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REVIEW

The etiology and significance of fractures in infants and young children: a critical multidisciplinary review

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Abstract This paper addresses significant misconceptions regarding the etiology of fractures in infants and young children in cases of suspected child abuse. This consensus statement, supported by the Child Abuse Committee and endorsed by the Board of Directors of the Society for Pediatric Radiology, synthesizes the relevant scientific data distinguishing clinical, radiologic and laboratory findings of metabolic disease from findings in abusive injury. This paper discusses medically established epidemiology and etiologies of childhood fractures in infants and young children. The authors also review the body of evidence on the role of vitamin D in bone health and the relationship between vitamin D and fractures. Finally, the authors discuss how courts should properly assess, use, and limit medical evidence and medical opinion testimony in criminal and civil child abuse cases to accomplish optimal care and protection of the children in these cases.

Keywords Child abuse · Children · Fractures · Infants · Metabolic bone disease · Non-accidental trauma · Radiography · Rickets · Vitamin D

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Introduction

This paper addresses significant misconceptions regarding the etiology of fractures in infants and young children. These misconceptions — propagated by medical witnesses and the news media in child abuse legal cases — concern the prevalence of metabolic bone disease, particularly rickets, and the appropriate medical workup of children suspected of being physically abused [1–5]. This consensus statement, supported by the Child Abuse Committee and endorsed by the Board of Directors of the Society for Pediatric Radiology, reviews and synthesizes relevant scientific data. This statement is derived from an empirical assessment of the quality and accuracy of the medical literature and addresses the threshold question of when such literature is generally medically accepted in the pediatric health care community. This review of the medical literature also considers the court admissibility and the reliability of expert medical opinions based on such literature.

The contributing board-certified physician authors each have one or more pediatric imaging subspecialty board certifications from the American Board of Radiology or the American Board of Pediatrics (both members of the American Board of Medical Specialties). Additionally, all authors have 10–40 years of individual clinical experience diagnosing and treating children. The non-physician author is a law professor with nearly 2 decades of experience researching and writing on the appropriate use of child abuse evidence in court. We address the following questions:

(1) What does the medical literature establish regarding the epidemiology and etiologies of childhood fractures in infants and young children?
(2) When fractures are present, what factors suggest seeking additional screening for physical abuse/non-accidental trauma (NAT)?
(3) What medical, laboratory, and imaging studies assist in screening for physical abuse/NAT?
(4) What do large, randomized, and/or controlled studies establish about the relationship between serum vitamin D value and bone health?
(5) What laboratory and imaging studies assist in diagnosing rickets?
(6) What is the relationship between serum vitamin D levels and:
    (a) rickets?
    (b) skull fractures, rib fractures, or any other fractures?
    (c) retinal hemorrhages?
    (d) subdural hematomas?
(7) What is the role of serum vitamin D levels and serum calcium in maternal-fetal-neonatal bone health?
(8) Does the medical literature establish that some pediatric fractures are highly specific for physical abuse/NAT?
(9) How should courts assess, use and limit medical evidence and medical opinion testimony in criminal and civil child abuse cases?

Epidemiology and etiology of childhood fractures

The reported cases

The annual incidence of confirmed maltreatment of children has been reported as 1–2% of the U.S. child population, and physical abuse comprises one-fourth of these cases [6]. The rate of physical abuse is highest among infants and children younger than 2 years, in whom fractures are the second most common injury after bruises. It has been reported that 1–4 per 1,000 children younger than 2 years are treated annually for fractures from all causes, with 10% of these on average evaluated for possible physical abuse [7–10].

Fractures from child abuse are much more common than fractures caused by underlying medical conditions such as rickets and osteogenesis imperfecta. Given the rarity of these medical conditions, it has been estimated that children younger than 3 years are nearly 100 times more likely to have a fracture caused by abuse than a fracture caused by a metabolic abnormality such as rickets and approximately 20 times more likely to have a fracture caused by abuse than by osteogenesis imperfecta [10, 11].

