomplete injuries. Recent p-QCT data describing longitudinal changes in lower extremity cortical and trabecular BMD over time suggest there is a therapeutic window 2 to 8 years post injury during which antiresorptive therapies are most likely to be effective. 33 Oral aminobisphosphonates should be used with caution in patients with spinal cord lesions at or above T6, as esophageal dysmotility is common after SCI and the risk of esophageal erosions is increased compared to individuals without SCI. 34 Alendronate may also elicit atrial fibrillation among patients with SCI and a propensity for autonomic dysfunction. The propensity for arrhythmia is specific to patients with SCI and lesions above T6.

Initiation of SLOP therapy necessitates repeat BMD testing to evaluate effectiveness. Follow-up testing is typically done every 1 to 2 years at the same facility, using the same acquisition and analysis protocols. An increase in BMD above the least significant change (LSC) suggests effective therapy, while a decrease of greater than the LSC prompts re-evaluation of the treatment protocol and of patient adherence.

Increased survival and life expectancy post SCI have shifted the emphasis to minimizing complications, including osteoporosis and fragility fracture.

Increasing awareness of bone risks

In the absence of guidelines for the diagnosis and treatment of SLOP in people suffering from spinal cord injury, clinicians should be aware of the important risk of fractures, especially of the lower extremities, which lead to significant complications and declines in functional ability. As in the case described above, men with paraplegia warrant BMD and fracture risk screening. The patient should undergo nutritional assessment to ensure a balanced diet, including appropriate amounts of calcium and vitamin D, and should be counselled on the role of rehab interventions (FES and passive standing) and lifestyle modifications to augment his bone mass. Pharmacologic therapy should be considered to maintain BMD of the hip and knee regions. Given recent worries about the spontaneous, atypical femoral fractures in non-SCI patients who have been on alendronate for some years, a maximum of 10 to 13 years of therapy is recommended. This recommendation is speculative in nature, as these patients are particularly susceptible to similar lower limb fractures. 35,36

References


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Bones and stones
The link between osteoporosis and nephrolithiasis
By Ramsey Sabbagh, MD, FRCPC

Nephrolithiasis (kidney stone disease) is relatively common in the general population, with a lifetime incidence of approximately 12% in men and 6% in women. Once affected by a kidney stone, an individual’s risk of having a recurrent episode can be higher than 50% over the next 10 years. Over 80% of all kidney stones are calcium (most commonly calcium oxalate)-based. There are several potential risk factors for kidney stone formation, including dietary/lifestyle considerations, metabolic abnormalities and genetic susceptibility, and approximately half of all calcium-containing stone formers are found to have hypercalcaturia. Clinically, hypercalcaturia is defined as a daily urine calcium excretion exceeding 6.25 mmol in women or 7.5 mmol in men (or > 0.1 mmol/kg/day in either women or men), in two consecutive 24-hour urine collections. If serum calcium is normal and secondary causes (Table 1) such as primary hyperparathyroidism have been ruled out, hypercalcaturia is defined as “idiopathic” or “primary.” Indeed, the majority of hypercalcaturic calcium stone formers have idiopathic hypercalcaturia (IH).

A classification scheme for hypercalcaturia developed over 30 years ago divided the condition into absorptive, resorptive and renal subtypes. Diagnosis was based on urine sample results following an overnight fast, a low calcium diet and an oral calcium load. However, categorizing patients using such a method is difficult in the clinical setting and does not alter potential therapeutic interventions. It is now thought that IH in most patients may involve a complex combination of absorptive, resorptive and renal mechanisms: increased intestinal calcium absorption, excessive calcium release from bone and abnormal renal calcium handling, respectively. This is supported by evidence of bone mineral loss among individuals classified as absorptive hypercalcaturic, even though such patients have an increase in intestinal calcium absorption.

Kidney stones and bone mineral loss
The relationship between renal stones, specifically hypercalcaturic nephrolithiasis, and bone mineral density (BMD) is well established. A large epidemiologic study evaluating factors associated with BMD in men showed that a history of renal stones was associated with lower BMD. In addition, population-based studies have shown an increased fracture risk among participants with a kidney stone history: This relationship was stronger among males nevertheless, hypercalcaturia plays an important role in osteoporosis among postmenopausal women, with a prevalence as high as 19% in those referred to a metabolic bone disease clinic. Indeed, menopause is associated with an increase in renal calcium excretion, but a large epidemiologic study showed a correlation only between surgical menopause and incident kidney stones.

