Vitamin D Status in Abused and Nonabused Children Younger Than 2 Years Old With Fractures

WHAT’S KNOWN ON THIS SUBJECT:
Bone fragility attributed to suboptimal levels of vitamin D has been offered as an alternative explanation for suspected nonaccidental trauma in young children with fractures. Although common, it is unknown whether vitamin D insufficiency increases fracture susceptibility in children.

WHAT THIS STUDY ADDS:
This study is the first to examine vitamin D levels in young children presenting with fractures and indicates that a strong association between suboptimal vitamin D status and fractures related to abuse is unlikely.

OBJECTIVE:
To examine vitamin D levels in children with (1) suspected abusive and accidental fractures, (2) single and multiple fractures, and (3) fracture types highly associated with inflicted trauma.

DESIGN AND METHODS:
A study of children younger than 2 years of age with fractures admitted to a large children’s hospital was performed. Bivariate analysis and test for trend were performed to test for the association of vitamin D status and biochemical markers of bone health with the primary outcomes of fracture etiology, number, and type.

RESULTS:
Of 118 subjects in the study, 8% had deficient vitamin D levels (<20 ng/mL; <50 nmol/L), 31% were insufficient (≥20 < 30 ng/mL; ≥50 < 78 nmol/L), and 61% were sufficient (≥30 mg/mL; ≥78 nmol/L). Lower vitamin D levels were associated with higher incidences of hypocalcemia (P = .002) and elevated alkaline phosphatase (P = .05) but not hypophosphatemia (P = .30). The majority of children sustained accidental fractures (60%); 31% were nonaccidental and 9% were indeterminate. There was no association between vitamin D levels and any of the following outcomes: child abuse diagnosis (P = .32), multiple fractures (P = .24), rib fractures (P = .16), or metaphyseal fractures (P = .49).

CONCLUSIONS:
Vitamin D insufficiency is common in young children with fractures but is not more common than in previously studied healthy children. Vitamin D insufficiency was not associated with multiple fractures or diagnosis of child abuse. Nonaccidental trauma remains the most common cause of multiple fractures in young children.
Bone fragility secondary to suboptimal vitamin D status has been offered as an alternative explanation to nonaccidental trauma in children with unexplained fractures. If suboptimal vitamin D status does in fact increase fracture susceptibility, there is a potential for misdiagnosis of otherwise unexplained fractures as abuse. Conversely, abused children may be at additional risk if inflicted fractures are inappropriately attributed to suboptimal vitamin D status.

Vitamin D status is determined by circulating levels of 25-hydroxyvitamin D [25(OH)D]. Although serum levels of 25(OH)D are inadequate in many American children, optimal 25(OH)D levels are unknown, and multiple criteria have been suggested to define vitamin D deficiency, insufficiency, and sufficiency. Although marked vitamin D deficiency is associated with clinical rickets, no studies have established vitamin D insufficiency as a cause of increased fracture susceptibility in children. This is in contrast to adults, in whom there is evidence that low 25(OH)D levels are associated with fracture predisposition. Importantly, children with overt radiographic rickets infrequently manifest fractures. A recent study found fractures in only 17.5% of children with rickets and exclusively in those who were mobile. Nonetheless, it has been suggested that moderately reduced vitamin D levels may account for multiple unexplained fractures in infants suspected to be child-abuse victims.

Keller and Barnes reported on 4 infants diagnosed with abuse and proposed that vitamin D deficiency rickets rather than abuse was the cause of the bone lesions, although rickets was not diagnosed by the treating physicians and traditional radiographic signs of rickets were absent. In 2 cases, the 25(OH)D level was not measured; in the other 2 cases the 25(OH)D levels were 17.8 ng/mL and 19.6 ng/mL. The authors further proposed that suboptimal vitamin D status in the absence of typical rickets can produce radiological abnormalities that resemble the rib fractures and classic metaphyseal lesions often identified in abused infants.

Although rib and metaphyseal radiographic abnormalities occur in several metabolic bone disorders, including rickets, these fractures also are frequently the result of abuse. Although few injuries are pathognomonic for physical abuse, infants with multiple unexplained fractures often are victims of inflicted injury. The hypothesis that an infant with suboptimal vitamin D status and normally mineralized bones is susceptible to fractures with high specificity for child abuse is a new concept that lacks scientific support. There is no accepted association between serum 25(OH)D levels and bone strength in children; furthermore, a definitive correlation between the severity of rickets and circulating levels of 25(OH)D has not been established. To investigate whether vitamin D inadequacy is an explanation for fractures attributed to abuse, we evaluated children younger than 2 years of age with fractures to compare vitamin D status: (1) between children diagnosed with abusive and accidental fractures; (2) between children with single and multiple fractures; and (3) among children with different types of fractures. In this age group, multiple, rib, and metaphyseal fractures have high specificity for abuse. We hypothesize that vitamin D status will not be associated with a diagnosis of abuse or with the number or type of fractures identified in children younger than 2 years of age.