Under-reporting of abuse

The incidence of abusive fractures is likely greater than these data suggest. In young children, 20% of fractures caused by abuse may be incorrectly attributed to other causes [12–14]. For example, one study found that of 100 children younger than 3 years who presented to an emergency department with long-bone fractures, 31 had indicators suggestive of abuse but only one was referred to child protective services for additional assessment [15]. In addition, the use of skeletal surveys to detect occult fractures in young children who present with non-skeletal injuries concerning for abuse is variable, suggesting that occult skeletal injuries are missed in some children [16]. Equally important, incorrectly diagnosing abuse in a child with non-inflicted fractures can also have serious consequences for children and families and can delay the diagnosis of medical conditions that require treatment. Finally, physicians have a legal mandate to report suspected child abuse, and national U.S. statistics indicate that only about 20% of reported cases of suspected abuse are eventually substantiated as abuse by investigators, either because reported injuries were found to be accidental or related to a medical condition, or because the evidence required to substantiate the report was not available [17].

Medical evaluation

The evaluation for suspected abusive fractures requires an extensive multidisciplinary assessment that includes consideration and exclusion of other possible causes. The American Academy of Pediatrics has reviewed how the medical evaluation should proceed [18]. It is essential to obtain a detailed history to determine how an injury occurred, with details about the child's pre-injury activity and position and final post-injury position and location (Table 1). In addition to the history of present illness, the physician must consider the medical history, the age of the child, the developmental status of the child, the location and type of fracture, the reported mechanism of injury causing the particular type of fracture, and the presence of other injuries.

The child should have a complete physical examination, and the growth chart should be reviewed. Most children with long-bone fractures have swelling, pain or other signs or symptoms, such as decreased use of the extremity, but some have minimal external signs of injury [19]. Most children with fractures (58–91%) do not have bruising associated with the fracture; the presence or absence of such bruising therefore does not help to determine which fractures are caused by child abuse [20–23]. The child should be examined for other injuries that could be caused by child abuse in addition to signs of medical conditions associated with metabolic bone disease or mimics of abuse (Table 2) [24].

Laboratory evaluation

The history and physical examination determines the type of laboratory workup (Table 3). Serum calcium, phosphorus and alkaline phosphatase should be evaluated in children with fractures suspicious for abuse. It is prudent to obtain a vitamin D level and parathyroid hormone if there is evidence of demineralization or other findings suggestive of rickets [18]. If osteogenesis imperfecta is suspected, sequence analysis of the *COL1A1* and *COL1A2* genes may be more sensitive than biochemical tests of type I collagen and may identify a mutation to guide testing of other family members [18]. Consultation with subspecialists in pediatric endocrinology, genetics, orthopedics and bone mineral metabolism may be helpful in deciding which children to test and which tests to order.

Vitamin D level

Vitamin D level is a laboratory value and not a diagnosis of disease, and metabolic bone disease cannot be accurately diagnosed solely on the basis of a vitamin D level. Considerable scientific controversy surrounds the amount of vitamin D needed by humans [25–29]. This controversy is largely related to proposed extra-skeletal effects of vitamin D such as autoimmune disease, diabetes and cardiovascular disease [30]. Vitamin D is involved in the regulation of up to 2,000 genes, which suggests it has a role in many physiological processes in addition to maintenance of calcium concentrations. Although 25-hydroxyvitamin D levels of ≥20 ng/ml have long

Table 1 Important risk factors in the medical history for abuse and for other specific medical etiologies of fracture in children younger than 2 years

Feature	Risk factor
Age	Infants and children <2 years
Developmental abilities	Non-ambulating child, injury developmentally inconsistent
Reported history of injury	Inconsistency among child, parent, witness; vague description or absence of any history of injury
Timeliness for seeking medical help	Delay in seeking medical care
Maternal/neonatal history	Mother's medical history and pregnancy, total parenteral nutrition, hepatobiliary disease, or hypercalciuria
Medications	Diet and medications (steroids, diuretics, anti-epileptics, heparin, warfarin, cyclosporine, glucocorticoids, medroxyprogesterone acetate, cancer drugs, and thyroid hormone) that can predispose to fractures
Other medical diseases	Renal insufficiency, metabolic acidosis, malabsorption, cerebral palsy, neuromuscular disorders, genetic diseases
Prior injuries	Prior bruises, injuries, fractures, prior hospitalizations
Family history	Multiple fractures, early onset hearing loss, abnormally developed dentition, blue sclera, short stature
Psychosocial history	Others in the home, intimate-partner violence, substance abuse, mental illness, previous involvement with child protective services and/or law enforcement

