Osteoporosis in childhood

Building better bone

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Childhood osteoporosis can impact on development and function and can have manifestations into adult life. It can result from a genetic cause (primary) or be secondary to other medical disorders or treatments. Monitoring and maximising the bone health of all children is important, especially those with risk factors for osteoporosis.

Bone health in children is increasingly a topic of interest, particularly as poor bone health is often secondary to other diseases or medical treatments. Childhood osteoporosis impacts on development and function, and it also leads to health issues and mortality in adulthood.[1,2] For these reasons, it is important that all healthcare providers consider bone health in children.

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Bone strength relates to bone mass, size and quality, with quality being influenced by bone structure, formation, resorption and mineralisation.[3] Peak bone mass and strength is achieved in late adolescence,[4,5] with 25% of peak bone mass gained around the time of the pubertal growth spurt.[3] This is also the period of highest fracture risk during childhood, as individuals participate in higher-risk physical activity and their bone mineralisation lags behind bone growth.[6] Traumatic fractures occur in up to 50% of children, with the highest incidence being in early to mid-puberty.[6]

Genetic factors account for 80% of bone mass variability;[7] muscular, hormonal, environmental and nutritional factors are also important. It is the modifiable factors that can be optimised while children are still growing to reduce the risk of osteoporosis.

Definitions

In 2014, the International Society for Clinical Densitometry (ISCD) updated its definition for paediatric osteoporosis. It is important to note that the definition of osteoporosis in children is different from that in adults. A diagnosis of osteoporosis in children requires:

- vertebral compression (crush) fractures, in the absence of local disease or significant trauma, or

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Key points

- **Bone health is an important issue for all children.**
- **Osteogenesis imperfecta is the most common primary (genetic) cause of osteoporosis in children. Common secondary causes include glucocorticoid use, immobility due to neurological and muscular diseases, chronic inflammation and malabsorption.**
- **Clinical criteria are the focus of the definition of osteoporosis in children, with bone fragility being demonstrated by clinical outcomes. The condition cannot be diagnosed purely on the basis of low bone mineral density.**
- **Children with primary osteoporosis tend to present early in life with multiple fractures. Conversely, secondary osteoporosis in children is often asymptomatic, even when vertebral fractures are present.**
- **Monitoring and maximising the bone health of all children is important, especially those with risk factors for osteoporosis.**
- **In some patients, the use of bisphosphonates may be appropriate after other factors that affect bone health have been addressed.**

- a clinically significant fracture history, as well as bone mineral density (BMD) at least two standard deviations below age- and sex-related values (i.e. Z-score of -2.0 or lower).

A clinically significant fracture history is defined by the ISCD to be: two or more long bone fractures by 10 years of age; three or more long bone fractures by 19 years of age; or a vertebral compression fracture.[8] Reference values for children must be used in Z-score calculations, and the scores need to be adjusted for height.[9] Osteoporosis in children is further defined as primary (where there is an inherent bone abnormality) or secondary (as a consequence of another disease or as a side effect of treatment).[2] In practice, it can be difficult to decide what constitutes mild to moderate trauma.[8] Even healthy bones can fracture from high-energy trauma, but a fracture that is caused by low-energy trauma is more likely to reflect underlying risk for future fractures and abnormal bone.[8] Children who fracture may have lower BMD.[10]

There are important differences between the definitions of osteoporosis for children and adults. Clinical criteria are the focus of the definition of osteoporosis in children, with bone fragility being demonstrated by clinical outcomes, whereas densitometry is the focus used in adults.[11] The term 'osteopenia' is not used to describe low bone mass or density in children (unlike in adults).[9]

Aetiology

The primary and secondary causes of osteoporosis in children are discussed in

Table 1. Primary causes of osteoporosis in childhood

Cause	Comments
Osteogenesis imperfecta (OI)	Features include bone fragility, small bones and short stature Collagen defects affect other organs (eye, ear, dermis, blood vessels, joints, teeth) There are five subtypes of OI, which are defined by distinct phenotypes. The original Sillence classification has been modified to reflect new understandings of genetics and pathophysiology:[12,13] • type 1 – nondeforming bone fragility with blue sclerae • type 2 – perinatally lethal OI with deformity and pulmonary hypoplasia • type 3 – progressively deforming OI • type 4 – common variable OI with normal sclerae • type 5 – OI with calcification in interosseous membranes Non-accidental injury should be considered as a possible differential diagnosis for OI
Idiopathic juvenile osteoporosis	Typically presents as bone pain in lower limbs, walking difficulties and multiple fractures (including vertebral compression fractures) Improves in adolescence[14] Diagnosis of exclusion
Osteoporosis-pseudoglioma syndrome	Causes severe childhood osteoporosis and blindness

