



CLINICAL REPORT

Evaluating Children With Fractures for Child Physical Abuse

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KEY WORD

fractures

ABBREVIATIONS

CML—classic metaphyseal lesions
CPR—cardiopulmonary resuscitation
CT—computed tomography
OI—osteogenesis imperfect

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abstract

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Fractures are common injuries caused by child abuse. Although the consequences of failing to diagnose an abusive injury in a child can be grave, incorrectly diagnosing child abuse in a child whose fractures have another etiology can be distressing for a family. The aim of this report is to review recent advances in the understanding of fracture specificity, the mechanism of fractures, and other medical diseases that predispose to fractures in infants and children. This clinical report will aid physicians in developing an evidence-based differential diagnosis and performing the appropriate evaluation when assessing a child with fractures. *Pediatrics* 2014;133:e477–e489

INTRODUCTION

Fractures are the second most common injury caused by child physical abuse; bruises are the most common injury.¹ Failure to identify an injury caused by child abuse and to intervene appropriately may place a child at risk for further abuse, with potentially permanent consequences for the child.^{2–4} Physical abuse may not be considered in the physician's differential diagnosis of childhood injury because the caregiver may have intentionally altered the history to conceal the abuse.⁵ As a result, when fractures are initially evaluated, a diagnosis of child abuse may be missed.⁵ In children younger than 3 years, as many as 20% of fractures caused by abuse may be misdiagnosed initially as noninflicted or as attributable to other causes.³ In addition, fractures may be missed because radiography is performed before changes are obvious or the radiographic images are misread or misinterpreted.² However, incorrectly diagnosing physical abuse in a child with noninflicted fractures has serious consequences for the child and family. To identify child abuse as the cause of fractures, the physician must take into consideration the history, the age of the child, the location and type of fracture, the mechanism that causes the particular type of fracture, and the presence of other injuries while also considering other possible causes.

DIFFERENTIAL DIAGNOSIS OF FRACTURES

Trauma: Child Abuse Versus Noninflicted Injuries

Fractures are a common childhood injury and account for between 8% and 12% of all pediatric injuries.^{6–8} In infants and toddlers, physical

abuse is the cause of 12% to 20% of fractures.⁹ Although unintentional fractures are much more common than fractures caused by child abuse, the physician needs to remain aware of the possibility of inflicted injury. Although some fracture types are highly suggestive of physical abuse, no pattern can exclude child abuse.^{10,11} Specifically, it is important to recognize that any fracture, even fractures that are commonly noninflicted injuries, can be caused by child abuse. Certain details that can help the physician determine whether a fracture was caused by abuse rather than unintentional injury include the history, the child's age and developmental stage, the type and location of the fracture, the age of the fracture, and an understanding of the mechanism that causes the particular type of fracture. The presence of multiple fractures, fractures of different ages or stages of healing, delay in obtaining medical treatment, and the presence of other injuries suspicious for abuse (eg, coexisting injuries to the skin, internal organs, or central nervous system) should alert the physician to possible child abuse.

Child's Age and Development

The physician should consider the child's age and level of development. Approximately 80% of all fractures caused by child abuse occur in children younger than 18 months,¹² and approximately one-quarter of fractures in children younger than 1 year are caused by child abuse.^{1,9,13–15} Physical abuse is more likely to be the cause of femoral fractures and humeral fractures in children who are not yet walking compared with children who are ambulatory,^{15–18} and the percentage of fractures caused by abuse declines sharply after the child begins to walk.^{9,19,20}

Fracture Specificity for Abuse

Fractures With High Specificity for Abuse

As shown in Table 1, certain fractures have high specificity for or strong association with child abuse, particularly in infants, whereas others may have less specificity.²¹ Rib fractures in infants, especially those situated posteromedially, and the classic metaphyseal lesions of long bones, have high specificity for child abuse. Fractures of the scapula, spinous process, and sternum also have high specificity for abuse but are uncommon.

Rib fractures are highly suggestive of child abuse. Most abusive rib fractures result from anterior-posterior compression of the chest. For this reason, rib fractures are frequently found in infants who are held around the chest, squeezed, and shaken. Rib fractures have high probability of being caused by abuse.^{15,17,21} The positive predictive value of rib fractures for child abuse in children younger than 3 years was 95% in one retrospective study.²² Other less common causes of rib fractures in infants include significant trauma sustained during childbirth or a motor vehicle crash as well as minor trauma

in infants who have increased bone fragility.^{23–25}

Cardiopulmonary resuscitation (CPR) has been proposed as a cause of rib fractures, but conventional CPR with 2 fingers of 1 hand rarely causes fractures in children.^{26,27} Recent recommendations that CPR be performed using 2 hands encircling the rib cage have raised concerns that this technique might cause rib fractures. An analysis of infants who were discovered during autopsy to have rib fractures and had received 2-handed chest compressions antemortem suggested that 2-handed CPR is associated with anterior-lateral rib fractures of the third to sixth ribs.²⁸ In this small study, no posterior rib fractures were observed. The fractures in these infants were always multiple, uniformly involved the fourth rib, and were sometimes bilateral. Additional research is needed to examine the relationship between the 2-handed CPR technique and rib fractures.

Classic metaphyseal lesions (CMLs) also have high specificity for child abuse when they occur during the first year of life.^{21,29} CMLs are the most common long bone fracture found in infants who die with evidence of inflicted injury.³⁰ CMLs are planar fractures through the primary spongiosa of the metaphysis. These fractures are caused when torsional and tractional shearing strains are applied across the metaphysis, as may occur with vigorous pulling or twisting of an infant's extremity.³¹ Fractures resembling CMLs radiographically have been reported after breech delivery³² and as a result of treatment of clubfoot.³³

Depending on the projection of the radiograph, CMLs can have the appearance of a corner or a bucket-handle fracture. Acute injuries can

TABLE 1 Specificity of radiologic findings in infants and toddlers¹⁹

High specificity ^a
CMLs
Rib fractures, especially posteromedial
Scapular fractures
Spinous process fractures
Sternal fractures
Moderate specificity
Multiple fractures, especially bilateral
Fractures of different ages
Epiphyseal separations
Vertebral body fractures and subluxations
Digital fractures
Complex skull fractures
Common, but low specificity
Subperiosteal new bone formation
Clavicular fractures
Long-bone shaft fractures
Linear skull fractures

^a Highest specificity applies in infants.

be difficult to visualize radiographically. CMLs commonly heal without subperiosteal new bone formation or marginal sclerosis. They can heal quickly and be undetectable on plain radiographs in 4 to 8 weeks.³¹

Fractures With Moderate Specificity for Abuse

Although many children who have been abused will have only a single fracture,³⁴ the presence of multiple fractures, fractures of different ages and/or stages of healing, and complex skull fractures have moderate specificity for physical abuse. In addition, epiphyseal separations, vertebral body fractures, and digital fractures have moderate specificity for abuse. The presence of multiple fractures or fractures of different ages can be signs of bone fragility but should also evoke consideration of child abuse. Besides the predictive value of the particular pattern of fractures, many other factors, such as the history and the child's age, must be considered when determining whether the injury was inflicted.