Table 2 Important risk factors found on physical examination for abuse and for specific medical etiologies of fractures

Element	Concerning risk factor
Growth parameters	Abnormal height or weight may suggest neglect or endocrine or metabolic disorders
Bruises	Bruising in a child who is not yet cruising, or bruising in unusual locations such as the ears, neck, trunk, or genitals, should raise suspicion for child abuse
Eyes	Retinal hemorrhages suggest abuse. Blue sclera are seen in certain types of osteogenesis imperfecta
Neurological	Cerebral palsy and other neuromuscular disorders are associated with osteopenia and abuse
Hair	Sparse or kinky hair is associated with Menkes disease
Teeth	Dentinogenesis imperfecta is identified in older children with osteogenesis imperfecta

been regarded as adequate to prevent rickets and osteomalacia, proposed extra-skeletal effects have led many physicians to recommend higher vitamin D levels, designating <20 ng/ml as deficiency, ≥30 ng/ml as sufficiency, and 20–29 ng/ml as insufficiency [31, 32].

As a result of this controversy, "insufficiency" has been used to indicate levels that are not associated with rickets or osteomalacia but are considered by some not to provide the full extra-skeletal benefits of vitamin D. Although many observational studies point to extra-skeletal benefits of vitamin D, no randomized clinical trial has demonstrated that vitamin D supplementation prevents malignancy, autoimmune disease, diabetes and cardiovascular disease [33]. Primarily for this reason, a 2011 report by the Institute of Medicine of the National Academy of Science concluded that insufficient evidence of extra-skeletal effects precluded the use of these effects to establish recommendations for human vitamin D requirements [34]. The Institute of Medicine concluded that ≥20 ng/ml is indicative of vitamin D sufficiency. The institute further recognized that a normal distribution for vitamin D requirements exists and that lower levels would be sufficient for many people. The Institute of Medicine selected ≥20 ng/ml as a level that satisfies the vitamin D requirement for the vast majority of the population, with 16 ng/ml satisfying the requirement for half of the population [34]. Of note, before the 2011 Institute of Medicine report, there was no established precedent for suggesting that children require levels greater than 20 ng/ml; guidelines of the Committee on Nutrition of the American Academy of Pediatrics, the Pediatric Endocrine Society and the European equivalents of these organizations have never required such high levels of vitamin D [35–37].

One study of 310 children reported that 15.6% had serum 25-hydroxyvitamin D concentrations less than 20 ng/ml and 2.1% had less than 10 ng/ml [38]. However, none had elevated serum parathyroid levels or clinical manifestations related to vitamin D deficiency. The correlation between serum parathyroid hormone and 25-hydroxyvitamin D was weak, and children with levels of 20–29 ng/ml of 25-hydroxyvitamin D did not have clinical disease or elevated levels of serum parathyroid hormone.

Further data have demonstrated no correlation between low vitamin D levels and the likelihood of either abusive or accidental fractures in children [39, 40]. These data establish that the prevalence of low vitamin D levels in children with fractures is the same as in populations of well babies with no fractures [41]. No study has demonstrated that low serum vitamin D level without radiographic bone changes increases susceptibility to bone fractures.

In summary, although some controversy exists regarding human vitamin D requirements, particularly regarding potential extra-skeletal effects, significant metabolic bone disease is not associated with vitamin D levels of 20 ng/ml or greater.

Table 3 Laboratory evaluation in children with fractures of unknown etiology

Etiology	Laboratory tests
Bone metabolism	Serum calcium, phosphorus, alkaline phosphatase, 25-hydroxyvitamin D
Renal/endocrine	Parathyroid hormone and 25-hydroxyvitamin D, as well as urinary calcium creatinine clearance
Micronutrients	Serum copper, vitamin C and ceruloplasmin concentrations should be considered if the child is at risk of scurvy or copper deficiency and has radiographic findings that include metaphyseal abnormalities
Collagen	Sequence analysis of the *COL1A1* and *COL1A2* genes and additional genes
Abdomen	Urinalysis, liver function studies, amylase, lipase

Specifically, levels of 20–29 ng/ml, currently designated as "insufficient," are not associated with significant bone changes and do not result in skeletal fragility [42–46].