Table 1[12-14] and Table 2,[15-23] respectively. Primary osteoporosis tends to present early with multiple fractures. In contrast, secondary osteoporosis is often asymptomatic, even when vertebral fractures are present.[24]

Primary causes

Osteogenesis imperfecta (OI) is the most common genetic cause of childhood osteoporosis, with an incidence of 1 in 20,000 births.[25] In 95% of patients, it is due to autosomal dominant inheritance of a mutation in either the *COL1A1* or *COL1A2* gene. Features of OI include bone fragility, small bones and short stature, but the presentation of OI and its severity depend on the subtype and there is variation within affected families. The collagen defect has effects on other organs, such as the ear (hearing loss), eye (blue sclerae in type 1 OI [Figure 1]), dermis (elasticity and poor healing), blood vessels (easy bruising), joints (hypermobility), and teeth (dentinogenesis imperfecta [Figure 2]). Complications of OI include short stature, scoliosis and dental malocclusion (resulting from mid-face hypoplasia but continued mandibular growth). Approximately 20% of patients develop hearing loss by the end of adolescence (types 1 and 3 OI),[26] which is mostly conductive and becomes disabling in 50% of patients by 50 years of age.[27] Vestibular dysfunction can occur. Basilar invagination, which can cause brainstem compression and hydrocephalus may also develop.[25,27]

Other causes of primary osteoporosis in children, which include osteoporosis pseudoglioma syndrome and idiopathic juvenile osteoporosis (which is a diagnosis of exclusion), are rare.

Secondary causes

Secondary osteoporosis in children develops as a consequence of chronic disease or poor mobility or as a medication side effect. Glucocorticoid medications are a common cause of osteoporosis, particularly when associated with immobility or underlying inflammatory disease. Bone development requires adequate calcium and vitamin D stores, as well as adequate muscle bulk and protein intake; nutritional deficiency secondary to poor oral intake or malabsorption may cause osteoporosis.[22] Although obesity is associated with increased fracture risk due to increased force on bone,[28] it does not cause reduced bone density.

Figure 1. Blue sclera in a child with osteogenesis imperfecta.

Assessment

The flowchart on page 12 outlines a pathway for the assessment and management for a child in whom there is a suspicion of bone fragility.[29]

History and examination

The first step involves detailed clinical assessment. Enquiry should be made regarding mobility, leg or back pain, social concerns, developmental milestones, easy bruising, nutrition, medical conditions and medication history. Hypermobility may manifest through difficulties with handwriting or foot pain with walking. A family history of fracture or hypermobility should be noted.

A careful fracture history should be taken, which includes the child's age at the time of fracture, fracture mechanism and healing after any previous fractures. Children have different fracture patterns from adults, due to rapid growth and bone turnover – buckle fractures, for example, are a common injury in children.[30] Vertebral compression fractures without trauma are considered pathological in children (Figure 3).[9] Fractures of the femur or pelvis are rare

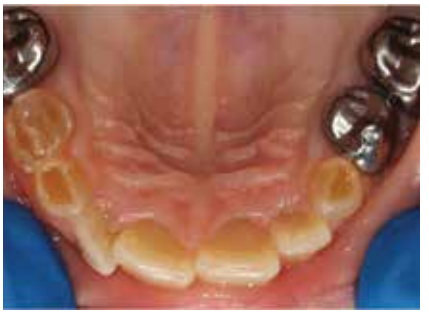

Figure 2. Dentinogenesis imperfecta.

(Figure 4). It is important to consider the possibility of non-accidental injury in situations in which an injury is inconsistent with a child's developmental ability or the mechanism history, or when a child presents with repeated injuries.