Common Fractures With Low Specificity for Child Abuse

Long bone fractures (other than CMLs), linear skull fractures, clavicle fractures, and isolated findings of subperiosteal new bone formation have low specificity for child abuse. In contrast, the single long bone diaphyseal fracture is the most common fracture pattern identified in abused children.^{1,13,34}

An understanding of the extent and type of load that is necessary to cause a particular long bone fracture can help to determine whether a specific fracture is consistent with the injury described by the caregiver.^{35,36} Transverse fractures of the long bones are caused by the application of a bending load in a direction that is perpendicular

to the bone, whereas spiral fractures are caused by torsion or twisting of a long bone along its long axis. Oblique fractures are caused by a combination of bending and torsion loads.³⁷ Torus or buckle fractures are the result of compression from axial loading along the length of the bone. Although earlier studies suggested that spiral fractures should always raise suspicion for child abuse,¹² more recent studies do not show that any particular fracture pattern can distinguish between abuse and nonabuse with absolute certainty.^{16,38}

Falls are common in childhood.³⁹ Short falls can cause fractures, but they rarely result in additional significant injury (eg, neurologic injury).^{11,40–42} In a retrospective study of short falls, parents reported that 40% of the children before 2 years of age had suffered at least 1 fall from a height of between 6 inches and 4 feet. Approximately one-quarter of these children suffered an injury; bruises were the most common injury observed.⁴³

The femur, humerus, and tibia are the most common long bones to be injured by child abuse.^{1,34} Femoral fractures in the nonambulatory child are more likely caused by child abuse, whereas these fractures in ambulatory children are most commonly noninflicted.^{10,16,43–45}

Certain femur fractures may occur as a result of a noninflicted injury in young children. Several studies have demonstrated that a short fall to the knee may produce a torus or impacted transverse fracture of the distal femoral metaphysis.^{46,47} Oblique distal femur metaphyseal fractures have been reported in children playing in a stationary activity center, such as an Exersaucer (Evenflo, Picqua, OH).⁴⁸

In both ambulatory and nonambulatory children, under some circumstances, falls on a stairway can cause a spiral femoral fracture. For example, a fall

down several steps and landing with 1 leg folded or twisted underneath a child can lead to excessive torsional loading of the femur and a spiral fracture.⁴⁶ In ambulatory children, noninflicted femoral fractures have been described in children who fell while running or who fell and landed in a split-leg position.⁴⁵

A fracture of the humeral shaft in a child younger than 18 months has a high likelihood of having been caused by abuse.^{15,49,50} In contrast, supracondylar fractures in ambulatory children are usually noninflicted injuries resulting from short falls.¹⁵

Physicians should also be aware of a particular mechanism reported to produce a noninflicted spiral-oblique fracture of the humerus in 1 case report.⁵¹ When the young infant was rolled from the prone position to the supine while the child's arm is extended, the torsion and stress placed on the extended arm appeared to cause a spiral-oblique fracture of the midshaft of the humerus.

Linear skull fractures of the parietal bone are the most common skull fracture among young children, usually children younger than 1 year.¹³ A short fall from several feet onto a hard surface can cause a linear, nondiastatic skull fracture.^{19,52} The majority of linear skull fractures are not inflicted.⁵³ By contrast, complex or bilateral skull fractures are typical of nonaccidental trauma.

Syndromes, Metabolic Disorders, Systemic Disease

Preexisting medical conditions and bone disease may make a child's bones more vulnerable to fracture. Some conditions may manifest skeletal changes, such as metaphyseal irregularity and subperiosteal new bone formation. These entities should be considered in the differential diagnosis of childhood fractures.

Osteogenesis Imperfecta

Osteogenesis imperfecta (OI) is a heterogeneous family of diseases, usually caused by heterozygous mutations of the genes *COL1A1* and *COL1A2*,⁵⁴ but mutations in these and other genes can cause autosomal recessive forms of OI. The *COL1A1* and *COL1A2* genes encode the chains of type I collagen, which forms the structural framework of bone. Although it is a genetic disorder, many children have de novo mutations or autosomal-recessive disease and no family history of bone fragility. In addition, the presentation of the disease within affected members of the same family can be quite variable. Phenotypic expression of the disease depends on the nature of the mutation, its relative abundance attributable to mosaicism, and its expression in target tissues.⁵⁵ Some types of OI involve reduced production of collagen, and the symptoms resolve or lessen after puberty.⁵⁶ Table 2 lists the various signs and symptoms that can be present in a case of OI.

The diagnosis of OI is often suggested by a family history of fractures, short stature, blue sclera, poor dentition, and radiographic evidence of low bone density or osteopenia. The fractures are most commonly transverse in nature, occurring in the shafts of the

long bones. It is unusual to have multiple long bone fractures or rib fractures, particularly in infancy, without other clinical and radiographic evidence of OI.^{57,58}

OI has been misdiagnosed as child abuse.⁵⁹ On the other hand, OI is often suggested as the cause of fractures in children who have been abused. If fractures continue to occur when a child is placed in a protective environment, a more thorough evaluation for an underlying bone disease is needed. Child abuse is more common than OI,⁶⁰ and children with OI and other metabolic or genetic conditions may also be abused.^{61,62}

Preterm Birth

Preterm infants have decreased bone mineralization at birth, but after the first year of life, bone density normalizes.^{63,64} Osteopenia of prematurity has been well described as a complication in low birth weight infants.⁶⁵ Infants born at less than 28 weeks' gestation or who weigh less than 1500 g at birth are particularly vulnerable. Osteopenia of prematurity is multifactorial. Infants are also at risk if they receive prolonged (for 4 or more weeks) total parenteral nutrition, have bronchopulmonary dysplasia, and/or have received a prolonged course of diuretics or steroids.⁶⁶ Osteopenia commonly presents between 6 and 12 weeks of life. Osteopenia of prematurity can be ameliorated if infants are monitored closely and receive the nutritional and mineral supplementation initiated in the NICU.

Fractures associated with osteopenia of prematurity usually occur in the first year of life.⁶⁷ Rib fractures are typically encountered incidentally, whereas long bone fractures commonly present with swelling of the extremity. Osteopenia of prematurity can be associated with rickets, and in such cases, metaphyseal irregularities may be present.

Although osteopenia of prematurity may make the infant more vulnerable to fracture, preterm infants are also at an increased risk of abuse.⁶⁸

Vitamin D Deficiency Rickets

Suboptimal vitamin D concentrations and rickets have been proposed as causes of fractures in infants.⁶⁹ Vitamin D insufficiency in otherwise healthy infants and toddlers is common. Approximately 40% of infants and toddlers aged 8 to 24 months in an urban clinic had laboratory evidence of vitamin D insufficiency (serum concentrations of 25-hydroxyvitamin D of ≤ 30 ng/mL).⁷⁰ Prolonged breastfeeding without vitamin D supplementation was a critical factor that placed these infants at risk, although increased skin pigmentation and/or lack of sunlight exposure may also have contributed. Rickets is characterized by demineralization, loss of the zone of provisional calcification, widening and irregularity of the physis, and fraying and cupping of the metaphysis.⁷¹ Despite the high prevalence of vitamin D insufficiency in infants and toddlers, rickets is uncommon.⁷²

The claim that vitamin D deficiency or insufficiency causes skeletal lesions that lead to the incorrect diagnosis of child abuse in infants is not supported in the literature. A systematic clinical, laboratory, and radiologic assessment should exclude that possibility.^{73–75} Schilling et al found no difference in serum concentrations of 25-hydroxyvitamin D in young children with fractures suspicious for abuse and noninjured fractures.⁷⁶ Vitamin D insufficiency was not associated with multiple fractures, in particular rib fractures or CMLs, the high specificity indicators of abuse. Perez-Rossello et al studied radiographs of 40 healthy older infants and toddlers with

TABLE 2 Characteristics of Osteogenesis Imperfecta

Fragile bones with few, some, or many of the following findings:
Poor linear growth
Macrocephaly
Triangular-shaped face
Blue sclerae
Hearing impairment as a result of otosclerosis
Hypoplastic, translucent, carious, late-erupting, or discolored teeth
Easy bruisability
Inguinal and/or umbilical hernias
Limb deformities
Hyperextensible joints
Scoliosis and/or kyphosis
Wormian bones of the skull
Demineralized bones

vitamin D insufficiency and deficiency and concluded that radiographic rachitic changes were uncommon and very mild. In this population, the reported fracture prevalence was zero.⁷²