METHODS

Study Population
We performed an observational study of children younger than 2 years of age admitted to The Children’s Hospital of Philadelphia with a fracture from August 2008 to September 2009. Children were excluded if they had a disease that predisposed them to fracture, were taking medications that affect bone metabolism, or were premature infants who had never been discharged from the hospital after birth. The study was approved by the institutional review board, and written informed consent was obtained from parents of all subjects.

Data Collection
Demographic information and birth history were collected through caregiver interview. Clinical history, laboratory values, radiologic and physical examination findings, and child abuse evaluations were obtained through chart review.

Study Variables
Primary outcomes of the study were fracture etiology (accidental, indeterminate, and nonaccidental), fracture number (single or multiple), and presence or absence of fracture type associated with abuse (rib or metaphyseal). The diagnosis of nonaccidental injury was made by the child-protection team, independent of this study. The diagnosis used standard criteria, including history, physical examination findings, laboratory and imaging results, and consultation with other subspecialists, and was based on American Academy of Pediatrics guidelines. Children were classified as abused if 1 or more of the following criteria were met: (1) In addition to fracture, other unexplained nonskeletal injuries (cutaneous, intracranial, or abdominal) were identified; (2) no history of trauma was provided to explain a fracture in a nonambulatory child; and (3) no history of trauma was provided to explain multiple fractures in an ambulatory or nonambulatory child. Bruising or swelling over frac-
ture sites and small isolated extra-axial hemorrhages underlying skull fractures were not considered additional injuries.

Predictor variables were divided into 3 categories: (1) vitamin D status (25(OH)D); (2) biochemical bone status (parathyroid hormone [PTH], calcium, phosphorus, total alkaline phosphatase [ALKP]); and (3) radiologic bone status (evidence of rickets or demineralization on standard radiographs).

We used categories proposed by previous investigators to define vitamin D status. Serum 25(OH)D concentrations were stratified as follows: vitamin D deficient (<20 ng/mL; <50 nmol/L); vitamin D insufficient (≥20 < 30 ng/mL; ≥50 < 78 nmol/L); and vitamin D sufficient (≥30 ng/mL; ≥78 nmol/L).5,11 There is little consensus regarding the ideal 25(OH)D level.12,13,25 Although many experts agree that 30 ng/mL represents an optimal level for 25(OH)D,25,26 American Academy of Pediatrics references a threshold of 25 ng/mL.27 The Institute of Medicine suggests that 20 ng/mL represents the lower limit of normal.28 Elevated PTH was defined as a PTH more than 56 pg/mL. Hypocalcemia was defined as calcium less than 9.2 mg/dL in infants (younger than 1 year of age) and less than 8.7 mg/dL in children 1 year of age or older. Hypophosphatemia was defined as phosphorus less than 4.8 mg/dL in infants and less than 3.8 mg/dL in children 1 year of age or older. Elevated ALKP was defined as more than 345 units/L in infants and more than 320 units/L for children 1 year of age or older.

**Laboratory Measurements**

Serum 25(OH)D was measured using high-performance liquid chromatography coupled with tandem mass spectrometry; PTH was measured by a 2-site chemiluminescence immunoassay (Immulite 2000; Seimens Diagnostics Newark, DE). Serum calcium, phosphorus, and ALKP were measured by end-point assay in a multichannel analyzer (Fusion 5.1 Analyzer; Ortho Clinical Diagnostics, Rochester, NY). Samples were analyzed in multiple assays. Interassay coefficients of variation were 4.2% to 10.0% for 25(OH)D, 6.8% to 8.8% for PTH, 1.4% to 1.6% for calcium, 1.5% to 2.4% for phosphorus, and 2.1% to 2.4% for ALKP.