A clinical examination must be conducted, with specific attention paid to dysmorphic features, hearing loss, scleral colour, dentition and skin hyperelasticity. It should also note growth, pubertal status, scoliosis, spinal tenderness and deformity. Evidence of possible abuse, such as bruises of a variety of ages, bite or burn marks, should also be noted.

Screening investigations

Useful screening blood tests for a child when there is a suspicion of bone fragility include: a full blood count; calcium, magnesium and phosphate levels; coeliac serology; thyroid function tests; 25-hydroxyvitamin D (25-OH vitamin D) level; liver function tests; and parathyroid hormone level. A urinary calcium/creatinine ratio is a useful measure of urinary calcium excretion. There may also be a role for urine screening of metabolic diseases, depending on the child's clinical features.

It is important to note that bone markers, particularly alkaline phosphatase, are influenced by growth, and so levels of these markers may be transiently abnormal in children.[31]

Specific investigations

Lateral spine x-ray

Although vertebral compression fractures are diagnostic of childhood osteoporosis,[8] they are often asymptomatic and frequently

Table 2. Secondary causes of osteoporosis in childhood

Cause	Comments
Immobility (may be due to cerebral palsy, spinal cord injury or Duchenne muscular dystrophy)	Poor nutrition and lower mobility level correlates with lower BMD[15] Children with cerebral palsy who are unable to walk have between 6 and 12% fracture prevalence[16] Malnutrition, poor growth, pubertal delay, medications and chronic lung disease may have additive negative effects
Glucocorticoid medications	Fracture risk increases with dose and length of exposure; duration of increased risk after treatment unclear; no threshold for increased risk[1] Delayed puberty due to reduced gonadotropin and sex hormone production has additive negative effect
Other medications	Antiepileptic medications affect vitamin D degradation[17] Other secondary causes include aromatase inhibitors, proton pump inhibitors, excess thyroxine replacement and diuretics[18]
Malnutrition	Bone development requires adequate calcium intake and vitamin D stores, as well as adequate muscle bulk and protein intake May be due to malabsorptive disorders, such as cystic fibrosis, coeliac disease, inflammatory bowel disease
Chronic inflammation	Inflammatory markers stimulate bone resorption; medications (e.g. glucocorticoids) and malabsorption may have additive negative effect
Endocrine diseases	Hypothalamic–pituitary–adrenal axis, gonadal function, parathyroid and thyroid function all impact on bone health Hyperparathyroidism results in hypercalcaemia due to increased bone resorption Adrenal excess results in glucocorticoid excess and associated cushingoid appearance and poor growth
Pubertal delay	Men with pubertal delay have higher fracture rate and smaller bones[19] Women with pubertal delay have increased risk of fracture[20]
Malignancy	Haematological malignancy is associated with increased fracture risk and reduced BMD at diagnosis; BMD decreases further during treatment but usually normalises post-treatment[21] Cranial irradiation negatively impacts on BMD
Thalassaemia	Iron deposition causes hypogonadism, hypothyroidism, diabetes and/or hypopituitarism; there is also marrow expansion from haemopoiesis Iron is toxic to bone-forming cells[22]
Transplant surgery (solid organ or bone marrow)	Immunosuppressive medications (glucocorticoids and calcineurin inhibitors) and immobility lead to bone loss Reduced renal function after transplant can lead to secondary hyperparathyroidism Patients may have pre-existing bone disease prior to transplantation
Prematurity (bone mineralisation occurs mostly in the third trimester)	Bone mineral content decreases after birth so clinical onset of bone fragility is usually six to 12 weeks after birth Reaching normal levels of bone mineralisation can take up to 12 months in low birthweight infants[23] Patients often have raised alkaline phosphatase, hypophosphataemia, elevated parathyroid hormone levels and a low vitamin D level

Abbreviation: BMD = bone mineral density.

AN APPROACH TO INVESTIGATION AND MANAGEMENT OF SUSPECTED BONE FRAGILITY IN CHILDREN[29]*

Child with suspected bone fragility

Clinical assessment (mobility, growth, puberty, nutrition, dysmorphic features, joint hypermobility, family history, signs and symptoms of underlying disease)

Exclusion of non-accidental injury

Screening investigations for bone health (CMP, FBC, vitamin D, LFTs, PTH, coeliac serology, TFTs)

Detailed fracture history

Clinically significant?