In a study of 45 young children with radiographic evidence of rickets, investigators found that fractures occurred only in those infants and toddlers who were mobile.⁷⁷ Fractures were seen in 17.5% of the children, and these children were 8 to 19 months of age. The fractures involved long bones, anterior-lateral and lateral ribs, and metatarsal and metaphyseal regions. The metaphyseal fractures occurred closer to the diaphysis in the background of florid metaphyseal rachitic changes and did not resemble the juxtaphyseal corner or bucket handle pattern of the CML. In infant fatalities in which abuse is suspected, rachitic changes appear to be rare histologically.⁷⁸

Osteomyelitis

Osteomyelitis in infants can present as multiple metaphyseal irregularities potentially resembling CMLs.⁷⁹ Typically, the lesions become progressively lytic and sclerotic with substantial subperiosteal new bone formation. Other signs of infection are often present, such as fever, increased erythrocyte sedimentation rate, elevated C-reactive protein concentration, and elevated white blood cell count.

Fractures Secondary to Demineralization From Disuse

Any child with a severe disability that limits or prevents ambulation can be at risk for fractures secondary to disuse demineralization, even with normal handling.^{80,81} The fractures are usually diaphyseal rather than CMLs. Often, these fractures occur during physical therapy and range-of-motion exercises. It can be difficult to distinguish between

inflicted and noninflicted fractures occurring in these children. At the same time, children with disabilities are at an increased risk of being maltreated.^{82–84} When multiple or recurrent fractures occur in a disabled child, a trial change in caregivers may be indicated to determine whether the fractures can be prevented. This is an extreme intervention and should be reserved for unusual circumstances.⁶⁵

Scurvy

Scurvy is caused by insufficient intake of vitamin C, which is important for the synthesis of collagen. Although rare today because formula, human milk, fruits, and vegetables contain vitamin C, scurvy may develop in older infants and children given exclusively cow milk without vitamin supplementation and in children who eat no foods containing vitamin C.^{85–87} Although scurvy can result in metaphyseal changes similar to those seen with child abuse, other characteristic bone changes, including osteopenia, increased sclerosis of the zones of provisional calcification, dense epiphyseal rings, and extensive calcification of subperiosteal and soft tissue hemorrhages, will point to the diagnosis of scurvy.

Copper Deficiency

Copper plays a role in cartilage formation. Copper deficiency is a rare condition that may be complicated by bone fractures. Preterm infants are born with lower stores of copper than term infants, because copper is accumulated at a faster rate during the last trimester.⁸⁸ Copper insufficiency may be observed in children with severe nutritional disorders, for example, liver failure or short gut syndrome.⁸⁹ This deficiency is not likely to be observed in full-term children younger than 6 months of age or preterm infants younger than 2.5

months of age, because fetal copper stores are sufficient for this length of time. In addition, human milk and formula contain sufficient copper to prevent deficiency. Psychomotor retardation, hypotonia, hypopigmentation, pallor, and a sideroblastic anemia are some of the characteristic findings of copper deficiency in infants. Radiologic changes that should lead to further evaluation for possible deficiency include cupping and fraying of the metaphyses, sickle-shaped metaphyseal spurs, significant demineralization, and subperiosteal new bone formation.

Menkes Disease

Menkes disease, also known as Menkes kinky hair syndrome, is a rare congenital defect of copper metabolism.⁹⁰ Menkes disease is an X-linked recessive condition and occurs only in boys. Although it has many of the features of dietary copper deficiency, anemia is not associated with Menkes disease. Metaphyseal fragmentation and subperiosteal new bone formation may be observed on radiographs, and the findings may be difficult to distinguish from fractures caused by abuse.⁹¹ Other signs of Menkes disease include sparse, kinky hair, calvarial wormian bones, anterior rib flaring, failure to thrive, and developmental delay. A characteristic finding is tortuous cerebral vessels. Intracranial hemorrhage can occur in Menkes disease but has not been reported in infants with copper deficiency.

Systemic Disease

Chronic renal disease affects bone metabolism because children with chronic renal disease may develop a metabolic acidosis that interferes with vitamin D metabolism. Chronic renal disease can cause renal osteodystrophy resulting in the same radiographic changes as nutritional

rickets. Because chronic liver disease (eg, biliary atresia) interferes with vitamin D metabolism, such children may be at an increased risk of fractures. Fanconi syndrome, hypophosphatasia, hypophosphatemic (vitamin D resistant) rickets, hyperparathyroidism, and renal tubular acidosis also cause clinical variants of rickets.

Temporary Brittle Bone Disease Hypothesis

Physicians should be aware of alternative diagnoses that are unsupported by research but are sometimes suggested when an infant has unexplained fractures. In 1993, Paterson proposed that some infants may be born with bones that are temporarily more fragile or vulnerable to fracture in the context of normal handling, which he called “temporary brittle bone disease.”⁹² Paterson suggested that some trace element deficiency, such as copper or a transient collagen immaturity, caused the disease but provided no scientific data that confirmed his hypotheses and offered no specific test that confirmed temporary brittle bone disease.⁶¹ Subsequent studies did not support his hypotheses, and his case analysis has been refuted.^{57,93–95}

Miller hypothesized that temporary brittle bone disease is a result of fetal immobilization or intrauterine confinement that leads to transient bone loss or osteopenia.^{96,97} In support of his hypothesis, he reported that 95% of 21 infants with multiple unexplained fractures had decreased fetal movements, according to their mothers.^{97,98} Although he used bone densitometry in each patient as a basis for his conclusions, none of the patients had had bone densitometry performed at the time of the fracture. The testing was performed 8 to 21 weeks later, and no infants were tested before 5 months of age. In addition, bone densitometry standards have not been established

for infants. He relied on the mother’s history of decreased fetal movements and provided no independent measurements of those movements. Palacios and Rodríguez found no evidence that oligohydramnios affects bone mass of the fetus, probably because fetal movement is only restricted in the last trimester of pregnancy by oligohydramnios and because the mechanical loading on the bones stimulating bone formation is conserved.⁹⁹

Medical Evaluation

History of Present Illness

It is essential to obtain a detailed history to determine how an injury occurred. If an injury in a nonverbal child was witnessed, the caregiver should be able to provide details about the child’s activity and position before an injury and the child’s final position and location after the injury occurred.⁴⁶ Verbal children with concerning fractures should be interviewed apart from caregivers and ideally by a professional who is skilled in forensic interviewing.

A comparison of the histories provided by caregivers of children with non-inflicted femoral fractures and by caregivers of children whose injuries were caused by abuse is instructive. When an injury was caused by abuse, the caregiver provided either no history of an injury or related a history of a low-energy event. By contrast, 29% of the caregivers of children with non-inflicted injuries provided some high-energy explanation, such as a motor vehicle collision or that the child fell from a height.¹⁶ Most of the low-energy mechanisms provided for the noninflicted injuries involved falls including stair falls and siblings landing on the femur during play.^{16,46}

The child’s response to the event may also provide important clues about the etiology. The majority of children with long bone fractures will have

some swelling, pain, or other signs, such as decreased use of the extremity, suggesting a fracture.^{100,101} Some children, however, will have minimal external signs of injury.¹⁰² The absence of any history of injury, a vague description of the event, a delay in seeking care, the absence of an explanation for an injury particularly in a nonambulatory child, or an inconsistent explanation should increase the physician’s concern that an injury was caused by child abuse (see Table 3).^{13,16}

Past Medical History

The past medical history is important and should include details about the mother’s pregnancy. If the child was born preterm, the infant’s bone mineral content may be reduced, and the infant may be at risk for fracture. A history of total parental nutrition, hepatobiliary disease, diuretic therapy, hypercalciuria, or corticosteroids may make the bones of a low birth weight infant even more vulnerable to fracture. In addition, chronic diseases, such as renal insufficiency or metabolic acidosis, malabsorption, cerebral palsy or other neuromuscular disorders, genetic diseases that affect skeletal development, or any illness that limits mobility, may affect bone strength. A thorough dietary history and history of medications that can

TABLE 3 When Is a Fracture Suspicious for Child Abuse?

