Vitamin D Deficiency and Fractures in Childhood

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In an article in this issue of *Pediatrics*, Schilling et al[1] explore the possible role of vitamin D deficiency in the pathogenesis of fractures in young children. This study was stimulated by articles that reported individual cases in which fractures initially ascribed to child abuse were later thought to be a result of rickets.[2,3]

In their study, Schilling et al found evidence of vitamin D deficiency, defined as a serum 25-hydroxyvitamin D level of <20 ng/mL, in ~8% of the children with fractures. The proportion was similar in children thought to have been abused and in those thought not to have been abused. They conclude that vitamin D deficiency was unlikely to have contributed to the fractures.

One difficulty with this logic is that included in the criteria for identifying abuse was the failure of the parents to explain the fractures. Although the lack of an explanation may be a pointer to nonaccidental injury, it is also a characteristic feature of fractures caused by bone diseases such as osteogenesis imperfecta, bone disease of prematurity, and, indeed, rickets.[4] Thus, the criteria for abuse could include patients whose fractures were actually caused by vitamin D deficiency.

The authors found 7 patients with vitamin D deficiency severe enough to raise the serum parathyroid hormone level. It seems odd, despite this finding, to conclude that the deficiency in those patients could not have contributed to the fractures.

Does rickets cause fractures? Older authors thought so.[5,6] Numerous case reports have included fractures.[7–17] In a recent retrospective study, fractures were found in 7 of 40 children younger than 24 months with overt radiologic evidence of rickets.[18]

It is often forgotten that rickets, like osteomalacia in adults, may cause pseudofractures (Looser zones, Milkman fractures),[2,4,9] which may be mistaken for true fractures. They can be distinguished from fractures, in part, by the lack of associated clinical signs and the lack of change in serial radiographs before vitamin D therapy is instituted. However, in a child with good evidence of vitamin D deficiency, distinguishing a pseudofracture from an undisplaced true fracture may be academic.

Another difficulty in the diagnosis of vitamin D deficiency is that the radiologic signs may be absent or unimpressive in cases of children with biochemically severe deficiency, which is particularly true of infants younger than 1 year.[2,19] One 13-month-old boy in Florida had severe hypocalcemia and an undetectable 25-hydroxyvitamin D level but showed no radiologic evidence of rickets in repeated skeletal surveys.[20] In a group of infants with deficiency severe enough to cause convulsions, one-third of them had no metaphyseal abnormalities.[21] Even in older children there is no relationship between the radiologic signs and the severity of the disorder as measured by serum 25-hydroxyvitamin D levels.[22]

One factor that contributes to the discrepancy between radiology and biochemistry is the phenomenon known to earlier pediatricians as the "paradox of rickets."[23] As the deficiency worsens, the epiphyseal changes become less obvious. The classical radiologic signs are not seen in a child who is not growing. For similar reasons, the serum alkaline phosphatase level is often inappropriately normal in children who are not growing.[24]

There is another concern that is highlighted by Schilling et al in their article: of 118 children with fractures, including 37 thought to have been abused, just 10 were evaluated by the metabolic bone service. This may well be a better proportion than in other centers, but is it good enough? One lesson from the recent literature is that fractures in childhood, including metaphyseal fractures, have a substantial differential diagnosis.

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