Vertebral compression fractures in absence of trauma

and/or

Two or more long bone fractures by 10 years of age or three or more long bone fractures by 19 years of age

Yes

Specific investigations
- Bone density assessment: DXA ± pQCT
- Genetic studies
- Lateral spine x-ray
- Consideration of bone biopsy if diagnosis is inconclusive

Treatment and management of complications
- Optimise bone health
- Maximise mobility and physical activity
- Minimise oxteotoxic medications
- Screen for vertebral compression fractures
- Consider bisphosphonate therapy
- Manage associated conditions (e.g. dentinogenesis imperfecta, hearing loss)

No

Optimisation of bone health
- Calcium intake
- Vitamin D
- Physical activity
- Pubertal progression
- Adequate nutrition
- Identification and treatment of malabsorption syndromes
- Avoidance of osteotoxic medications
- Consideration of DXA (but will not be diagnostic of osteoporosis)

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Abbreviations: CMP = calcium, magnesium and phosphate levels; DXA = dual x-ray absorptiometry; FBC = full blood count; LFTs = liver function tests; pQCT = peripheral quantitative computed tomography; PTH = parathyroid hormone level; TFTs = thyroid function tests.

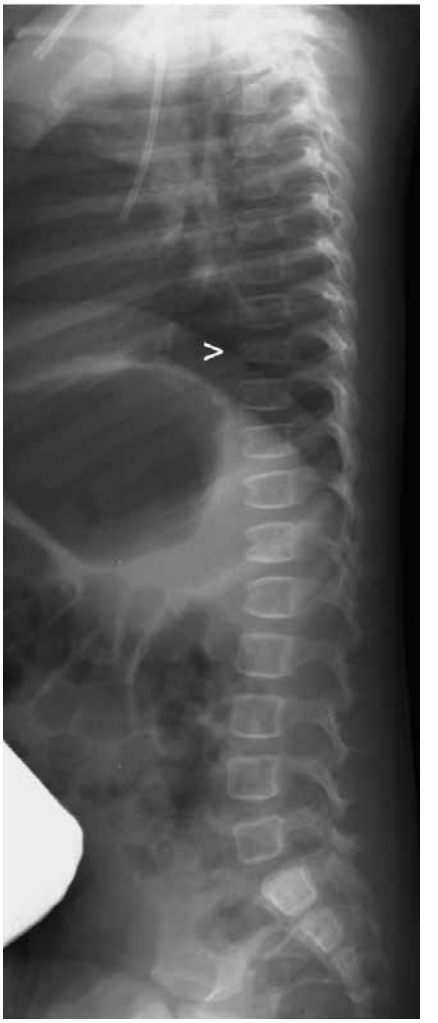

Figure 3. Vertebral compression fractures in an asymptomatic child on long-term glucocorticoid therapy.

occur without associated spinal tenderness.[32] Lateral spinal x-ray is therefore important in the diagnosis of vertebral fractures, and should be performed in children at high risk or with low BMD.

Dual x-ray absorptiometry

Dual x-ray absorptiometry (DXA) is the most commonly used method for assessing bone mineral content and density in children, but it is important to note that a diagnosis of osteoporosis in childhood does not reflect just DXA data.[9] A higher fracture risk in healthy children is associated with abnormal DXA results.[33] However, there is little known regarding bone density in children with

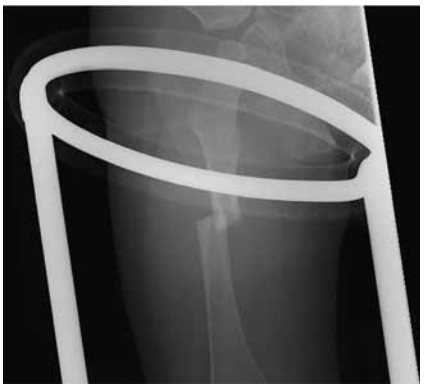

Figure 4. A femur fracture in a child with osteogenesis imperfecta.