Rickets

Rickets and osteomalacia are skeletal disorders caused by calcium and phosphate insufficiency, most often from true vitamin D deficiency, abnormal vitamin D metabolism (including liver and kidney abnormalities) or primary hypophosphatemic (phosphate wasting) conditions. Rickets is characterized by deficient and disordered endochondral ossification and mineralization of cartilage and osteoid at the physes in children, whereas osteomalacia refers to deficient mineralization for other types of bone formation such as membranous bone and normal remodeling [43]. A broader definition of vitamin D deficiency-related skeletal disease uses "hypovitaminosis D osteopathy" to include rickets, osteomalacia and secondary hyperparathyroidism, which is an adaptive response to hypocalcemia caused by vitamin D deficiency [47].

Manifestations

The radiologic manifestations of rickets in growing children include loss of the zone of provisional calcification with apparent physeal widening [42]. Unossified cartilaginous rests cause a frayed appearance of the subphyseal metaphysis. In addition to fraying, there is flaring, cupping and splaying of metaphyses as well as demineralization. These changes tend to appear in areas of most rapid growth, such as the distal radius, distal femur and proximal tibia. These findings are symmetrical and systemic; although faster-growing bones are affected to a greater extent, all bones are involved.

Three stages of vitamin D deficiency and bone health have been described by Fraser et al. [45], and the clinical, laboratory and radiographic findings are different in each stage (Table 4). Stage 1 is clinically silent and the child is asymptomatic, with no evidence of increased bone fragility or increased susceptibility to fractures. The only abnormality is a biochemical one, with the child having low calcium (hypocalcemia). Therefore, fractures with normal-appearing bone should not be attributed to fragility from rickets. In stage 2, hyperparathyroidism develops in response to the low calcium by increasing resorption of calcium from bone. In this stage, bone changes are evident and fractures can occur. In stage 3, the vitamin D deficiency is severe and there is increasing rickets and hyperparathyroidism.

In a study of 45 infants and toddlers younger than 24 months with proven rickets, fractures were present only in those with radiographically severe rickets; 17.5% had fractures in this cohort, but no fractures were found in infants who were not rolling over, crawling, cruising or walking [48]. Furthermore, none of the infants and toddlers with rickets and fractures had classic metaphyseal lesions or skull fractures.

Fractures in rickets

Fractures in rickets are noted in association with abnormal bone radiographs, which demonstrate osteopenia and metaphyseal cupping, fraying and splaying, and in general are not those most specifically associated with child abuse (posterior rib fractures and classic metaphyseal lesions). Examples of fractures found in children with rickets include transverse long-bone fractures, metaphyseal fractures (associated with abnormal metaphyses and not classic metaphyseal lesions), and anterior-lateral rib fractures (with abnormal ribs) [48]. Rickets is not associated with subdural hematoma or retinal hemorrhage [49]. These lesions are most commonly found in cases of non-accidental traumatic brain injury (also referred to as abusive head trauma, or AHT) [50]. Rickets is not fatal except in the most severe instances when the child's immune system is impaired and the terminal event is pneumonia, seizures or cardiac dysfunction [30, 36, 51].

Consequently, fractures do not occur in Fraser stage 1 [45], when hypocalcemia is the only manifestation. Fractures can occur in mobile full-term infants or toddlers with Fraser stages 2 or 3 disease, all of whom also have characteristic radiographic changes. Infants who are not rolling over, crawling, cruising or walking and who do not have Fraser stages 2 or 3 disease do not have fractures as a result of their disease [48].