**Statistical Analysis**

Statistical analyses were conducted using Stata 11.0 software (Stata Press, College Station, TX). χ² tests were conducted to determine associations between each categorical outcome and the dichotomous categorical predictor variables of interest. Fisher’s exact test was used for comparisons in which any category had fewer than 10 subjects. Test for linear trend was used to test for associations between each categorical outcome and vitamin D deficiency, insufficiency, and sufficiency. Nonparametric Wilcoxon rank-sum test was used to test for associations between the categorical outcomes and age as a continuous variable because age was not normally distributed. Spearman rank correlation coefficient was calculated to determine the association of age and PTH. Logistic regression analysis was performed to further investigate the relationship between the presence of rib fractures and PTH while adjusting for age. Power analysis was conducted using PASS 2008 software (NCSS, Kaysville, Utah). For power analysis, the more conservative 2-degrees-of-freedom χ² test was used rather than the 1-degrees-of-freedom Cochran-Armitage test.

**RESULTS**

**Subject Characteristics**

Of 165 patients who met inclusion criteria, 47 (28%) were not enrolled (Table 1), principally because of hospital discharge before being approached by a study interviewer or refusal to consent to phlebotomy.

Nonparticipating eligible children were more likely than included subjects to be white (68% vs 49%; P = .002), to have accidental injuries (91% vs 60%; P < .001), to have single fractures (85% vs 69%; P = .03). There were no differences in age or insurance (P > .54) (Table 1).

Among 118 enrolled subjects, 58 (49%) were white and 43 (36%) were African American; 64 (54%) were publicly insured. Age ranged from 6 to 673 days with a median of 203 days. Gestational age in weeks at birth ranged from 24 to 41 with a median of 40; 15 (13%) subjects were extra or moderate preterm (<34 weeks’ gestational age), 14 (12%) were late preterm (≥34 < 37 weeks’ gestational age), and 89 (75%) were full term (≥37 weeks’ gestational age).

**Vitamin D Status**

A total of 8% of the children were vitamin D deficient (<20 ng/mL), 31% of children were vitamin D insufficient (≥20 < 30 ng/mL), and 61% were vitamin D sufficient (≥30 ng/mL). Subjects with vitamin D deficiency and insuffi-
ciency were more likely than subjects with vitamin D sufficiency to have hypocalcemia (33% vs 11% vs 3%; \( P = .002 \)) and elevated ALKP (33% vs 18% vs 10%; \( P = .05 \)). Children with lower 25(OH)D levels tended to have lower phosphorus (13% vs 9% vs 5%; \( P = .30 \)) and higher PTH (44% vs 8% vs 11%; \( P = .08 \)); however, these trends did not reach statistical significance. Radiographic demineralization was noted in none of the vitamin D–deficient subjects, in 5% of the vitamin D–insufficient subjects, and in 7% of the vitamin D–sufficient subjects (\( P = .44 \)). Among children with skeletal surveys (73%), none had radiographic rickets.

**Fracture Etiology**

Accidental injuries were diagnosed in 60% of children, nonaccidental injuries in 51%, and indeterminate injuries in 9%. Most indeterminate injuries were isolated fractures in the absence of an appropriate history but were consistent with the child’s development. The majority (78%) of children diagnosed with nonaccidental fractures had additional nonskeletal injuries (Table 2). Fractures also were characterized as abusive in subjects with multiple fractures in the absence of a history of trauma (16%) and in nonambulatory infants with isolated fractures in the absence of a history of trauma (5%).

Premature birth tended to be more common in children diagnosed as abused; however, this did not reach statistical significance; abuse was diagnosed in 30% of term, 42% of late preterm, and 53% of moderate or early preterm subjects (\( P = .06 \)). The prevalence of vitamin D deficiency and insufficiency did not differ between abused and nonabused children (5% vs 9% and 27% vs 34%; \( P = .32 \)) (Table 3). Similarly, children with nonaccidental injuries were not more likely than children with accidental injuries to have elevations of PTH (\( P = .07 \)) or ALKP (\( P = .77 \)).

**Fracture Number and Type**

The number of fractures ranged from 1 to 15; most children (69%) sustained a single fracture (Table 4). Skeletal surveys were performed in 73% of children. Multiple fractures were not more common in premature subjects: 33% of term, 14% of late preterm, and 40% of moderate or early preterm subjects had multiple fractures (\( P = .95 \)). The prevalence of vitamin D deficiency and insufficiency did not differ between subjects with single and multi-

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**TABLE 3** Characteristics of Abused and Nonabused Subjects (\( n = 108 \))