Genetic and dietary factors
Several potential mechanisms linking BMD loss and IH have been proposed. Approximately half of all patients with IH have a family history of nephrolithiasis. Even though genetic factors may have a significant effect on both BMD and hypercalcaturia, a common genetic variant linking most cases of IH and BMD loss has yet to be discovered. Common genetic variations have been found, however, in small subsets of patients with renal stones and BMD loss. For example, Prié et al described cases of nephrolithiasis, bone demineralization and hypophosphatemia (low serum phosphorus) associated with a mutant type 2a sodium-phosphate cotransporter.

Dietary factors that may link hypercalcaturia with BMD loss have revolved around the balance of calcium, sodium and protein intake:

- In the past, patients with hypercalcaturic nephrolithiasis may have been advised to limit their calcium consumption, or done so independently, based on the notion that it might help minimize further stone formation. This restriction can lead to a negative calcium balance and has been associated with BMD loss. We now know that calcium restriction may in fact heighten the risk of new symptomatic renal stones in such patients, by allowing for increased intestinal oxalate absorption (and, as a result, greater urinary oxalate excretion). Therefore, a normal dietary calcium intake is essential in patients with IH and renal stones.

- While a high-sodium diet is not usually the sole cause of hypercalcaturia, it may aggravate this condition by inducing volume expansion, reducing sodium transport and, consequently, decreasing calcium reabsorption in the proximal tubule. Increasing dietary sodium by 100 mEq will raise urinary calcium excretion by approximately 0.6 mmol in a normal subject, but may increase it by over 1 mmol in someone with IH. In addition, Martini et al showed that a high salt intake was predictive of low BMD in calcium stone formers after multivariate adjustments, and reducing dietary sodium may have a positive effect on bone metabolism.

- A diet rich in animal protein is associated with hypercalcaturia and stone disease. Bone buffering of the acid...
load provided by animal protein causes calcium release from bone, and the acid load may directly inhibit calcium reabsorption in the renal tubule. Acid derived from animal protein may also reduce urinary excretion of citrate, an inhibitor of stone formation. While some research has supported a detrimental effect of an acid load on bone, studies have been conflicting.

Management

Dietary management

Patients with hypercalciuric nephrolithiasis should maintain a diet with a sodium intake of less than 100 mEq (2.3 g) per day and an animal protein intake of less than 1 g per kg of body weight per day.

What about calcium supplements?

A common clinical dilemma involves the use of calcium supplements in patients with osteoporosis who have a history of renal stones. Results from large epidemiologic studies have varied. In the Nurses’ Health Study I, supplementation was associated with a 1.2 relative risk of stone formation, while other studies did not report an increased risk. A reason for this discrepancy may be related to the timing of calcium supplement ingestion and whether or not the supplement is taken with a meal. When it is taken without food, no intestinal oxalate is available to bind with the calcium, leading to an increase in calcium absorption and urinary excretion, without reduction of daily oxalate absorption/excretion. It may be wise to advise such patients to obtain most of their daily calcium from food; if supplements are necessary (e.g. in the case of lactose-intolerance), patients should be reminded to take them with meals. Using a calcium citrate salt may provide the added benefit of citrate in stone formers.

The role of citrate

Citrate is a natural urinary inhibitor of calcium stone formation, and hypocitraturia may contribute to renal stone disease in some patients. Hypocitraturia may be idiopathic or secondary to conditions associated with a chronic metabolic acidosis, such as renal tubular acidosis or chronic diarrhea. In addition, a high animal protein diet, which generates a significant acid load, may also reduce urinary citrate. Considering the effect of acid buffering on bone mineral loss, some studies have investigated the effect of citrate supplementation (an alkali equivalent) on BMD in recurrent calcium stone formers, demonstrating a mild benefit in lumbar spine and distal radius BMD.

Is vitamin D supplementation safe?