Table 4 Findings necessary to diagnose vitamin D deficiency as it is related to bone health (from Fraser et al. [45])

Stage	25(OH) Vit D	Calcium	PTH	Alkaline phosphatase[a]	Radiographs	Bone fragility/ fractures	Urine calcium
1	Deficient	Low	Normal	Normal	Normal	Normal/no	Low
2	Deficient	Normal/low	Elevated	Elevated	Mild rickets and/or hyperparathyroidism	Abnormal/yes	Low
3	Deficient	Low	Elevated	Elevated	Severe rickets and/or hyperparathyroidism	Abnormal/yes	Low

OH hydroxy, *PTH* parathyroid hormone, *Vit* vitamin

[a] Appropriate for age correction

Mineral metabolism in the fetus and neonate

Mineral metabolism in developing fetuses is different from that in newborns and older infants because the placenta meets the fetal need for minerals by actively transporting calcium, phosphorus and magnesium from the maternal circulation. Moreover, the placental transport mechanisms preserve adequate levels of these minerals in the fetus even when maternal serum concentrations are substantially deficient. The fetus maintains higher mineral concentrations than the mother and normal adults, and such high levels appear necessary for normal skeletal formation and mineralization [52]. Although fetal bone and mineral metabolism is critically dependent on parathyroid hormone (PTH) and PTH-related protein (PTHrP), these processes do not require vitamin D or its active metabolite, calcitriol [52].

Fetal circulation contains high levels of PTHrP and low levels of PTH and calcitriol. Postnatally, the loss of the placenta leads to a rapid transition in mineral homeostasis in newborns, with a rapid fall in serum calcium levels and a corresponding increase in serum phosphorus levels. These changes stimulate secretion of PTH from the parathyroid glands, which leads to increased serum calcitriol and calcium and decreased serum phosphorus. Nevertheless, calcium absorption in newborns is principally passive and facilitated by high levels of lactose present in human milk. As newborns mature, vitamin D (calcitriol)-dependent active transport of calcium becomes the dominant mechanism for intestinal absorption. However in preterm infants, intestinal calcium absorption is completely passive and even unresponsive to endogenous or administered calcitriol [53].

Rickets of prematurity is common in babies of fewer than 28 weeks' gestation and who weigh less than 1.5 kg (3.3 lbs) [54]. This is because 80% of bone mineral accumulation occurs during the third trimester. Therefore metabolic bone disease of prematurity is usually caused by hypophosphatemia that results from the inability to provide these infants with adequate amounts of phosphorus, rather than from a defect in vitamin D homeostasis [55]. The radiographic findings in metabolic bone disease of prematurity are generalized osteopenia and fractures with classic rachitic changes that may not be apparent if the infant is growing very slowly [56]. The diagnosis is based on the history and radiographic findings.

Biochemical vitamin D deficiency in neonates is more common than objective evidence of bone disease based on histology, radiology or clinical signs, such as craniotabes (thin or soft skull) or rachitic rosaries (expansion of the anterior ends of the ribs). Some reports suggest that mild features of rickets, principally craniotabes, may be present at birth in cases of severe maternal vitamin D deficiency. The specificity of craniotabes as a diagnosis of rickets is questionable [57–59].

In fact, congenital rickets is exceedingly rare. The incidence in a review of 337.68 million people (inclusive of adults) in India over 42 years revealed only 3 cases of congenital rickets [60]. Sporadic case reports and small series describe infants with rickets at birth or shortly thereafter, but these infants had markedly abnormal radiologic and biochemical features. The mothers of these newborns had severe medical problems such as untreated renal failure or severe osteomalacia and hypocalcemia with serum levels of 25-hydroxyvitamin D that were less than 10 ng/ml [61–64]. Similarly, maternal hypocalcemia caused by hypoparathyroidism or pseudohypoparathyroidism has been associated with intrauterine fetal hyperparathyroidism, skeletal demineralization, intrauterine fractures, and bowing of the long bones [64]. This is compatible with reduced placental calcium transfer that prompts secondary hyperparathyroidism to develop in the fetus.

There is compelling evidence that fetal vitamin D deficiency per se has little adverse effect on the neonatal skeleton. This evidence can be found in the extreme examples of children who have genetic defects that result in vitamin-D-dependent rickets. Even children with these defects have normal bones with neither fractures nor rickets at birth [65].