- No history of injury
- History of injury not plausible—mechanism described not consistent with the type of fracture, the energy load needed to cause the fracture, or the severity of the injury
- Inconsistent histories or changing histories provided by caregiver
- Fracture in a nonambulatory child
- Fracture of high specificity for child abuse (eg, rib fractures)
- Multiple fractures
- Fractures of different ages
- Other injuries suspicious for child abuse
- Delay in seeking care for an injury

predispose to fractures are important. The physician should inquire about previous injuries including bruises and determine the child's developmental abilities, because children who are not yet mobile are much more likely to have fractures caused by abuse.

Family History

A family history of multiple fractures, early-onset hearing loss, abnormally developed dentition, blue sclera, and short stature should suggest the possibility of OI.

Social History

The physician should obtain a complete psychosocial history, including asking who lives in the home and who has provided care for the child. The history should inquire about intimate partner violence, substance abuse including drugs and alcohol, mental illness, and previous involvement with child protective services and/or law enforcement.

Physical Examination

The child should have a comprehensive physical examination, and the growth chart should be carefully reviewed. Abnormal weight may suggest neglect or endocrine or metabolic disorders. Any signs or symptoms of fractures, such as swelling, limitation of motion, and point tenderness should be documented. The physician should do a complete skin examination to look for bruises and other skin findings because bruises are the most common injury caused by child abuse. The majority of children with fractures do not have bruising associated with the fracture; the presence or absence of such bruising does not help to determine which fractures are caused by child abuse.^{103,104} Bruising in a child who is not yet cruising or bruising in unusual locations, such as the ears,

neck, or trunk should raise suspicion for child abuse.^{105,106} The child should be examined for other injuries caused by child abuse, in addition to signs of other medical conditions associated with bone fragility. Blue sclerae are seen in certain types of OI. Sparse, kinky hair is associated with Menkes disease. Dentinogenesis imperfecta is occasionally identified in older children with OI.

Laboratory Evaluation

The clinical evaluation should guide the laboratory evaluation. In children with fractures suspicious for abuse, serum calcium, phosphorus, and alkaline phosphatase should be reviewed, although alkaline phosphatase may be elevated with healing fractures. The physician should consider checking serum concentrations of parathyroid hormone and 25-hydroxyvitamin D, as well as urinary calcium excretion (eg, random urinary calcium/creatinine ratio) in all young children with fractures concerning for abuse, but these levels should certainly be assessed if there is radiographic evidence of osteopenia or metabolic bone disease. Screening for abdominal trauma with liver function studies as well as amylase and lipase concentration should be performed when severe or multiple injuries are identified. A urinalysis should be performed to screen for occult blood. Serum copper, vitamin C, and ceruloplasmin concentrations should be considered if the child is at risk for scurvy or copper deficiency and has radiographic findings that include metaphyseal abnormalities.

If OI is suspected, sequence analysis of the *COL1A1* and *COL1A2* genes that are associated with 90% of cases of OI as well as other genes associated with less common autosomal-recessive forms of OI may be more sensitive than biochemical tests of

type I collagen and may identify the mutation to guide testing of other family members.¹⁰⁷ Some of the less common forms of OI are OI types IIB and VII, *CRTAP*; OI type VI, *FKBP10*; OI type VIII, *LEPRE1*; OI type IX, *PP1B*; OI type X, *SERPINH1*; OI type XI, *SP7*; OI type XII, *SERPINF1*; and OI type XIII, *BMP1*. DNA sequencing can be performed using genomic DNA isolated from peripheral blood mononuclear cells or even saliva, whereas the biochemical analysis of type I collagen requires a skin biopsy. Doing both DNA analysis and skin biopsy is not indicated in most cases. Consultation with a pediatric geneticist may be helpful in deciding which children to test and which test to order.¹⁰⁸

Imaging Approach

Children younger than 2 years with fractures suspicious for child abuse should have a radiographic skeletal survey to look for other bone injuries or osseous abnormalities.¹⁰⁹ Additional fractures are identified in approximately 10% of skeletal surveys, with higher yields in infants.¹¹⁰ Skeletal surveys may be appropriate in some children between ages 2 and 5 years, depending on the clinical suspicion of abuse. If specific clinical findings indicate an injury at a particular site, imaging of that area should be obtained regardless of the child's age.

The American College of Radiology has developed specific practice guidelines for skeletal surveys in children.¹¹¹ Twenty-one images are obtained, including frontal images of the appendicular skeleton, frontal and lateral views of the axial skeleton, and oblique views of the chest. Oblique views of the chest have been shown to increase the sensitivity, specificity, and accuracy of the identification of rib fractures.¹¹² A full 4 skull series should be obtained if there are concerns of

head injury. Computed tomography (CT) 3-dimensional models are valuable adjuncts to the radiographs and have the potential to replace the skull series.¹¹³ This has not been studied systematically in this context, however. Because lateral views of the extremities increase yield, some authors suggest that these views be included in the imaging protocol.¹¹⁴ Fractures may be missed if the guidelines are not followed or if the images are of poor quality.¹¹⁵ A repeat skeletal survey should be performed approximately 2 to 3 weeks after the initial skeletal survey if child abuse is strongly suspected.^{109,116} The follow-up examination may identify fractures not seen on the initial skeletal survey, can clarify uncertain findings identified by the initial skeletal survey, and improves both sensitivity and specificity of the skeletal survey.^{116,117} In one study, 13 of 19 fractures found on the follow-up examination were not seen on the initial series.¹¹⁶ The number of images on the follow-up examination may be limited to 15 views by omitting the views of the skull, pelvis, and lateral spine.¹¹⁸

Radiography may assist in assessing the approximate time when an injury occurred because long bone fractures heal following a particular sequence.¹¹⁹ If the healing pattern is not consistent with the explanation provided, the accuracy of the explanation should be questioned.

Bone scintigraphy may be used to complement the skeletal survey but should not be the sole method of identifying fractures in infants. Although it has high overall sensitivity, it lacks specificity for fracture detection and may fail to identify CMLs and skull fractures.^{109,119,120} Scintigraphy does have high sensitivity for identifying rib fractures, which can be difficult to detect before healing. In toddlers and older children, the use of bone scintigraphy or skeletal survey depends on

the specific clinical indicators of abuse.¹⁰⁹

Because brain injuries are often occult, head imaging should be considered for any child younger than 1 year with a fracture suspicious for abuse.¹²¹ Imaging studies may help clarify whether the child has been abused, provide further support for a diagnosis of child abuse, and identify other injuries that require treatment. Additional imaging may be needed if the child has signs or symptoms of chest, abdominal, or neck injury.

Chest CT can identify rib fractures that are not seen on chest radiographs.¹²² CT is particularly useful in detecting anterior rib fractures and rib fractures at all stages of healing—early subacute, subacute, and old fractures. Although CT may be more sensitive in identifying these injuries, a chest CT exposes the child to significantly more radiation than chest radiography. Every effort should be made to reduce children's exposure to radiation while at the same time considering the risk to the child if abuse is not identified.¹²³ Therefore, selective application of this technique in certain clinical settings is appropriate.