Research is lacking on the isolated effect of vitamin D supplements in calcium stone formers. Studies have demonstrated that high doses of vitamin D are safe in non-stone formers, with no significant effect on urinary calcium. Although usual vitamin D dosing of 400–800 IU/day for osteoporosis patients is likely safe in stone formers, one must keep in mind that some people with IH have elevated 1.25[OH]2 vitamin D levels associated with a “hyper-sensitive” 1α-hydroxylase enzyme (the kidney enzyme responsible for converting the major circulating form of vitamin D, 25[OH]D3, to the active metabolite 1.25[OH]2D3). Augmenting 25[OH] vitamin D with supplementation in such patients may increase 1.25[OH]2 vitamin D, intestinal calcium absorption and urinary calcium excretion. Therefore, it is advisable to monitor the urinary calcium response in individuals who are calcium stone formers and who are taking vitamin D supplements for bone health.

Thiazide diuretics

If a patient with reduced BMD is discovered to have hypercalcuiuria, secondary causes should be ruled out and dietary parameters optimized. If the IH persists (i.e. daily urinary calcium excretion in two consecutive collections > 6.25 mmol in women or 7.5 mmol in men, or > 0.1 mmol/kg/day in women or men), the mainstay of therapy is a thiazide diuretic. Thiazides reduce urinary calcium excretion by

Table 1. Causes of secondary hypercalciuria

<table>
<thead>
<tr>
<th>Dietary factors</th>
<th>Other causes</th>
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<tbody>
<tr>
<td>• Excessive calcium intake</td>
<td>1. Increased intestinal absorption of calcium</td>
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<tr>
<td>• Excessive salt intake (&gt; 6 g/day)</td>
<td>• Overdose of vitamin D</td>
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<tr>
<td>• High animal protein intake</td>
<td>• Increased production of 1.25[OH]2D3, due to primary hyperparathyroidism, sarcoidosis and other granulomatous diseases (tuberculosis), lymphoma</td>
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<td></td>
<td>• Severe hypophosphatemia (mesenchymal tumours, Fanconi syndrome)</td>
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<td></td>
<td>2. Increased osteoclastic bone resorption</td>
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<td></td>
<td>• Bone metastases, myeloma</td>
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<td></td>
<td>• Primary hyperparathyroidism</td>
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<td>• Flare of Paget’s disease</td>
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<td></td>
<td>• Prolonged immobility (mainly in young subjects)</td>
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<tr>
<td></td>
<td>• Hyperthyroidism</td>
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<td></td>
<td>3. Decreased reabsorption of calcium by the renal tubule</td>
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<td></td>
<td>• Loop diuretics</td>
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<tr>
<td></td>
<td>• Medullary sponge kidney (dilatation of the terminal collecting tubules producing a brush-like papillary blush or a “bouquet of flowers” image on the intravenous urogram done to investigate nephrolithiasis)</td>
</tr>
<tr>
<td></td>
<td>• Glucocorticoid therapy, Cushing’s disease</td>
</tr>
<tr>
<td></td>
<td>• Renal tubule disorders responsible for hypercalcuiuria with nephrolithiasis</td>
</tr>
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as much as 50%,28 and calcium stone recurrence by over 50%.29,30 Further, they have been associated with a beneficial effect on BMD and hip fracture risk.31-33 The typical starting dose of hydrochlorothiazide is 25 mg daily, but it is not uncommon to require at least 50 mg/day for adequate urinary calcium reduction. A 24-hour urine collection can be repeated after 6-8 weeks of therapy. If a reduction below the hypercalciuric range defined above has not been achieved, one should consider increasing the thiazide dose. It is important to avoid diuretic-associated hypokalemia (low serum potassium), which may induce hypocitraturia. Some clinicians add a potassium-sparing diuretic, such as amiloride, to thiazide therapy to avoid hypokalemia and possibly further reduce calcium excretion in a synergistic manner.

Bisphosphonate therapy

Several studies have demonstrated a beneficial effect of bisphosphonates on urinary calcium parameters and BMD in people with IH.34,35 A recent randomized controlled trial studied the effect of alendronate and indapamide (a thiazide-related diuretic), alone or in combination, on 24-hour urine calcium and BMD after one year of therapy in 77 hypercalciuric postmenopausal women with low BMD. The authors concluded that combination therapy was superior to alendronate alone with regards to minimizing urinary calcium excretion and optimizing lumbar spine BMD.36

Key diagnostic and management points

A significant link exists between low BMD and nephrolithiasis. The following are some key points:

- Measurement of 24-hour urinary calcium excretion is an important component of secondary bone loss evaluation, since an abnormal result has specific therapeutic implications. IH is diagnosed when elevated urinary calcium is found in at least two urine collections, under optimal dietary conditions, in a normocalcemic state, and when secondary causes of hypercalciuria have been eliminated.
- High sodium and animal protein intakes may exacerbate hypercalciuria and BMD loss, and should be avoided.
- IH patients should maintain a normal daily calcium intake, with supplements (taken with meals) if needed. Calcium citrate may be a preferable supplement form.
- Patients may benefit from a thiazide diuretic to reduce their urinary calcium excretion. Care must be taken to avoid diuretic-associated hypokalemia.
- Vitamin D supplementation is likely safe in IH when given at usual doses for bone health, but there is little research addressing this issue.
- The addition of a bisphosphonate may be appropriate in particular patients with high risk of fracture, but further studies are required.
- Finally, individuals with IH and recurrent stone formation may benefit from referral to a renal stone specialist for a complete metabolic evaluation.

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- Finally, individuals with IH and recurrent stone formation may benefit from referral to a renal stone specialist for a complete metabolic evaluation.
George Ioannidis, PhD, responds: Osteoporosis-related fractures are a major health concern in Canada. The consequences of fracture are serious, including death. In a recent CaMos (Canadian Multicentre Osteoporosis Study) study of 7783 community-dwelling women and men aged 50 years and older, researchers found a 2.7 and 3.2 fold increase in death over a 5-year period in individuals who developed a new spine or hip fracture, respectively (CMAJ 2009;181[5]:265-71). Other fractures (e.g. wrist, forearm, ribs) were not found to have an impact on mortality. Findings were adjusted for other factors that might influence mortality, such as comorbid diseases, medications, health-related habits (e.g. smoking, physical activity), education level and quality of life. In contrast to other studies suggesting increased mortality for men after fractures, this study did not find differences between men and women.

While a broken hip may not be a direct cause of death in the early post-fracture phase, the resultant immobility can be a trigger for life-threatening processes such as pneumonia, DVTs, etc. Moreover, it may lead to a progressive decline in health due to lack of mobility and loss of muscle mass and strength, in turn causing disability and other negative health consequences, including eventual death. Vertebral fracture was found to be an independent predictor of death. It may influence death directly by causing chronic back pain, immobility and postural change, and increased risk of infection.

These findings are a wake-up call for physicians to carefully monitor individuals who are at risk or have had an osteoporotic fracture. All postmenopausal women and men over 50 years of age should be assessed for risk factors for fracture. Those with major risk factors (prior fracture, use of systemic glucocorticoid therapy, family history of fracture, propensity to fall) and individuals over age 65 should have a bone mineral density (BMD) test. For people at high risk, treatment should be initiated; in Canada, we have access to therapies that can reduce the risk of new fractures by approximately 50%, and some of these work as early as within 6 months. Calcium and vitamin D are important additions to pharmacologic interventions.

Steps should be taken to reduce the likelihood of falling — a major cause of injury among older people. Physical activity should be encouraged to improve strength, balance and flexibility. Since poor vision may also contribute to accidents and falls, regular eye exams should be performed (Harwood RH et al. Br J Ophthalmol 2005;89:53-9).

Patients need to take care to remove fall hazards around the home: keep stairs and walkways uncluttered, secure or remove rugs, and rearrange kitchens cupboards and closets so that items are within easy reach. Finally, following a fracture, appropriate rehabilitation efforts need to be conducted to improve strength, range of motion and mobility so that individuals can return home, instead of to long-term care facilities.

Appropriate osteoporosis management should improve patient health-related outcomes and, hopefully, reduce the likelihood of death following a fracture. However, further research is needed to determine the precise relationship between fracture rates and death.

Panagiota Klentrou, PhD, explains: Recent cross-sectional studies have reported low bone mass in competitive male cyclists (Medelli J et al. J Clin Densitom 2009;12: 28-34; Smathers AM et al. Med Sci Sports Exerc 2009;41: 290-6). A longitudinal study of a group of 27- to 44-year-old male competitive cyclists followed for one year has also found that BMD decreased significantly in the peripheral regions, but not in the lumbar spine (Barry DW, Kohrt WM. J Bone Miner Res 2008;23:484-91). On the other hand, this is not necessarily unique to cycling — past studies have shown that some endurance athletes have low BMD, possibly related to low body weight and fat mass (Hind K et al. Bone 2006;39:880-5). While of great interest, these studies reflect a select group of competitive athletes and may not apply to the general population.