Fractures in child abuse

Fractures of every type and location occur in abused children, and no fracture is absolutely diagnostic of abuse. Thus a comprehensive team approach to the evaluation with appropriate imaging is crucial [15, 66]. Any fracture is highly suspicious of abuse in the setting of an absent, inappropriate or changing history. However, certain fractures and distributions of fractures are seen almost exclusively in abused children [18, 67–69]. Skeletal surveys are crucial in the evaluation of young children suspected of being abused [24, 70, 71]. When highly specific fractures are identified, particularly without an appropriate traumatic explanation and in the absence of metabolic disease, child abuse can be diagnosed based upon well-founded, reproducible scientific studies [67, 72, 73]. Our understanding of these specific fractures comes from decades of research performed independently by many investigators [18, 67–70, 74].

Multiple fractures at various stages of healing are highly specific for child abuse when there is no underlying metabolic abnormality or skeletal dysplasia. The distribution of fractures can be diagnostic of abuse. Rib fractures, particularly posterior rib fractures, in young children are highly specific for abuse [18, 67, 72]. Acute rib fractures are not always easily detected radiographically (or clinically) and thus follow-up skeletal survey or skeletal scintigraphic examination might provide corroborative evidence of these fractures [75, 76]. A targeted repeat skeletal survey can be especially valuable when there is a question of a fracture [77].

Because of the relative plasticity of the young child's ribcage, rib fractures are uncommon outside of child abuse. A 2003 study demonstrated a positive predictive value of rib fractures for child abuse of 95%, which increased to 100% when accounting for the clinical history [78].

Another fracture strongly associated with child abuse is the classic metaphyseal lesion, which is also known as a "bucket-handle" or "corner" fracture [79]. These fractures have long been recognized as highly specific for child abuse, but not until 1986 was the underlying pathology understood [80, 81]. A recent animal study suggests that these lesions are more properly described as classic metaphyseal fractures given their histopathology [82]. Metaphyseal abnormalities can be seen in other diseases but are distinct from the classic metaphyseal lesion and can be distinguished from them by additional findings and signs [83, 84].

Fractures of the scapula, sternum and spinous process are rare, but when found without an appropriate traumatic history they are highly specific for child abuse [85]. There is extensive evidence that multiple healing fractures in various stages, rib fractures in young children, classic metaphyseal lesions, scapula fractures, sternum fractures and spinous process fractures are highly specific for child abuse; this evidence includes scientific studies with large numbers of subjects and reproducible results [10, 18, 67, 85]. Fracture findings should be combined with other organ system injury findings, psychosocial and medical evaluation, and laboratory results to form the basis of the diagnosis of child abuse.

Societal ramifications of child abuse

Child abuse is a significant public health problem in the United States. According to child protective services agencies, more than 679,000 children were victims of maltreatment in 2013 in the United States, and more than 1,400 of those children died from abuse and neglect [86]. Non-accidental trauma is the most common cause of multiple fractures in young children [10, 18, 67]. Undiagnosed child abuse and neglect can lead to chronic abuse and increased morbidity and mortality [87–89]. The long-term impact of child abuse is substantial and complex. In addition to overt physical injuries, child maltreatment causes stress that can disrupt early brain development [90]. Psychological support is needed in addition to medical treatment to prevent the enduring harms of childhood abuse, and such support cannot be provided if abuse is erroneously missed by unsuspecting physicians, misinterpreted by child protective services agents, or dismissed by uninformed decision-makers in family or criminal courts. Physical abuse in infancy can result in a repeated pattern of abuse over generations [91].

The financial costs for victims and society are substantial. A recent Centers for Disease Control study showed that the total estimated cost associated with just 1 year of new confirmed cases of child maltreatment is $124 billion [92]. This and similar studies indicate the importance of not only preventing but also treating the long-term effects of child abuse to reduce the costs to society and families. While the financial costs are significant, the cost in human suffering is immeasurable.

Part of the financial burden is tied to the need to research cases of suspected abuse carefully. The major goal of the hospital-based child protection teams is to identify abuse to assist in the protection of the child. For this reason, these teams conduct medical evaluation so that other potential diagnoses can be excluded. The stakes are high in these decisions: if a case of abuse is missed, the possibility of further injury or death is substantial; similarly, if an erroneous finding of abuse is carried through the legal process, the result is irreparable damage to the child and the family [92].