Other modalities may become available in the future that will provide more accurate identification of skeletal injuries. Whole-body short tau inverse recovery imaging, a magnetic resonance imaging (MRI) technique, may identify rib fractures not recognized on the radiographic skeletal survey.¹²⁴ In a study of 21 infants with suspected abuse, whole-body MRI at 1.5-Tesla was insensitive in the detection of CMLs and rib fractures. In some cases, whole-body MRI identified soft tissue edema and joint effusions that led to the identification of skeletal injuries with additional radiographs.¹²⁵ Bone scintigraphy with ¹⁸F-sodium fluoride positron emission tomography (¹⁸F-NaF PET) bone scan may be useful in cases

of equivocal or negative skeletal surveys when there is high clinical suspicion of abuse. If available, a ¹⁸F-NaF positron emission tomography bone scan has better contrast and spatial resolution than ^{99m}Tc-labeled methylene diphosphonate.¹²⁰

Although bone densitometry by dual-energy x-ray absorptiometry is useful to predict bone fragility and fracture risk in older adults, interpretation of bone densitometry in children and adolescents is more problematic.¹²⁶ In adults, bone densitometry is interpreted using T scores, which describe the number of SDs above or below the average peak bone mass for a gender- and race-adjusted reference group of normal subjects. Because peak bone mass is not achieved until approximately 30 years of age, in children, z scores must be used to express bone density, because z scores express the child's bone mineral density as a function of SDs above or below the average for an age- and gender-matched norm control population.¹²⁷ In addition, because bone size influences dual-energy x-ray absorptiometry, z scores must also be adjusted for height z scores.¹²⁸ The International Society for Clinical Densitometry recommends that the diagnosis of osteoporosis in childhood should not be made on the basis of low bone mass alone but should also include a clinically significant history of low-impact fracture. The recommendations currently apply to children 5 years and older, although reference data are available for children as young as 3 years.^{129,130} Unfortunately, there are limited reference data for the young, nonverbal child who is most at risk for suffering fractures caused by child abuse.

Evaluation of Siblings

Siblings, especially twins, and other young household members of children who have been physically abused

should be evaluated for maltreatment.¹³¹ In a study of 795 siblings in 400 households of a child who had been abused or neglected, all siblings in 37% of households and some siblings in 20% of households had suffered some form of maltreatment.¹³² In this study, which included all manifestations of maltreatment, siblings were found to be more at risk for maltreatment if the index child suffered moderate or severe maltreatment. In addition to a careful evaluation, imaging should be considered for any siblings younger than 2 years, especially if there are signs of abuse.

DIAGNOSIS

When evaluating a child with a fracture, physicians must take a careful history of any injury event and then determine whether the mechanism described and the severity and timing are consistent with the injury identified (see Table 3).¹³³ They must consider and evaluate for possible diagnoses in addition to other signs or symptoms of child abuse. A careful evaluation for other injuries is important because the presence of additional injuries that are associated with child abuse increases the likelihood that a particular fracture was inflicted.^{16,43} It is important to remember that even if a child has an underlying disorder or disability that could increase the likelihood of a fracture, the child may also have been abused because children with disabilities and other special health care needs are at increased risk of child abuse.^{83,84} Physicians should keep an open mind to the possibility of abuse and remember that child abuse occurs in all socioeconomic

groups and across all racial and ethnic groups. Many of these diagnoses are complex. If a physician is uncertain about how to evaluate an injury or if they should suspect a fracture was caused by child abuse, they should consult a child abuse pediatrician or multidisciplinary child abuse team to assist in the evaluation, particularly if the child is nonambulatory or younger than 1 year of age.¹³⁴ In certain circumstances, the physician will need to consult an orthopedist, endocrinologist, geneticist, or other subspecialists.

All US states, commonwealths, and territories have mandatory reporting requirements for physicians and other health care providers when child abuse is suspected. Physicians should be aware of and comply with the reporting requirements of their state. Typically, the standard for making a report is when the reporter “suspects” or “has reason to believe” that a child has been abused or neglected. Sometimes determining whether that “reasonable belief” or “reasonable suspicion” standard has been met can be nuanced and complex. The physician should keep in mind that incontrovertible proof of abuse or neglect is not required by state statutes, and there may be cases in which it is reasonable to consult with a child abuse pediatrician about whether a report should be made.