<table>
<thead>
<tr>
<th>Fracture Type</th>
<th>Accidental, ( n = 71 ), 60%</th>
<th>Nonaccidental, ( n = 37 ), 31%</th>
<th>( P )</th>
</tr>
</thead>
<tbody>
<tr>
<td>Number of fractures, ( n ) (%)</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Single</td>
<td>63 (89)</td>
<td>12 (32)</td>
<td>&lt;.001</td>
</tr>
<tr>
<td>Multiple</td>
<td>8 (11)</td>
<td>25 (68)</td>
<td></td>
</tr>
</tbody>
</table>

**TABLE 4** Characteristics of Subjects by Number of Fractures (\( n = 118 \))

<table>
<thead>
<tr>
<th>Fracture Type</th>
<th>Single, ( n = 81 ), 69%</th>
<th>Multiple, ( n = 37 ), 31%</th>
<th>( P )</th>
</tr>
</thead>
<tbody>
<tr>
<td>Injury classification, ( n ) (%)</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Accidental</td>
<td>63 (78)</td>
<td>8 (22)</td>
<td>&lt;.001</td>
</tr>
<tr>
<td>Nonaccidental</td>
<td>12 (15)</td>
<td>25 (68)</td>
<td></td>
</tr>
<tr>
<td>Indeterminate</td>
<td>6 (7)</td>
<td>4 (11)</td>
<td></td>
</tr>
<tr>
<td>25(OH)D, ( n ) (%)</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Deficient, ( \leq 20 ) ng/mL</td>
<td>5 (6)</td>
<td>4 (11)</td>
<td></td>
</tr>
<tr>
<td>Insufficient, ( \geq 20 &lt; 30 ) ng/mL</td>
<td>24 (30)</td>
<td>15 (35)</td>
<td>.24</td>
</tr>
<tr>
<td>Sufficient, ( \geq 30 ) ng/mL</td>
<td>52 (64)</td>
<td>20 (54)</td>
<td></td>
</tr>
<tr>
<td>Elevated PTH, ( n = 117 ), ( n ) (%)a</td>
<td>8 (10)</td>
<td>7 (19)</td>
<td>.24</td>
</tr>
<tr>
<td>Elevated ALKP, ( n = 113 ), ( n ) (%)b</td>
<td>8 (10)</td>
<td>8 (22)</td>
<td>.15</td>
</tr>
<tr>
<td>Rib fracture, ( n ) (%)</td>
<td>2 (2)</td>
<td>22 (58)</td>
<td>&lt;.001</td>
</tr>
<tr>
<td>Metaphyseal fracture, ( n ) (%)</td>
<td>3 (4)</td>
<td>11 (30)</td>
<td>&lt;.001</td>
</tr>
</tbody>
</table>

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\( ^a \) PTH more than 56 pg/mL.

\( ^b \) ALKP more than 345 units/L for those less than 12 months old; ALKP more than 320 units/L for more than or equal to 12 months but less than 24 months old.

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*a* Includes cutaneous (bite marks and bruises not directly overlying fractures), abusive head trauma (intracranial hemorrhage, excluding small extraaxial hemorrhages associated with a skull fracture as the presenting injury), or abdominal trauma.

*b* For 2 of the subjects in this group, there was a confession of abuse.

1. One subject was 3 months old with fussiness and was found to have a spiral fracture of left humerus; the other subject was 4 months old with fussiness and was found to have a mid shaft femur fracture.
Elevated PTH, 25(OH)D, preterm subjects (Pterm, and 33% of moderate or early present in 19% of term, 14% of late preterm subjects (Table 5). Rib fractures were more likely than children with multiple fractures to have elevated PTH or ALKP (all P > .11).

**Evaluation for Other Causes of Bone Fragility**

Two children with known osteogenesis imperfecta were excluded from the study. A total of 10 children were evaluated by the metabolic bone service and 2 had skin biopsies to evaluate possible osteogenesis imperfecta, both of which were normal. Although the association was not statistically significant, elevated ALKP tended to occur more commonly in early preterm (27%) than in late preterm (17%) and term (15%) subjects (P = .64). A total of 3 infants were born at less than 32 weeks’ gestational age and had abnormal ALKP, calcium, or phosphorus, raising concern for possible osteopathy of prematurity as the fracture etiology.²⁹ They did not, however, have radiographic demineralization. Two of these subjects had rib fractures that were categorized as accidental; the third subject had traumatic brain injury and a skull fracture and was diagnosed as abused.