The role of the court

The court is not an appropriate forum for the presentation of new or unsubstantiated theories of causation of disease. Rather, the court must ensure the accuracy of legal decisions. However, more and more highly sophisticated medical and scientific concepts are being introduced into the courtroom, placing the courts in the difficult circumstance of legitimizing scientific concepts [93]. As a preliminary matter, courts must understand the basic likelihood of competing causal explanations. In addition, judges and juries must evaluate the quality of the evidence. This task becomes increasingly complicated when expert witnesses propose unproven scientific hypotheses and image interpretation with greater certainty and greater acceptance than medical guidelines support.

Judges now must note the qualitative distinction between original well-designed studies published in well-respected peer-reviewed publications versus commentaries/letters. Whenever medical information shapes or determines a legal outcome, the type (peer review vs. non-peer review) and reputation of the journal, the author's background and prior publications, the research method and the cited references must be carefully scrutinized. The references must reinforce the authors' findings, and these references must be accepted by general medical practice and include other authors. Similarly, courts must scrutinize all potential experts to determine whether their opinions are based on the experience of day-to-day treatment and diagnosis of children and infants.

Courts can avoid promulgating unsubstantiated and unsafe theories and opinions by relying on medical and ethical guidelines from the American College of Radiology, the Society for Pediatric Radiology and the American Academy of Pediatrics [18, 66, 71, 94, 95]. As an example, the Committee on Child Abuse of the Society for Pediatric Radiology helped refute the legitimacy of temporary brittle bone disease (temporary

fragile bone disease), a disease that has not been proved to exist [96].

Conclusion

A major misrepresentation concerning the etiology of fractures in young children is the possibility of metabolic bone disease such as rickets predisposing children to fractures, including those known to be highly specific for child abuse. In this communication, we have emphasized the need to diagnose rickets by objective findings. To this end, the following concepts are supported by the preponderance of medical literature and highly respected professional committees and are generally accepted in the medical community:

(1) Vitamin D levels do not denote bone disease. The category of "insufficient" (greater than 20 ng/ml to less than 30 ng/ml) is not a valid threshold for bone health [34].
(2) Rickets is a metabolic bone disease that must be diagnosed by objective historical, laboratory and radiologic findings. When hypocalcemia is present but there are no radiographic findings of rickets, there is no evidence of bone fragility and increased susceptibility to fractures.
(3) Fetal and neonatal bone health is not correlated with maternal vitamin D levels.
(4) Congenital rickets is a rare phenomenon and has only been seen in newborns born to severely impaired mothers (e.g., renal failure, placental insufficiency). In such cases, there are always radiographic changes in the newborn indicative of poor bone health.
(5) Rickets is not associated with retinal hemorrhages or subdural hematomas, which occur with abuse.
(6) The fractures with high specificity for child abuse — classic metaphyseal lesions and posterior rib fractures — are not sequelae of rickets.
(7) Death is extremely rare in childhood rickets and is usually caused by superimposed infection in a malnourished child.
(8) Classic metaphyseal lesions (corner fractures) are not controversial fractures; they have validated medical literature support in correlating histopathological studies as well as animal studies.

Child abuse is an important issue in our society, and it is a solemn responsibility of the court to protect the child. The determination of whether fractures are caused by abuse should be based on an extensive evaluation and generally accepted procedures as suggested by established professional societies such as the Society for Pediatric Radiology, the American College of Radiology and the American Academy of Pediatrics. Decisions should be based on established scientific evidence and not unproven theories. Although it is important to question the literature and to test new hypotheses with scientific rigor, the evaluation of medical findings must be done with proper scientific technique. If this technique is lacking, the subsequent propositions cannot be used to adjudicate the findings in a court of law. Guidelines for the workup of suspected abuse are in place for a reason [18, 66, 71]: They are based on well-founded studies to provide guidance in the appropriate care of children.

On the Society for Pediatric Radiology website, a comprehensive list of reference articles can be found on the Child Abuse Committee webpage: http://www.pedrad.org/Specialties/Child-Abuse-Imaging.

Compliance with ethical standards

Conflicts of interest Dr. Christian and Dr. Palusci provide medico-legal expert work in child abuse cases. All other authors have no disclosures.

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