Bone health in children


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Skeletal maturation (and attainment of peak bone mass) occurs at approximately 18 years of age for females and 20 for males. After achieving peak bone mass, there is generally a plateau in bone mineral density (BMD) followed by a steady decrease after the third or fourth decade of life. Peak bone mass is a key contributor to adult bone strength. As peak BMD is achieved relatively early in life, children with low BMD are considered at greater risk of developing adult osteopaenia / osteoporosis (OP), and possibly increased risk of fractures and suchlike. Therefore, early childhood risk factors are important.

Measurement of childhood BMD

As children have continuous physiologic change in bone size and shape, it may be difficult to accurately interpret children's bone mineral density (BMD) results. Adult reference values do not factor for body weight and pubertal stage, thus adult BMD references and definitions of osteoporosis and osteopaenia do not apply to children.

Z-scores (in the form of standard deviations) are a more accurate reflection of bone health as it is standardised to age-related norms. A Z-score of -2 is considered significant.

Currently there is no agreement to the definitions of childhood osteopaenia and osteoporosis for children, hence the term "low bone mineral density" (LBMD) is sometimes preferentially used. A child is thought to have osteoporosis (OP) when they have LBMD (z-score -2) and a history of clinically significant fracture/s.

Childhood risk factors

Whereas an individual's optimal peak bone mass is primarily influenced by genetic factors (i.e. gender, ethnicity, hormonal status), it is environmental factors (e.g. nutritional state, physical activity/lifestyle, sunlight exposure) that can alter skeletal maturation and influence whether someone reaches their genetically determined peak bone mass.

There is a worrying trend of increasing childhood and adolescent obesity. Combined with a sedentary lifestyle and lack of regular exercise (especially weight-bearing exercise), poor nutrition and suboptimal vitamin D intake, some children may not reach their optimal bone mass and skeletal strength.

As it happens, other secondary causes of childhood LBMD/osteoporosis are more common – medication (glucocorticoids, methotrexate, heparin, anticonvulsants), chronic disease (including rheumatologic or systemic inflammatory disease), and immobilisation (cerebral palsy, neurologic conditions, post-traumatic osteoporosis).

Primary causes include osteogenesis imperfecta and juvenile idiopathic osteoporosis. These children often do not reach optimal bone mass.

Strengthening childhood bones

Optimising children's bone health is crucial in order to reach maximal bone mass. Management points to achieve this include:

- Adequate control of underlying disease.
- Ensuring adequate calcium and vitamin D consumption.
- Optimising growth – nutritional intake, hormonal factors.
- Sunlight exposure – adequate but not excessive, and not exacerbating underlying disease (e.g. systemic lupus erythematosus).
- Optimising levels of physical activity – especially weight-bearing exercise.
- Minimising factors that have adverse effects on bone (e.g. reducing glucocorticoid medications to minimal effective dose, or considering steroid-sparing agents)
- Consideration of anti-resorptive medications – bisphosphonates.

Adequate daily oral calcium intake is essential for normal bone homeostasis, remodelling and growth. Current recommendations for daily intake vary with age:

1-3 years = 500mg

4-8 years = 800mg

9-18 years = 1300mg

Calcium supplementation in children has been found to produce modest improvements in BMD, however whether supplement cessation leads to sustained effects remains inconclusive.

Steroid warning

It is well know that long-term corticosteroid use has negative effects on bone (increased risk of LBMD, OP, fracture, avascular necrosis), along with numerous other adverse effects such as growth impairment, cataracts, obesity, and adrenal suppression.

While physicians discourage the use of long-term systemic corticosteroid therapy, exposure is sometimes inevitable. In rheumatology patients, strategies to reduce corticosteroid adverse events include choosing non-steroidal anti-inflammatory drugs (NSAIDs), short-term oral corticosteroid course, pulse intravenous corticosteroid therapy, intra-articular corticosteroid injections, and using steroid-sparing disease modifying agents (e.g. methotrexate, leflunomide) early in the course of disease. With the advent of biologic medications which modify the immune system (e.g. etanercept, adalimumab, infliximab, anakinra, tocilizumab, rituximab), there are now more treatment options available for patients who may have refractory disease.

Bisphosphonates in children

Clinically, bisphosphonates are only licensed for use in adults with post-menopausal osteoporosis, osteoporotic patients who develop fractures from minimal trauma, and in other conditions (Paget's disease, refractive hypocalcaemia secondary to malignancy, multiple myeloma, and steroid-induced osteoporosis). Physicians must seek PBS authorisation.

Bisphosphonate "off-label" use in children (with appropriate prior approval) can help specific childhood conditions. In osteogenesis imperfecta, they reduce bone pain and fractures, and improve quality of life. Beneficial effects have been demonstrated in avascular necrosis (e.g. Perthes' disease, slipped capital femoral epiphyses with avascular necrosis of femoral head, avascular necrosis secondary to corticosteroid use, and osteonecrosis secondary to chronic disease).

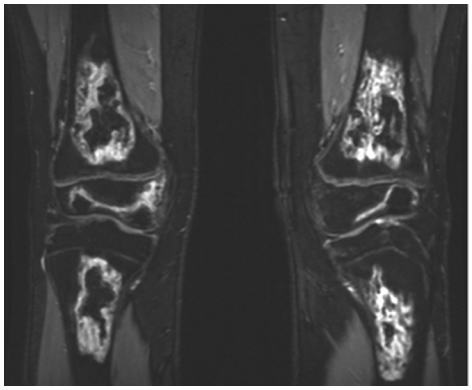

Figure 1. Avascular necrosis of bone (bilateral distal femur and proximal tibia) in a 15-year old boy previously treated with corticosteroids as part of chemotherapy for acute lymphoblastic leukaemia.

In children, the most common side effects of bisphosphonate use are transient fever, headaches, "flu-like illness", and asymptomatic hypocalcaemia. No cases of mandibular osteonecrosis have been reported. Studies are being conducted worldwide (and in PMH) regarding the safety and efficacy of bisphosphonates in children.

References available upon request

Take home points

- Low bone mineral density and osteoporosis can occur in children.
- Peak bone mass is reached in late teens, but environmental factors (e.g. obesity, decreased physical activity, poor nutrition) affect whether individuals reach their expected peak bone mass.
- Suboptimal peak bone mass may lead to future morbidity (e.g. fractures).
- Long-term systemic corticosteroid use should be avoided wherever possible particularly in children to prevent the occurrence of osteopaenia, osteoporosis, fracture and avascular necrosis.
- 'Off label' bisphosphonate therapy in children may help osteogenesis imperfecta, and steroid-induced osteoporosis or avascular necrosis of bone.