Among the 8 children with fracture(s) and no additional injuries who were diagnosed as abused (Table 2), 6 were vitamin D sufficient and 2 were insufficient [25(OH)D levels of 27.2 ng/mL and 29.4 ng/mL]. All 8 subjects were born at more than 36 weeks’ gestational age. The metabolic physicians evaluated 5 of these subjects and determined that there was no indication of metabolic bone disease. Finally, even when excluding these subjects from the analysis, the abused children were no more likely than the nonabused children to have vitamin D deficiency (7% vs 8%) or insufficiency (28% vs 34%; P = .51).

**TABLE 5** Characteristics of Subjects With Rib or Metaphyseal Fractures (n = 118)

<table>
<thead>
<tr>
<th></th>
<th>Rib Fractures</th>
<th>Metaphyseal Fractures</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>Absent, n = 84</td>
<td>Present, n = 24</td>
</tr>
<tr>
<td>25(OH)D, n (%)</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Deficient, &lt;20 ng/mL</td>
<td>4 (5)</td>
<td>4 (17)</td>
</tr>
<tr>
<td>Insufficient, ≥20 &lt; 30 ng/mL</td>
<td>31 (33)</td>
<td>6 (25)</td>
</tr>
<tr>
<td>Sufficient, ≥30 ng/mL</td>
<td>58 (62)</td>
<td>14 (58)</td>
</tr>
<tr>
<td>Elevated PTH, n = 117, n (%)a</td>
<td>8 (9)</td>
<td>7 (29)</td>
</tr>
<tr>
<td>Elevated ALKP, n = 115, n (%)b</td>
<td>11 (12)</td>
<td>5 (21)</td>
</tr>
<tr>
<td>Radiologic abnormalities, n (%)c</td>
<td>5 (5)</td>
<td>2 (8)</td>
</tr>
</tbody>
</table>

FIGURE 1

*Age and PTH in young infants with and without rib fractures. CI indicates confidence interval.*

ple fractures. Similarly, children with single fractures were not more likely than children with multiple fractures to have elevations of PTH or ALKP (all P > .15).

A total of 20% of children had rib fractures (Table 5). Rib fractures were present in 19% of term, 14% of late preterm, and 33% of moderate or early preterm subjects (P = .32). Although 17% of children with rib fractures were vitamin D deficient compared with 5% of children without rib fractures, there was not a statistically significant association between vitamin D levels and the presence of rib fractures (P = .16). Children with rib fractures were more likely than children without rib fractures to have elevated PTH (P = .01). This finding could reflect the difference in age between these two groups (age ranged from 12 to 321 days, with a median of 110 days, in children with rib fractures compared with 6 to 673 days, with a median of 253 days, in those without rib fractures; P = .0001). Analysis of PTH values in all children showed an inverse relationship (P = .0005, r = −0.32) between age and PTH. There was not a statistically significant difference in PTH levels between infants with and without rib fractures after controlling for age in a logistic regression model (odds ratio: 2.6 95% confidence interval: 0.8–8.9; P = .12) (Fig 1). There were no associations between rib fractures and elevated ALKP or radiographic demineralization.

Metaphyseal fractures were identified in 12% of children (Table 5). Children with metaphyseal fractures were not more likely than children without these fractures to have vitamin D deficiency or insufficiency or elevations of PTH or ALKP (all P ≥ .11).
DISCUSSION

We found a high prevalence of vitamin D insufficiency among children younger than 2 years of age presenting with fractures to an urban pediatric hospital. A total of 39% had 25(OH)D levels below 30 ng/mL, but none had clinical or radiographic signs of rickets. Our results are similar to those of Gordon et al, who found that among 380 healthy 8- to 24-month-old children in Boston, 40% had 25(OH)D less than 30 ng/mL and 12% had 25(OH)D less than 20 ng/mL. Using the results of Gordon et al as a normative sample, we found no difference in the prevalence of vitamin D insufficiency in children with fractures compared with healthy children. Furthermore, we found that the incidence of vitamin D insufficiency was similar in children presenting with a single fracture compared with those with multiple fractures.

