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SYMPOSIUM: NONACCIDENTAL TRAUMA IN CHILDREN

Unexplained Fractures: Child Abuse or Bone Disease

A Systematic Review

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Abstract

Background Child abuse and neglect (CAN) is a serious problem that has major implications for the welfare of the child involved. Unexplained fractures are of particular concern to the orthopaedic surgeon, who must often consider alternative diagnoses to CAN.

Questions/purposes We therefore (1) determined which bone diseases most commonly mimic CAN; (2) what types of osteogenesis imperfecta (OI) are most commonly confused with CAN and why; and (3) what specific findings in OI and bone disease render a mistaken diagnosis of CAN more likely.

Methods A systematic review of the literature was performed. We identified studies that compared cases of CAN with cases in which patients had bone disease that resulted in an unexplained fracture. We also included studies in which patients with fractures resulting from underlying bony pathology were misclassified as CAN and were subsequently reclassified as bone disease as a result of further investigation. Our search netted only five studies that directly compared and contrasted CAN with metabolic or genetic bone disease in the same study.

Results The published literature suggests OI is most frequently confused with CAN, although metaphyseal dysplasia, disorders of phosphate metabolism, and temporary brittle bone disease are also documented in the literature identified by our search. Difficulty in differentiating these bony diseases from CAN stems from ambiguity in the history and physical examination at the time of presentation.

Conclusions Bone disease is a diagnosis of exclusion in the differential diagnosis of CAN.

Introduction

Child abuse and neglect (CAN) is a serious problem, and an improper diagnosis carries tremendous consequences for the patient, his or her family, and the doctor. A substantial degree of morbidity and mortality is associated with a missed diagnosis, with a reinjury rate of 50% and a mortality rate of 10% for unprotected children [6]. In addition, the emotional trauma for the patient and family undergoing a prolonged investigation by Child Protective Services (and perhaps even the wrongful removal of custody) demonstrates the power of a misguided diagnosis of abuse [21, 22]. With fractures representing the second most common manifestation of child abuse after soft tissue trauma [14], orthopaedic surgeons are at the forefront of evaluating patients with potential CAN.

Many studies have described the musculoskeletal manifestations of child abuse: rib fractures, skull fractures, humeral and femur fractures are among the most common [2, 7, 10, 12, 14–17, 19, 24, 33]. Recent studies

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demonstrated that the combination of patient age and fracture location can reproducibly differentiate abuse from accidental trauma [19]. Among these factors, a suspicious history, physical or radiographic evidence of prior injury, and age younger than 18 months are associated with CAN [1].

In 1962, Kempe et al. introduced the concept of "battered child syndrome." The authors included osteogenesis imperfecta (OI) as part of the differential diagnosis of a patient presenting with potential abuse [11]. The authors noted that OI should be easily distinguishable from abuse based on history and physical examination. Despite their assertions, there is little evidence to suggest clinicians can readily differentiate patients with OI (and other metabolic bone disorders with a propensity for fracture) from those who have sustained their fractures as a result of CAN [21–23]. Although patients with OI/metabolic bone disease can present with the "classic" features of OI (multiple fractures after minor trauma at an early age, blue sclera, osteopenia, wormian bones, dentinogenesis imperfecta, and a family history of "easy" fractures and/or bone disease [21–23, 29]), many times these features are not present [22, 23, 29]. In these cases, it is not clear how to further investigate the presence of these rare conditions in a cost-effective and directed manner for patients presenting with potential abuse. Unfortunately, the burden falls on the clinician to distinguish between these conditions with limited data to guide the decision-making process. Although there is an abundance of literature differentiating abuse from accidental trauma, there exists relatively little work on the role that bone diseases such as OI play in the evaluation of a patient with suspected abuse.

Therefore, we addressed the following questions: (1) What diseases and conditions that cause easy fracture can be confused with CAN? (2) What types of OI are most commonly confused with CAN and why? (3) What specific findings in OI and bone disease render a mistaken diagnosis of CAN more likely?

Materials and Methods

We searched the Medline and EMBASE computerized literature databases from January 1950 to June 2009. Articles were identified using an electronic search of keyword terms and their respective combinations (Table 1). The search was guided by our institution's research librarian on August 22, 2009 . A total of 104 unique references were identified with this search. Reference lists from the articles retrieved were further scrutinized as well to identify any additional studies of interest (Fig. 1). All studies from the mentioned searches were then reviewed. Studies were included in this systematic review if they matched the following criteria: (1) they were in English language; (2) they had a Level I, II, III, or IV study design by "Journal of Bone and Joint Surgery" criteria (http://www.2.ejbjs.org/misc/instrux.dt levels); (3) they had a minimum of 10 patients in their series with bony injury; (4) both patients with CAN or a condition that results in a bone disease with a propensity for fracture with minimal trauma were present in the study or patients were included who were diagnosed with CAN that subsequent investigation revealed to be bone disease; and (5) the patients were younger than 18 years old, or older patients could not be distinguished from younger ones in the body of the text or tables. We excluded review articles, case reports, or studies in which the inclusion criteria were not explicit. Two authors performed the initial search (AK, KB) and then three of the authors (AK, KB, NP) independently reviewed the 104 references and selected the appropriate studies based on these criteria. If one or more author selected a paper, it moved on to the next phase; in the final phase of review (elimination by full text review), there was