Lending further confusion to this story is a study by a group of British researchers who examined the link between physical activity and self-reported incidence of fractures in 34,000 men and women, 20 to 89 years old, in the UK. They found a significant increased risk of fractures associated with bicycling and a moderately increased risk related to other sports. It was hypothesized that this was likely due to a higher incidence of injury (Appleby PN et al. J Bone Miner Metab 2008;26:191-8).

As always, the balance of the benefit of the activity vs the risk that it may impart becomes key. There doesn’t appear to be substantial data to indicate that casual bicycling would negatively impact bone health. However, people who are at increased risk of fractures may want to pick and choose their cycling activities carefully to help minimize the risk of falls and potential injury. Since there are a variety of exercises and activities recommended for people with low BMD, taking intense cycling off the list may have merit.
Lindy Fraser Memorial Award

The Lindy Fraser Memorial Award was created in honour of an Ottawa woman who was among the first in Canada to be treated with newer osteoporosis therapies, and who, in 1981, went on to create the first self-help group for others with this condition. Her voluntary efforts on behalf of her fellow citizens prepared the ground for the development of Osteoporosis Canada.

It is with great pleasure that we announce the Lindy Fraser Memorial Award winner for 2009 — Dr. Suzanne Morin. Dr. Morin was nominated by her colleagues on the Scientific Advisory Council (SAC) and is recognized for her dedication to Osteoporosis Canada, where she has served as a consultant since 2005.

Dr. Morin has served on the Osteoporosis Canada Board and as Chair of the Board Development Committee, and is currently a member of the SAC Executive Committee. She was a driving force behind the establishment of Osteoporosis Canada’s Montreal Chapter, and has been actively involved on their executive as president. Dr. Morin is also a tireless promoter and speaker at scientific and public forums.

Dr. Morin is Associate Professor in the Department of Medicine at McGill University, and Internal Medicine Clinic Director at the Montreal General Hospital. Her research interests include pharmacologic therapies and health-related outcomes for osteoporosis, particularly following hip fractures.

Kudos to Dr. Suzanne Morin from all of us at Osteoporosis Canada!

OC–CaMos Fellowship Award

Osteoporosis Canada (OC) and the Canadian Multicentre Osteoporosis Study (CaMos) have recently formed a new partnership to provide a one-year fellowship award of $20,000 for a graduate student or postdoctoral fellow to pursue research training with CaMos investigators. By supporting bright young minds, the award encourages advances in research aimed at improving osteoporosis prevention, diagnosis and management.

OC and CaMos chose the following recipients for the 2009 award from a strong group of applicants:

• Lisa-Ann Fraser, Department of Clinical Epidemiology & Biostatistics, Health Research Methodology Program, McMaster University; Research project — The study of Canadian women who sustain a fragility fracture, to determine if a care gap is present (CaMos Mentor, Dr. Alexandra Papaioannou)

• Kyle Nishiyama, Department of Mechanical Engineering, University of Calgary; Research project — In vivo quantification of cortical bone porosity by high-resolution peripheral quantitative computer tomography (CaMos Mentor, Dr. Steven Boyd)

Best wishes to Lisa-Ann Fraser and Kyle Nishiyama as well as to all the candidates as they embark on their exciting careers in the field of osteoporosis.

For more information, please visit www.camos.org.

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Complete references are available upon request: osteo@parkpub.com
Osteoporosis Canada is a national, not-for-profit organization dedicated to educating, empowering and supporting individuals and communities in the risk reduction and treatment of osteoporosis. The organization, guided by its Scientific Advisory Council (SAC) made up of osteoporosis experts from across the country, works with healthcare professionals to make the latest prevention, diagnostic and treatment options available to Canadians.

www.osteoporosis.ca

INTERNATIONAL SOCIETY FOR CLINICAL DENSITOMETRY (ISCD) 16th ANNUAL MEETING

Evolution of Skeletal Health over the Bone and Joint Decade

March 10-13, 2010
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ISCD BONE DENSITOMETRY COURSES

The ISCD offers courses for clinicians, technologists, scientists, researchers and healthcare providers.

For information and locations around the world, contact Anabela Gomes:
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