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REFERENCES

- Loder RT, Feinberg JR. Orthopaedic injuries in children with nonaccidental trauma: demographics and incidence from the 2000 kids' inpatient database [published correction appears in *J Pediatr Orthop*. 2008;28(6):699]. *J Pediatr Orthop*. 2007;27(4):421–426
- Jenny C, Hymel KP, Ritzen A, Reinert SE, Hay TC. Analysis of missed cases of abusive head trauma [see comment; published correction appears in *JAMA*. 1999;282(1):29]. *JAMA*. 1999;281(7):621–626
- Ravichandiran N, Schuh S, Bejuk M, et al. Delayed identification of pediatric abuse-related fractures. *Pediatrics*. 2010;125(1):60–66
- Skellern C, Donald T. Suspicious childhood injury: formulation of forensic opinion. *J Paediatr Child Health*. 2011;47(11):771–775
- O'Neill JA, Jr, Meacham WF, Griffin JP, Sawyers JL. Patterns of injury in the battered child syndrome. *J Trauma*. 1973;13(4):332–339
- Gallagher SS, Finison K, Guyer B, Goodenough S. The incidence of injuries among 87,000 Massachusetts children and adolescents: results of the 1980–81 Statewide Childhood Injury Prevention Program Surveillance System. *Am J Public Health*. 1984;74(12):1340–1347
- Spady DW, Saunders DL, Schopflocher DP, Svenson LW. Patterns of injury in children: a population-based approach. *Pediatrics*. 2004;113(3 pt 1):522–529
- Rennie L, Court-Brown CM, Mok JYQ, Beattie TF. The epidemiology of fractures in children. *Injury*. 2007;38(8):913–922
- Leventhal JM, Martin KD, Asnes AG. Incidence of fractures attributable to abuse in young hospitalized children: results from analysis of a United States database. *Pediatrics*. 2008;122(3):599–604
- Schwend RM, Werth C, Johnston A. Femur shaft fractures in toddlers and young children: rarely from child abuse. *J Pediatr Orthop*. 2000;20(4):475–481
- Hennrikus WL, Shaw BA, Gerardi JA. Injuries when children reportedly fall from a bed or couch. *Clin Orthop Relat Res*. 2003; (407):148–151
- Worlock P, Stower M, Barbor P. Patterns of fractures in accidental and non-accidental injury in children: a comparative study. *Br Med J (Clin Res Ed)*. 1986;293(6539):100–102
- Leventhal JM, Thomas SA, Rosenfield NS, Markowitz RI. Fractures in young children. Distinguishing child abuse from unintentional injuries. *Am J Dis Child*. 1993;147(1):87–92
- Leventhal JM, Larson IA, Abdo D, et al. Are abusive fractures in young children becoming less common? Changes over 24 years. *Child Abuse Negl*. 2007;31(3):311–322
- Kemp AM, Dunstan F, Harrison S, et al. Patterns of skeletal fractures in child abuse: systematic review [review]. *BMJ*. 2008;337:a1518
- Hui C, Joughin E, Goldstein S, et al. Femoral fractures in children younger than three years: the role of nonaccidental injury. *J Pediatr Orthop*. 2008;28(3):297–302
- Pandya NK, Baldwin K, Wolfgruber H, Christian CW, Drummond DS, Hosalkar HS. Child abuse and orthopaedic injury patterns: analysis at a level I pediatric trauma center. *J Pediatr Orthop*. 2009;29(6):618–625
- Coffey C, Haley K, Hayes J, Groner JL. The risk of child abuse in infants and toddlers with lower extremity injuries. *J Pediatr Surg*. 2005;40(1):120–123
- Kleinman PK. The spectrum of nonaccidental injuries (child abuse) and its imitators. In: Hodler J, Zollikofer CL, Schulthess GK, eds. *Musculoskeletal Diseases 2009–2012*. Milan, Italy: Springer Italia; 2009:227–233
- Clarke NMP, Shelton FRM, Taylor CC, Khan T, Needhirajan S. The incidence of fractures in children under the age of 24 months—in relation to non-accidental injury. *Injury*. 2012;43(6):762–765
- Kleinman PK. *Diagnostic Imaging of Child Abuse*. 2nd ed. St. Louis, MO: Mosby; 1998
- Barsness KA, Cha E-S, Bensard DD, et al. The positive predictive value of rib fractures as an indicator of nonaccidental trauma in children [see comment]. *J Trauma*. 2003;54(6):1107–1110
- Bulloch B, Schubert CJ, Brophy PD, Johnson N, Reed MH, Shapiro RA. Cause and clinical characteristics of rib fractures in infants. *Pediatrics*. 2000;105(4). Available at: www.pediatrics.org/cgi/content/full/105/4/E48
- Kleinman PK, Schlessinger AE. Mechanical factors associated with posterior rib fractures: laboratory and case studies. *Pediatr Radiol*. 1997;27(1):87–91
- Bixby SD, Abo A, Kleinman PK. High-impact trauma causing multiple posteromedial rib fractures in a child. *Pediatr Emerg Care*. 2011;27(3):218–219
- Feldman KW, Brewer DK. Child abuse, cardiopulmonary resuscitation, and rib fractures. *Pediatrics*. 1984;73(3):339–342
- Spevak MR, Kleinman PK, Belanger PL, Primack C, Richmond JM. Cardiopulmonary resuscitation and rib fractures in infants. A postmortem radiologic-pathologic study. *JAMA*. 1994;272(8):617–618
- Matshes EW, Lew EO. Two-handed cardiopulmonary resuscitation can cause rib fractures in infants. *Am J Forensic Med Pathol*. 2010;31(4):303–307
- Kleinman PK, Perez-Rossello JM, Newton AW, Feldman HA, Kleinman PL. Prevalence of the classic metaphyseal lesion in infants at low versus high risk for abuse. *AJR Am J Roentgenol*. 2011;197(4):1005–1008
- Kleinman PK, Marks SC, Jr, Richmond JM, Blackbourne BD. Inflicted skeletal injury: a postmortem radiologic-histopathologic study in 31 infants. *AJR Am J Roentgenol*. 1995;165(3):647–650
- Kleinman PK. Problems in the diagnosis of metaphyseal fractures. *Pediatr Radiol*. 2008;38(suppl 3):S388–S394
- O'Connell A, Donoghue VB. Can classic metaphyseal lesions follow uncomplicated caesarean section? *Pediatr Radiol*. 2007;37(5):488–491
- Grayev AM, Boal DK, Wallach DM, Segal LS. Metaphyseal fractures mimicking abuse during treatment for clubfoot [see comment]. *Pediatr Radiol*. 2001;31(8):559–563
- King J, Diefendorf D, Aphthorp J, Negrete VF, Carlson M. Analysis of 429 fractures in 189 battered children. *J Pediatr Orthop*. 1988;8(5):585–589
- Pierce MC, Bertocci G. Injury biomechanics and child abuse. *Annu Rev Biomed Eng*. 2008;10:85–106
- Pierce MC, Bertocci GE, Vogetley E, Moreland MS. Evaluating long bone fractures in children: a biomechanical approach with illustrative cases. *Child Abuse Negl*. 2004;28(5):505–524
- Pierce MC, Bertocci G. Fractures resulting from inflicted trauma: assessing injury and history compatibility. *Clin Pediatr Emerg Med*. 2006;7(3):143–148
- Rex C, Kay PR. Features of femoral fractures in nonaccidental injury. *J Pediatr Orthop*. 2000;20(3):411–413
- Haney SB, Starling SP, Heisler KW, Okwara L. Characteristics of falls and risk of injury in children younger than 2 years. *Pediatr Emerg Care*. 2010;26(12):914–918
- Nimityongskul P, Anderson LD. The likelihood of injuries when children fall out of bed. *J Pediatr Orthop*. 1987;7(2):184–186