Table 1. This list includes search terms entered into the Medline and EMBASE search engines for identification of human studies in English from the year 1950 to June 2009

Number	Search term
1	exp Child Abuse/
2	exp Shaken Baby Syndrome/
3	exp Brain Injuries/
4	1 and 3
5	nonaccidental trauma.mp.
6	exp Osteogenesis Imperfecta/
7	unexplained fractures.mp.
8	exp Bone Diseases, Metabolic/
9	brittle bone.mp.
10	battered child.mp.
11	1 or 10 or 2
12	4 or 7 or 5
13	8 or 6 or 9
14	11 and 12
15	11 and 13
16	13 and 12
17	limit 16 to (English language and humans and "all child [0 to 18 years]")
18	14
19	limit 18 to (English language and humans and "all child [0 to 18 years]")
20	15
21	limit 20 to (English language and humans and "all child [0 to 18 years]")
22	(1 or 5 or 7 or 10) and (6 or 8 or 9)
23	limit 22 to (English language and humans and "all child [0 to 18 years]")

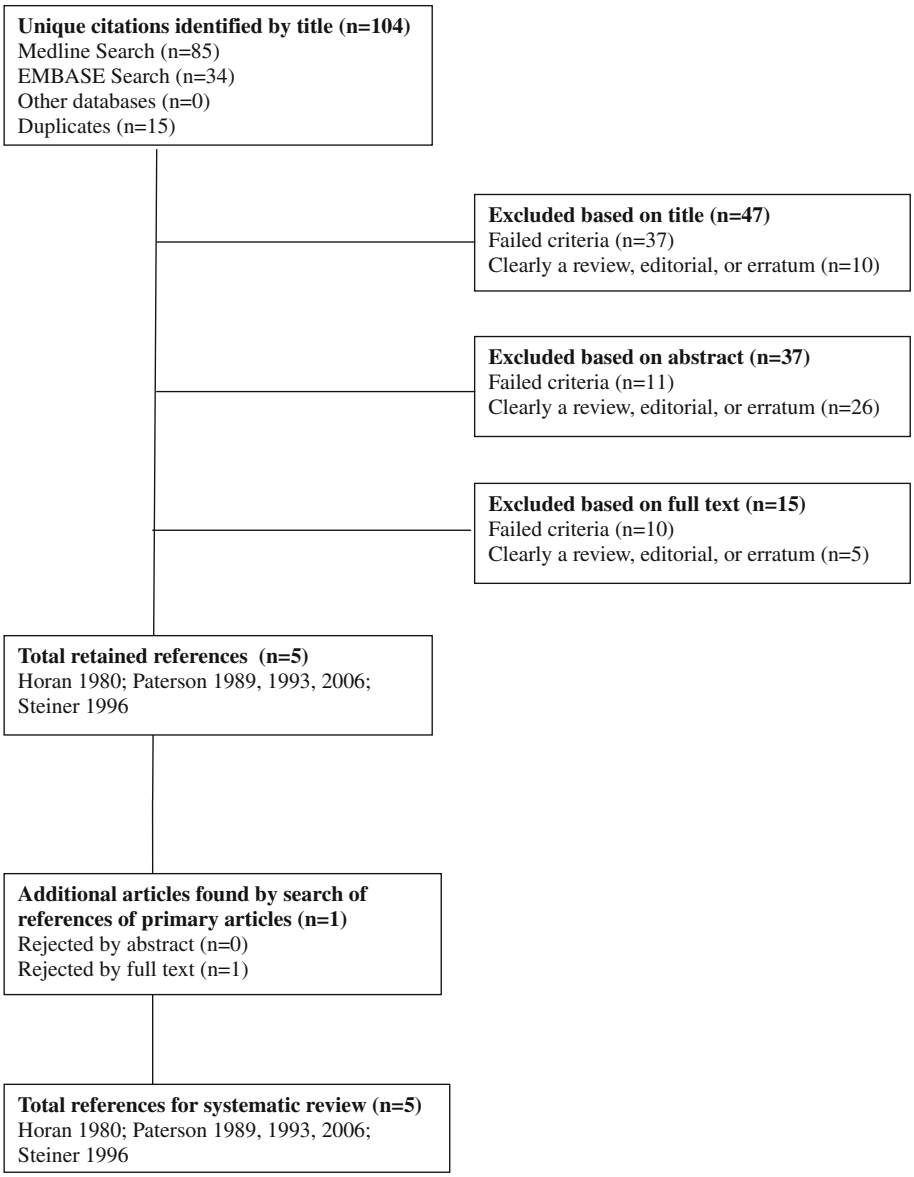

Fig. 1 This flow diagram presents the systematic review process used in this study.

no disagreement over which papers would be ultimately included (Fig. 1).