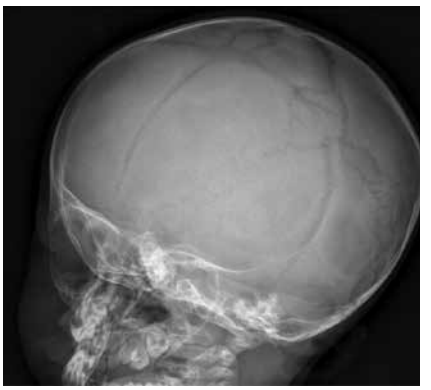

Figure 5. Skull x-ray showing Wormian bones in a child with osteogenesis imperfecta.

chronic diseases, and a child's bone can fracture despite having normal densitometry values.[34]

There are limitations of DXA with regard to bone size. Bone size is continually increasing in children, leading to an apparent increase in DXA bone density over time due to bone growth, but this is not a true increase in bone density. In children with short stature, this can lead to misinterpretation because bone density will be low for age due to the short stature and not because of any underlying bone disorder.[35] BMD in children must be reported using Z-scores (instead of T-scores), reflecting standard deviations from the mean for age and sex, and also needs to be adjusted for height.

Peripheral quantitative computed tomography (pQCT)

Peripheral quantitative computed tomography (pQCT) is an emerging technique currently used mostly in a research capacity. In contrast to DXA, pQCT is a three-dimensional measurement and gives volumetric measures of bone density for trabecular and cortical bone separately.

Genetic testing

Genetic testing may be considered after specialist review. Approximately 95% of Caucasian children with OI have a mutation in either the *COL1A1* or *COL1A2* gene, and mutations in non-collagen genes have also been identified recently as causes of OI. Next generation sequencing may assist our understanding of the genetics of osteoporosis.[36]

Bone biopsy

A bone biopsy from the iliac crest can be useful when the cause for osteoporosis in a child is unclear, such as when no mutation is found on genetic testing but there is a phenotype of bone fragility. Bone volume, turnover and microarchitecture can be analysed, with the aim of eliciting the underlying aetiology. However, the use of bone biopsy has been limited because of its invasive nature and the requirement for general anaesthesia.[37]

Skeletal survey and bone scan

In infants presenting with fracture, it is important to differentiate OI from non-accidental injury, assisted by skeletal survey and bone scans. Cortical thinning and multiple Wormian bones (accessory skull bones completely surrounded by a suture line [Figure 5]) are seen in 70% of infants with OI on Towne's view skull x-ray.[38] Although these bones can be seen in children who do not have OI, the presence of more than 10 Wormian bones significantly increases the probability of this diagnosis.[39]

Old fractures that are suspicious for child abuse, such as posterior rib or metaphyseal corner ('bucket handle') fractures, may be evident on skeletal survey. Subclinical or old fractures may be revealed through nuclear bone scans.

Management

The aims of management of osteoporosis in children are to address the underlying cause, improve function and mobility, and limit deformity.[40] These aims are best achieved with a multidisciplinary team that includes physiotherapists, occupational therapists, paediatricians, rehabilitation specialists and orthopaedic surgeons. Identification and appropriate management is necessary for comorbid conditions, such as thyroid dysfunction, coeliac disease, malnutrition and obesity. Suggestions for specialist referral are provided in Box 1.

Optimising bone health

Monitoring and maximising the bone health of all children is important, especially those with risk factors for osteoporosis. This includes avoiding or minimising osteotoxic medications such as glucocorticoids, if possible, encouraging and improving mobility and maintaining good general nutrition, including adequate protein intake.

Calcium and vitamin D

Adequate calcium intake is important for bone development,[41] with international recommendations for calcium suggesting a daily intake of 800 to 1300 mg in children.[42] Although calcium supplementation appears to have little impact on bone density in healthy children,[43] in children who are deficient in dietary calcium, increased dairy intake has been shown to improve BMD and to reduce fracture risk in adulthood.[3]

Vitamin D deficiency is common in infants and children.[44,45] Those at particularly high risk include:

- infants of vitamin D deficient mothers
- exclusively breastfed infants
- premature infants
- children with dark skin
- children who wear veils
- children with limited sun exposure.

The majority of vitamin D is obtained from sun exposure, with diet providing only 10% of vitamin D requirements. Malabsorption also increases the risk of vitamin D deficiency, and medications (such as anticonvulsants) may affect vitamin D metabolism. Rickets and hypocalcaemia can result from severe deficiency.