It has been hypothesized that low vitamin D levels render infants more susceptible to fractures, and vitamin D insufficiency has been offered as an alternative diagnosis in children with fracture types concerning for abuse. Our study did not find a statistically significant association between vitamin D status and the presence of rib or metaphyseal fractures. Moreover, children diagnosed as abused were not more likely to have suboptimal levels of 25(OH)D. Although children with rib fractures were more likely to have elevated PTH, this finding was no longer significant when adjusted for age. Our study had low power to detect a small difference in the frequency of vitamin D deficiency between children with and without rib fractures. Currently, there is no standard definition of optimal vitamin D status in children. One commonly cited definition of suboptimal 25(OH)D in adults is the concentration at which PTH increases, reflecting the physiologic response to decreased calcium absorption from the small intestine. Rickets is a growth-plate disorder that results from a primary or secondary metabolic perturbation that leads to hypophosphatemia. The associated mineralization defect further reduces bone strength. Although vitamin D deficiency is the most common cause of rickets, current scientific evidence does not support the interpretation of a suboptimal serum level of vitamin D as evidence of rickets, particularly in the absence of clinical or radiological signs of rickets. Rickets is not a subclinical diagnosis. In our study, 7 of 46 subjects with 25(OH)D less than 30 ng/mL had elevated PTH levels. Although hypocalcemia and elevated ALP were more common in subjects with low vitamin D levels, no subjects had radiologic rickets, and low vitamin D levels were not associated with radiologic demineralization. This may be because film-based radiographs are insensitive and digital radiographs are unsuitable for determination of skeletal mineralization and because the interreporter reliability between radiologists determining demineralization on radiographs is poor.

The absence of a control group without fractures was the primary limitation of the study. Although imperfect, comparing our subjects with fractures to previously reported healthy children revealed no differences in vitamin D status between the two groups. Moreover, analyzing patients with single and multiple fractures was informative. The power of the study to detect small differences was limited by the sample size. In comparing vitamin D levels among children with abusive fractures to those with accidental fractures, the sample size of 108 achieved an 80% power to detect a moderate effect size (W) of 0.29 using a 2-degrees-of-freedom \( \chi^2 \) test with a significance of 0.05. The study had low power to detect smaller effect sizes. Skeletal surveys were not obtained in 27% of the subjects, some of whom may have had multiple fractures or rib or metaphyseal fractures. However, we reviewed subjects who received skeletal surveys and found no difference in vitamin D levels between children with single versus multiple fractures and no association between rib or metaphyseal fractures and vitamin D status. Finally, we recognize that any study that attempts to evaluate a child abuse mimic can be contaminated by ascertainment bias that leads to the incorrect attribution of injuries to abuse rather than the disease entity under investigation. In this study, however, 78% of abused children had multiorgan injuries that could not be attributed to poor vitamin D status (Table 2). Of the children diagnosed as abused in the setting of unexplained fractures alone, 75% were vitamin D sufficient and the remaining 2 children had very minor decreases in their 25(OH)D levels.

CONCLUSIONS

Vitamin D insufficiency is common among children younger than 2 years of age with fractures, but suboptimal vitamin D status is not more common in children with unexplained fractures than in previously studied healthy children. Suboptimal vitamin D status has been proposed as an alternative explanation for nonaccidental fractures in young children. In our study, however, suboptimal vitamin D status was not associated with a diagnosis of abuse or the presence of multiple fractures or rib or metaphyseal fractures, suggesting that a strong association between vitamin D insufficiency and fracture types related to abuse is unlikely. Our findings indicate that a low 25(OH)D should not discourage consideration of abuse when a child presents with unexplained fractures.
REFERENCES

30. Demay MB, Sabbagh Y, Carpenter TO. Calcium and vitamin D: what is known about the effects on growing bone. Pediatrics. 2007;119:S141–S144
ERRATA


On page 838, under the Fracture Etiology heading, paragraph 2, line 10, this reads: “and nonabused children (5% vs 9%).” This should have read: “and nonabused children (5% vs 8%).”

On page 839, in Table 5, under the Rib Fractures, Absent, n/H1100594 column heading, the first entry reads: “4(5).” This should have read: “5(5).”

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On page 562, in Table 6, the 6th entry under the Control column heading reads: “Not reported” and the 6th entry under the Other Effects/Comments column heading reads “PCE associated with long-term changes in brain composition, which were related to performance on neuropsychological tests; limited sample size and covariate control.” The entry under the Control column should have read: “Groups matched on gender, handedness, IQ, prematurity, age, race, and caregiver stability. Statistical control on A, M, T”; and the entry under the Comments column should have read: “PCE associated with long-term changes in brain composition, which were related to performance on neuropsychological tests; limited sample size.”

On page 563, the final sentence reads: “However, most studies have lacked covariate control, raising concerns about potential confounds.” The sentence should have read: “Although many studies matched comparison groups, sample sizes were small and often lacked statistical control, emphasizing the need for replication.” These changes do not alter the findings reported in the original manuscript.

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