By 16 years of age, up to 40% of otherwise healthy girls and 64% of boys will have experienced ≥1 broken bone, with a peak incidence in the peri-pubertal years. Is this just because youth are active or risk takers? An alternative explanation is that the fastest rate of bone mineralization occurs several months after the most rapid gains in bone size during adolescence. The incidence of childhood fractures appears to be on the rise in both the United States and Scandinavia, raising concerns about unhealthy lifestyle trends. Addressing this public health problem requires an understanding of which children are fracturing bones and why.

In the population-based retrospective study of emergency department visits from Ontario, Canada, in this issue of Pediatrics, Escott et al suggest the risk of a broken bone is not equal for all. The study compared the fracture incidence for those with or without a baseline fracture over a 7-year follow-up period. To focus on healthy youth (<16 years of age), children with osteogenesis imperfecta, low bone density, juvenile arthropathy, pathologic fracture, or a history of child abuse or neglect were excluded from analysis. Twenty-three percent of children with a baseline fracture had ≥1 additional fracture during the follow-up period as compared with only 11.3% of those without a fracture at baseline. This rate remained 60% greater than controls without a fracture at baseline after adjustment for sex; rural residence; and history of soft tissue injury, head trauma, or previous fracture.

Previous investigators have also found that a first fracture in childhood increases the likelihood of a future fracture. Authors of a longitudinal study of 605 children from New Zealand reported that the risk of a second fracture doubled after the first and tripled after the second broken bone. Similarly, a study of healthy white youth and youth of color from the United States found a history of previous fractures to be a risk factor for future fractures, especially among white youth.

The correlates of fracture risk remain controversial. The design of the Canadian study precluded analysis of detailed clinical risk factors and used a history of soft tissue or head injury as surrogate markers for increased risk-taking behavior or physical activity. The risk of repeated fracture remained increased after controlling for these variables, indicating other factors were to blame for bone fragility. Goulding et al found that children with ≥1 fracture had a higher rate of obesity, increased body fat, reduced dairy intake, decreased physical activity, and lower bone mineral density (BMD) than their fracture-free peers. By contrast, Wren et al reported higher fracture risk in those with less body fat. Participation in sports was a correlate for previous incident fractures during the 6-year study.

Investigators have also looked for differences in bone mass and microarchitecture in youth with fractures using dual-energy radiograph absorptiometry and peripheral quantitative computed tomography.
Data from cross-sectional and prospective studies of BMD indicated an association between reduced bone mineral for body size and increased fracture risk. In 1 study, fractures were less likely in those with higher BMD for age, except for white boys who paradoxically had an increased fracture risk with higher BMD z scores. Authors of a peripheral quantitative computed tomography-based study found that 8 to 15-year-olds with a history of forearm fractures had deficits in distal radius bone strength and trabecular microarchitecture as compared with controls without fractures.

Given the uncertainties about the causes of recurrent fractures in healthy children, how much evaluation is appropriate after a broken bone? At a minimum, it is important to explore how the injury occurred, the history of previous fractures in the child and family, and the child’s nutritional and physical activity. The site of the fracture and the extent of inducing trauma should guide how much laboratory investigation is warranted. Two-thirds of the fractures in healthy youth involve the distal radius and hand. By contrast, femur, hip, and spine fractures occurring without major trauma suggest abnormal bone fragility. Similarly, fractures at any skeletal site occurring after minimal trauma are uncommon and should prompt laboratory testing.

Recommended studies include the measurement of serum 25 hydroxyvitamin D, calcium, phosphate, creatinine, parathyroid hormone, antibodies for celiac disease, and urinary calcium excretion. Testing for thyroid, growth, or sex hormone abnormalities or genetic mutations may be appropriate in selected cases. The value of dual-energy radiograph absorptiometry in the assessment of bone fragility is controversial because the ability of BMD alone to predict fractures in children and teens is limited.

Children with ≥2 low-energy fractures may warrant a lateral thoracolumbar spine radiograph to look for vertebral fractures, 40% of which may be asymptomatic. A childhood fracture provides an opportune time to counsel families on the importance of early bone health for life. In addition to addressing risk-taking behavior, the practitioner should stress the importance of healthy weight, adequate calcium and vitamin D intake, and regular weight-bearing physical activity. Pharmacologic treatment of youth with low-trauma vertebral or femur fractures requires guidance from a practitioner with expertise in pediatric osteoporosis.

Pediatric bone health is an important public health issue. Casting more light on the causes of fractures in children is key to reducing these injuries and their recurrence.

**ABBREVIATION**

BMD: bone mineral density

**REFERENCES**