1. Children with suspected bone fragility: a referral guide

Who to refer

- Any child with vertebral fractures
- A child with two or more long bone fractures by 10 years or age or with three or more long bone fractures by 16 years of age
- A child with an underlying diagnosis that predisposes to bone fragility and who sustains a fracture (see Table 1)

When to refer

- Arrange referral while concurrently optimising bone health through nutrition, vitamin D, mobility, avoidance of osteotoxic medications

Where to refer

- General paediatrician
- Paediatric endocrinologist
- Clinical geneticist (if clinical suspicion of primary osteoporosis)

Note: if organising a dual x-ray absorptiometry (DXA) scan for a child, ensure that the radiology facility will report Z-scores and not T-scores

Table 3. Management of vitamin D deficiency[45]*

Age	Oral dose of vitamin D3	
	Treatment	Maintenance and prevention in individuals with ongoing risk factors
Preterm		
Mild deficiency	200 IU/kg/day, maximum 400 IU/day	200 IU/kg/day, maximum 400 IU/day
Moderate or severe deficiency	800 IU/day, review after one month	200 IU/kg/day, maximum 400 IU/day
<3 months old		
Mild deficiency	400 IU/day for 3 months	400 IU/day
Moderate or severe deficiency	1000 IU/day for 3 months	400 IU/day
3 to 12 months old		
Mild deficiency	400 IU/day for 3 months	400 IU/day
Moderate or severe deficiency	1000 IU/day for 3 months, or 50,000 IU stat	400 IU/day
1 to 18 years old		
Mild deficiency	1000 to 2000 IU/day for 3 months	400 IU/day
Moderate or severe deficiency	3000 to 4000 IU/day for 3 months, or 150,000 IU stat and repeat in 6 weeks	400 IU/day

* Summarised from the 2013 Australian and New Zealand position statement for vitamin D in pregnancy, infants, children and adolescents. For full details, including dosing and monitoring information, refer to reference 45 (http://aimss.org.au/articles/news/vitamin-d-and-health-pregnancy-infants-children-and-adolescents-australia-and-new).

Levels of 25-OH vitamin D need to be maintained above 30 nmol/L to prevent nutritional rickets[28] and above 50 nmol/L to optimise BMD.[45,46]

Australian guidelines for the management of vitamin D deficiency in children are summarised in Table 3.[45] As there are differences in climates and UV indices across Australia, it is difficult to make country-wide recommendations regarding adequate sun exposure to prevent vitamin D deficiency,[47] while still considering the need for sun protection. The Australasian Paediatric Endocrine Group recommends that all infants receive 400 IU of vitamin D daily until 12 months of age.[48]

Mobility and physical activity

Muscle load is crucial for bone development. Physical activity optimises bone mass, assists in muscle recovery after any immobilisation and improves motor coordination.[49] Children with significant osteoporosis have decreased gross motor, fine motor and self-care function.[50,51] Physical therapy aims to improve overall function by increasing muscle mass and strength,[40] and should begin as soon as there is evidence of gross motor delay or muscle weakness. Activities that promote core strength, such as swimming and pilates, are beneficial and have low risk of fracture.[52] Orthotics and supportive footwear may improve gait and reduce pain for patients who have pes planus. Advice should be provided about avoiding postures that promote hypermobility, such as external rotation of the hip.

At school, an inclusive approach to physical education while ensuring physical safety is important. Modifications to activities may be needed so a child can participate. Spinal precautions are needed in all settings, and high-impact activities (such as contact sports, gymnastics, trampolining and jumping on hard surfaces) should be avoided.[53]

Pubertal progression

As peak bone mass and strength are achieved in late adolescence,[4,5] delayed puberty is associated with increased risk of fracture and lower bone density.[19,20] Many secondary causes of osteoporosis, such as cerebral palsy, Duchenne muscular dystrophy and inflammatory bowel disease, are associated with pubertal delay. Timely induction of puberty

with sex hormones may be required to improve bone health and reduce fracture risk.[54] Our practice is to consider pubertal induction for girls at age 14 years and for boys at age 14.5 years.