41. Lyons TJ, Oates RK. Falling out of bed: a relatively benign occurrence. *Pediatrics*. 1993;92(1):125–127
42. Hansoti B, Beattie T. Can the height of fall predict long bone fracture in children under 24 months? *Eur J Emerg Med*. 2005; 12(6):285–286
43. Thomas SA, Rosenfield NS, Leventhal JM, Markowitz RI. Long-bone fractures in young children: distinguishing accidental injuries from child abuse. *Pediatrics*. 1991;88(3):471–476
44. Loder RT, O'Donnell PW, Feinberg JR. Epidemiology and mechanisms of femur fractures in children. *J Pediatr Orthop*. 2006;26(5):561–566
45. Baldwin K, Pandya NK, Wolfgruber H, Drummond DS, Hosalkar HS. Femur fractures in the pediatric population: abuse or accidental trauma? *Clin Orthop Relat Res*. 2011;469(3):798–804
46. Pierce MC, Bertocci GE, Janosky JE, et al. Femur fractures resulting from stair falls among children: an injury plausibility model. *Pediatrics*. 2005;115(6):1712–1722
47. Haney SB, Boos SC, Kutz TJ, Starling SP. Transverse fracture of the distal femoral metadiaphysis: a plausible accidental mechanism. *Pediatr Emerg Care*. 2009;25 (12):841–844
48. Grant P, Mata MB, Tidwell M. Femur fracture in infants: a possible accidental etiology. *Pediatrics*. 2001;108(4):1009–1011
49. Pandya NK, Baldwin KD, Wolfgruber H, Drummond DS, Hosalkar HS. Humerus fractures in the pediatric population: an algorithm to identify abuse. *J Pediatr Orthop B*. 2010;19(6):535–541
50. Strait RT, Siegel RM, Shapiro RA. Humeral fractures without obvious etiologies in children less than 3 years of age: when is it abuse? *Pediatrics*. 1995;96(4 pt 1):667–671
51. Hymel KP, Jenny C. Abusive spiral fractures of the humerus: a videotaped exception. *Arch Pediatr Adolesc Med*. 1996; 150(2):226–227
52. Laskey AL, Stump TE, Hicks RA, Smith JL. Yield of skeletal surveys in children \leq 18 months of age presenting with isolated skull fractures. *J Pediatr*. 2013;162(1):86–89
53. Wood JN, Christian CW, Adams CM, Rubin DM. Skeletal surveys in infants with isolated skull fractures. *Pediatrics*. 2009;123 (2). Available at: www.pediatrics.org/cgi/content/full/123/2/e247-e252
54. Byers PH, Steiner RD. Osteogenesis imperfecta. *Annu Rev Med*. 1992;43:269–282
55. Wallis GA, Starman BJ, Zinn AB, Byers PH. Variable expression of osteogenesis imperfecta in a nuclear family is explained by somatic mosaicism for a lethal point mutation in the alpha 1(I) gene (COL1A1) of type I collagen in a parent. *Am J Hum Genet*. 1990;46(6):1034–1040
56. Barsh GS, Byers PH. Reduced secretion of structurally abnormal type I procollagen in a form of osteogenesis imperfecta. *Proc Natl Acad Sci USA*. 1981;78(8):5142–5146
57. Sprigg A. Temporary brittle bone disease versus suspected non-accidental skeletal injury. *Arch Dis Child*. 2011;96(5):411–413
58. Greeley CS, Donaruma-Kwom M, Vettimattam M, Lobo C, Williard C, Mazur L. Fractures at diagnosis in infants and children with osteogenesis imperfecta. *J Pediatr Orthop*. 2013;33(1):32–36 doi:10.1097/BPO.1090-b1013e318279c318255d
59. Singh Kocher M, Dichtel L. Osteogenesis imperfecta misdiagnosed as child abuse. *J Pediatr Orthop B*. 2011;20(6):440–443
60. Gahagan S, Rimsza ME. Child abuse or osteogenesis imperfecta: how can we tell? *Pediatrics*. 1991;88(5):987–992
61. Ablin DS, Sane SM. Non-accidental injury: confusion with temporary brittle bone disease and mild osteogenesis imperfecta. *Pediatr Radiol*. 1997;27(2): 111–113
62. Knight DJ, Bennet GC. Nonaccidental injury in osteogenesis imperfecta: a case report. *J Pediatr Orthop*. 1990;10(4):542–544
63. Jenny C, Committee on Child Abuse and Neglect. Evaluating infants and young children with multiple fractures. *Pediatrics*. 2006;118(3):1299–1303
64. Backström MC, Kuusela A-L, Mäki R. Metabolic bone disease of prematurity. *Ann Med*. 1996;28(4):275–282
65. Naylor KE, Eastell R, Shattuck KE, Alfrey AC, Klein GL. Bone turnover in preterm infants. *Pediatr Res*. 1999;45(3):363–366
66. Harrison CM, Johnson K, McKechnie E. Osteopenia of prematurity: a national survey and review of practice. *Acta Paediatr*. 2008;97(4):407–413
67. Amir J, Katz K, Grunebaum M, Yosipovich Z, Wielunsky E, Reinsner SH. Fractures in premature infants. *J Pediatr Orthop*. 1988; 8(1):41–44
68. Bugental DB, Happany K. Predicting infant maltreatment in low-income families: the interactive effects of maternal attributions and child status at birth. *Dev Psychol*. 2004;40(2):234–243
69. Keller KA, Barnes PD. Rickets vs. abuse: a national and international epidemic. *Pediatr Radiol*. 2008;38(11):1210–1216
70. Gordon CM, Feldman HA, Sinclair L, et al. Prevalence of vitamin D deficiency among healthy infants and toddlers. *Arch Pediatr Adolesc Med*. 2008;162(6):505–512
71. Greenspan A. *Orthopedic Imaging: A Practical Approach*. 4th ed. Philadelphia, PA: Lippincott Williams & Wilkins; 2004
72. Perez-Rossello JM, Feldman HA, Kleinman PK, et al. Rachitic changes, demineralization, and fracture risk in healthy infants and toddlers with vitamin D deficiency. *Radiology*. 2012;262(1):234–241
73. Slovis TL, Chapman S. Vitamin D insufficiency/deficiency—a conundrum. *Pediatr Radiol*. 2008;38(11):1153
74. Slovis TL, Chapman S. Evaluating the data concerning vitamin D insufficiency/deficiency and child abuse. *Pediatr Radiol*. 2008;38(11):1221–1224
75. Jenny C. Rickets or abuse? *Pediatr Radiol*. 2008;38(11):1219–1220
76. Schilling S, Wood JN, Levine MA, Langdon D, Christian CW. Vitamin D status in abused and nonabused children younger than 2 years old with fractures. *Pediatrics*. 2011;127(5):835–841
77. Chapman T, Sugar N, Done S, Marasigan J, Wambold N, Feldman K. Fractures in infants and toddlers with rickets. *Pediatr Radiol*. 2010;40(7):1184–1189
78. Perez-Rossello JM, McDonald AG, Rosenberg AE, Ivey SL, Richmond JM, Kleinman PK. Prevalence of rachitic changes in deceased infants: a radiologic and pathologic study. *Pediatr Radiol*. 2011;41(suppl 1):S57
79. Ogden JA. Pediatric osteomyelitis and septic arthritis: the pathology of neonatal disease. *Yale J Biol Med*. 1979;52(5):423–448
80. Whedon GD. Disuse osteoporosis: physiological aspects. *Calcif Tissue Int*. 1984;36 (suppl 1):S146–S150
81. Presedo A, Dabney KW, Miller F. Fractures in patients with cerebral palsy. *J Pediatr Orthop*. 2007;27(2):147–153
82. Westcott H. The abuse of disabled children: a review of the literature. *Child Care Health Dev*. 1991;17(4):243–258
83. Sullivan PM, Knutson JF. The association between child maltreatment and disabilities in a hospital-based epidemiological study. *Child Abuse Negl*. 1998;22(4): 271–288
84. Sullivan PM, Knutson JF. Maltreatment and disabilities: a population-based epidemiological study. *Child Abuse Negl*. 2000;24(10):1257–1273