Bisphosphonate therapy

After other factors that impact on bone health in children have been addressed, the use of bisphosphonates may be considered. Bisphosphonates reduce bone resorption, leading to increased BMD and decreased bone pain.[55] The aims of bisphosphonate treatment are clinical: to improve motor function and mobility, and to reduce fracture frequency.

Most of the available data regarding the use of biphosphonates in children comes from studies of children who have moderate to severe OI. In children who are growing, bisphosphonates have been demonstrated to improve long bone and vertebral deformity.[2,56] They have also been shown to be effective in improving BMD and reducing pain in children with Duchenne's muscular dystrophy, cerebral palsy and immobility.[57]

The most widely used bisphosphonates in children are intravenous pamidronate and zoledronate, and oral risedronate and alendronate (all bisphosphonates are off-label for use in children). Both oral and intravenous bisphosphonates can be used to treat osteoporosis in children. However, oral bisphosphonates have not been shown to be effective with vertebral fractures.[58] Oral bisphosphonates should therefore only be used in children with mild osteoporosis without vertebral compression, while intravenous bisphosphonates are used in children with moderate-to-severe bone fragility or with vertebral compression fractures.

The main side effects of bisphosphonate treatment in children are hypocalcaemia and an acute phase response, both of which tend to only occur with the first dose of intravenous bisphosphonate.[2] Cessation of bisphosphonate treatment can lead to increased risk of fracture between treated and untreated bone.[59] Therefore, bisphosphonate treatment is often continued until a child reaches final height, so as to avoid new fragile bone forming as the child grows.[59]

There is a theoretical concern of bisphosphonate-related osteonecrosis of the jaw, but this has not yet been reported in children.[60] However, dental review before commencing treatment is important, particularly as the underlying condition may be associated with dental issues (such as dentinogenesis imperfecta). If possible, any necessary invasive dental procedures should be undertaken before bisphosphonate treatment is commenced.[60] There may be a risk of bisphosphonate-related osteonecrosis of the jaw well after treatment is ceased because bisphosphonates have a long half-life (a number of years). Bisphosphonates are contraindicated in pregnancy because of the potential for birth defects.

Additional requirements for children with osteogenesis imperfecta

In addition to general bone health measures and consideration of bisphosphonate therapy, children with OI may have other management requirements.

There are no consensus guidelines regarding monitoring for hearing loss in patients with OI. Our practice is to assess patients at diagnosis, at 10 years of age, at the time of transition to adult services, and then five yearly thereafter.

Dentinogenesis imperfecta is common in OI types 3 and 4 and dental review is essential every six to 12 months.[60] Prophylactic capping of molars is often required. There are also increased risks of malocclusion, impaction and altered rates of teeth eruption.[61]

Basilar invagination reflects an anomaly of the occipital bone, allowing the odontoid to abnormally prolapse into the foramen magnum.[62] Neurological complications of basilar invagination should be sought annually, especially in children with OI types 3 and 4, and MRI of the brain and cervical spine should be performed if there is any clinical suspicion. Dentinogenesis imperfecta is associated with an increased risk of basilar invagination.

Infants with recurrent fractures may have gross motor delay.[63] Hypermobility may lead to reduced fine motor skills, such as difficulty with handwriting and poor grasp, in which case occupational therapy can be of benefit.[40] Possible tools include pen grips, wrist or hand splints, slope boards and the use of keyboards (rather than writing).[53]

Conclusion

Childhood is a crucial period for bone development. Osteoporosis in children has many underlying causes, both primary and secondary. Vertebral fractures are often asymptomatic in children and so need to be actively screened for in the setting of osteoporosis. Investigations can be difficult to interpret in children because bone markers and densitometry change with age and growth. Therefore, osteoporosis cannot be diagnosed solely on the basis of densitometry alone without a history of fractures.

Bone health is an important issue in all children. Management strategies to improve bone health include encouraging physical activity, treating any underlying diseases that may impact on bone health, avoiding medications that are toxic to bone (if possible), promoting good nutrition and, if necessary, use of vitamin D supplements. In some patients, the use of bisphosphonates may be appropriate after other factors that affect bone health have been addressed. **ET**

References

A list of references is included in the website version of this article (www.endocrinologytoday.com.au).

COMPETING INTERESTS: None.

Don't miss

'Investigating and managing hirsutism in a young woman' on page 43.

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