85. Olmedo JM, Yiannias JA, Windgassen EB, Gornet MK. Scurvy: a disease almost forgotten. *Int J Dermatol*. 2006;45(8):909–913
86. Larralde M, Santos Muñoz A, Boggio P, Di Gruccio V, Weis I, Schygiel A. Scurvy in a 10-month-old boy. *Int J Dermatol*. 2007;46(2):194–198
87. Nishio H, Matsui K, Tsuji H, Tamura A, Suzuki K. Immunohistochemical study of tyrosine phosphorylation signaling in the involuted thymus. *Forensic Sci Int*. 2000;110(3):189–198
88. Shaw JC. Copper deficiency and non-accidental injury. *Arch Dis Child*. 1988;63(4):448–455
89. Marquardt ML, Done SL, Sandrock M, Berdon WE, Feldman KW. Copper deficiency presenting as metabolic bone disease in extremely low birth weight, short-gut infants. *Pediatrics*. 2012;130(3). Available at: www.pediatrics.org/cgi/content/full/130/3/e695-e698
90. Tümer Z, Møller LB. Menkes disease. *Eur J Hum Genet*. 2010;18(5):511–518
91. Bacopoulou F, Henderson L, Philip SG. Menkes disease mimicking non-accidental injury [published correction appears in *Arch Dis Child*. 2009;94(1):77]. *Arch Dis Child*. 2006;91(11):919
92. Paterson CR, Burns J, McAllion SJ. Osteogenesis imperfecta: the distinction from child abuse and the recognition of a variant form [see comment]. *Am J Med Genet*. 1993;45(2):187–192
93. Chapman S, Hall CM. Non-accidental injury or brittle bones. *Pediatr Radiol*. 1997;27(2):106–110
94. Paterson CR. Temporary brittle bone disease: fractures in medical care. *Acta Paediatr*. 2009;98(12):1935–1938
95. Paterson CR. Temporary brittle bone disease: the current position. *Arch Dis Child*. 2011;96(9):901–902
96. Miller ME. The lesson of temporary brittle bone disease: all bones are not created equal. *Bone*. 2003;33(4):466–474
97. Miller ME. Temporary brittle bone disease: a true entity? *Semin Perinatol*. 1999;23(2):174–182
98. Miller ME, Hangartner TN. Temporary brittle bone disease: association with decreased fetal movement and osteopenia. *Calcif Tissue Int*. 1999;64(2):137–143
99. Palacios J, Rodriguez JL. Extrinsic fetal akinesia and skeletal development: a study in oligohydramnios sequence. *Teratology*. 1990;42(1):1–5
100. Rivara FP, Parish RA, Mueller BA. Extremity injuries in children: predictive value of clinical findings. *Pediatrics*. 1986;78(5):803–807
101. Taitz J, Moran K, O'Meara M. Long bone fractures in children under 3 years of age: is abuse being missed in emergency department presentations? *J Paediatr Child Health*. 2004;40(4):170–174
102. Farrell C, Rubin DM, Downes K, Dormans J, Christian CW. Symptoms and time to medical care in children with accidental extremity fractures. *Pediatrics*. 2012;129(1). Available at: www.pediatrics.org/cgi/content/full/129/1/e128-133
103. Peters ML, Starling SP, Barnes-Eley ML, Heisler KW. The presence of bruising associated with fractures. *Arch Pediatr Adolesc Med*. 2008;162(9):877–881
104. Valvano TJ, Binns HJ, Flaherty EG, Leonhardt DE. Does bruising help determine which fractures are caused by abuse? *Child Maltreat*. 2009;14(4):376–381
105. Sugar NF, Taylor JA, Feldman KW; Puget Sound Pediatric Research Network. Bruises in infants and toddlers: those who don't cruise rarely bruise. *Arch Pediatr Adolesc Med*. 1999;153(4):399–403
106. Pierce MC, Kaczor K, Aldridge S, O'Flynn J, Lorenz DJ. Bruising characteristics discriminating physical child abuse from accidental trauma. *Pediatrics*. 2010;125(1):67–74
107. Shapiro JR, Sponsellor PD. Osteogenesis imperfecta: questions and answers. *Curr Opin Pediatr*. 2009;21(6):709–716
108. Marlowe A, Pepin MG, Byers PH. Testing for osteogenesis imperfecta in cases of suspected non-accidental injury. *J Med Genet*. 2002;39(6):382–386
109. American Academy of Pediatrics; Section on Radiology; American Academy of Pediatrics. Diagnostic imaging of child abuse. *Pediatrics*. 2009;123(5):1430–1435
110. Duffy SO, Squires J, Fromkin JB, Berger RP. Use of skeletal surveys to evaluate for physical abuse: analysis of 703 consecutive skeletal surveys. *Pediatrics*. 2011;127(1). Available at: www.pediatrics.org/cgi/content/full/127/1/e47-e52
111. American College of Radiology. *ACR Practice Guidelines for Skeletal Surveys in Children*. 2011. Available at: www.acr.org/~media/ACR/Documents/PGTS/guidelines/Skeletal_Surveys.pdf. Accessed May 5, 2013
112. Ingram JD, Connell J, Hay TC, Strain JD, MacKenzie T. Oblique radiographs of the chest in nonaccidental trauma. *Emerg Radiol*. 2000;7(1):42–46
113. Prabhu SP, Newton AW, Perez-Rossello JM, Kleinman PK. Three-dimensional skull models as a problem-solving tool in suspected child abuse. *Pediatr Radiol*. 2013;43(5):575–581
114. Karmazyn B, Duhn RD, Jennings SG, et al. Long bone fracture detection in suspected child abuse: contribution of lateral views [published online ahead of print October 6, 2011]. *Pediatr Radiol*. doi: 10.1007/s00247-011-2248-3
115. van Rijn RR. How should we image skeletal injuries in child abuse? *Pediatr Radiol*. 2009;39(suppl 2):S226–S229
116. Kleinman PK, Nimkin K, Spevak MR, et al. Follow-up skeletal surveys in suspected child abuse. *AJR Am J Roentgenol*. 1996;167(4):893–896
117. Bennett BL, Chua MS, Care M, Kachelmeyer A, Mahabee-Gittens M. Retrospective review to determine the utility of follow-up skeletal surveys in child abuse evaluations when the initial skeletal survey is normal. *BMC Res Notes*. 2011;4(1):354
118. Harlan SR, Nixon GW, Campbell KA, Hansen K, Prince JS. Follow-up skeletal surveys for nonaccidental trauma: can a more limited survey be performed? *Pediatr Radiol*. 2009;39(9):962–968
119. Mandelstam SA, Cook D, Fitzgerald M, Ditchfield MR. Complementary use of radiological skeletal survey and bone scintigraphy in detection of bony injuries in suspected child abuse. *Arch Dis Child*. 2003;88(5):387–390, discussion 387–390
120. Drubach LA, Johnston PR, Newton AW, Perez-Rossello JM, Grant FD, Kleinman PK. Skeletal trauma in child abuse: detection with 18F-NaF PET. *Radiology*. 2010;255(1):173–181
121. Rubin DM, Christian CW, Bilaniuk LT, Zaczynny KA, Durbin DR. Occult head injury in high-risk abused children. *Pediatrics*. 2003;111(6 pt 1):1382–1386
122. Wootton-Gorges SL, Stein-Wexler R, Walton JW, Rosas AJ, Coulter KP, Rogers KK. Comparison of computed tomography and chest radiography in the detection of rib fractures in abused infants. *Child Abuse Negl*. 2008;32(6):659–663
123. Brody AS, Frush DP, Huda W, Brent RL; American Academy of Pediatrics Section on Radiology. Radiation risk to children from computed tomography. *Pediatrics*. 2007;120(3):677–682
124. Stranzinger E, Kellenberger CJ, Braunschweig S, Hopper R, Huisman TAGM. Whole-body STIR MR imaging in suspected child abuse: an alternative to skeletal survey radiography? *Eur J Radiol Extra*. 2007;63(1):43–47
125. Perez-Rossello JM, Connolly SA, Newton AW, Zou KH, Kleinman PK. Whole-body MRI

- in suspected infant abuse. *AJR Am J Roentgenol*. 2010;195(3):744–750
126. Bachrach LK, Sills IN; Section on Endocrinology. Clinical report—bone densitometry in children and adolescents. *Pediatrics*. 2011;127(1):189–194
 127. Khoury DJ, Szalay EA. Bone mineral density correlation with fractures in non-ambulatory pediatric patients. *J Pediatr Orthop*. 2007;27(5):562–566
 128. Zemel BS, Kalkwarf HJ, Gilsanz V, et al. Revised reference curves for bone mineral content and areal bone mineral density according to age and sex for black and non-black children: results of the bone mineral density in childhood study. *J Clin Endocrinol Metab*. 2011;96(10):3160–3169
 129. Gordon CM, Baim S, Bianchi M-L, et al; International Society for Clinical Densitometry. Special report on the 2007 Pediatric Position Development Conference of the International Society for Clinical Densitometry. *South Med J*. 2008;101(7):740–743
 130. Henderson RC, Lark RK, Newman JE, et al. Pediatric reference data for dual x-ray absorptiometric measures of normal bone density in the distal femur. *AJR Am J Roentgenol*. 2002;178(2):439–443
 131. Lindberg DM, Shapiro RA, Laskey AL, Pallin DJ, Blood EA, Berger RP; ExSTRA Investigators. Prevalence of abusive injuries in siblings and household contacts of physically abused children. *Pediatrics*. 2012;130(2):193–201
 132. Hamilton-Giachritsis CE, Browne KD. A retrospective study of risk to siblings in abusing families. *J Fam Psychol*. 2005;19(4):619–624
 133. Asnes AG, Leventhal JM. Managing child abuse: general principles. *Pediatr Rev*. 2010;31(2):47–55
 134. Banaszkiwicz PA, Scotland TR, Myerscough EJ. Fractures in children younger than age 1 year: importance of collaboration with child protection services. *J Pediatr Orthop*. 2002;22(6):740–744

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