We obtained 85 articles from Medline and 34 articles from EMBASE for a total of 119 articles from these searches. After removal of 15 duplicate studies, a total of 104 papers were reviewed from the combined MedLine and EMBASE searches. Duplicates were electronically confirmed with use of RefWorks bibliographic software (ProQuest, LLC, Bethesda, MD). We initially excluded 47 articles by title for irrelevance to the topic in question, if they were picked up by chance, or if they were designated as reviews, editorials, or commentaries. An additional 37 articles were eliminated after reviewing the abstract; we eliminated articles by abstract only if they were case reports, erratum, reviews, or small case series (less than 10 study subjects). If there was any question over inclusion, we undertook a review of the full text of the work. We then reviewed the full text of the remaining 20 articles, of which 15 articles failed to meet our inclusion and exclusion criteria. This systematic review left five articles for analysis [8, 21–23, 29] (Appendix 1). The references of these articles (along with the prior individual searches) were manually searched for other potential articles of interest. One further article was identified in the references, but this article was eliminated because it only had two patients with bony injury [32].

Study quality was assessed by considering controls for bias, confounding, and chance within each study as suggested by the MOOSE group for meta-analysis of observational studies [30]. No study had a control group to control for chance; although some rudimentary statistical analyses were used, no study used logistic regression to

control for confounding factors. No blinding was used, and no controls for selection bias were present in controlling for the sample population.

Pooling the identified studies resulted in a total of 914 patients, 155 of whom represented cases mistakenly diagnosed as CAN. The most common condition mistaken for CAN was OI with 80% of our articles meeting criteria that dealt with this diagnosis [21–23, 29]. Pooling the studies in which confirmed cases of metaphyseal dysplasia/OI were present, there was an 18.7% (162 of 866) rate of initial misdiagnosis of CAN or bone disease [8, 21–23] and a 12.5% (six of 48) rate of OI in the study in which all children with suspected CAN were examined [29]. The mean age at which patients with OI were mistakenly diagnosed with CAN at the time of first fracture (data available for 26 patients) was 8.38 ± 7.76 months [22, 23].

Because the outcomes, design, and (in many cases) the study population of each study were too heterogeneous to compare directly, even with random effects models, we used data from individual studies to generate statistics (Table 2). In cases in which the data were similar between studies, we pooled the results for the purposes of generating percentages, but no statistics were generated because of concerns for heterogeneity. Additionally, because none of these studies produced effect sizes, forming some quantitative measure of heterogeneity would be arbitrary. Within each individual study to test hypotheses that findings were independent of etiology versus the alternative that they were related, the chi square test with Yates correction or the Fisher's exact test was used. These statistics were calculated using Java stat two-way contingency table analyses [5].

Results

Although many case reports and larger reviews suggest several conditions, including biliary atresia [3], dietary deficiencies [4, 28], rickets [9, 20], metabolic bone disease [31], and copper deficiency [25], can mimic CAN, the five articles that met our inclusion criteria only discussed metaphyseal dysplasias and OI as possible diagnoses that may be confused with CAN [8, 21–23, 29].

Sillence Type IV OI [26, 27] (Table 3) was most frequently mistaken for CAN [22, 23]. From the studies in our review, 25.5% (44 of 172) of patients with Type IV OI (compared with only 13.1% [77 of 589] of patients with other OI types) were confused with CAN [22, 23]. This corresponds to an odds ratio of 2.2 (1.4–3.4) for a greater likelihood of confusion of Type IV OI with CAN as

Table 2. Data for articles examining the etiology of children's presenting fracture*

Study lead author	Year	Journal	Initial misdiagnosis of CAN	Number of confirmed CAN	Number of confirmed bone disease	Pathology
Horan and Beighton [8]	1980	*J Bone Joint Surg Br*	N/A	7	6	Metaphyseal dysplasia; hypo-/hyperphosphatasia
Paterson and McAllion [22]	1989	*BMJ*	111*	0	802	OI (various types)
Paterson et al. [21]	1993	*Am J Med Genet*	32†	0	39	Temporary brittle bone disease/ OI
Steiner et al. [29]	1996	*J Pediatr*	N/A	42	6	OI
Paterson and McAllion [23]	2006	*Clin Orthop Relat Res*	12‡	0	12	OI

* One hundred eleven parents were accused of CAN at some point in the child's illness; most parents were vindicated in 1 week or less, 15 went to formal litigation, and one case was prosecuted but later acquitted; †32 patients in this series had an initial diagnosis of CAN; the remaining seven had an initial diagnosis of OI; ‡12 patients were initially believed to have fractures resulting from CAN but were later discovered to have OI; CAN = child abuse and neglect; OI = osteogenesis imperfect; N/A = not applicable.

Table 3. Sillence types of OI*

OI type	Clinical features	Inheritance pattern
I	Normal stature, little bony deformity, blue sclerae, 50% incidence of hearing loss	AD
II	Lethal in the perinatal period	AD; AR (rare)
III	Progressive deformity, moderate at birth, hearing loss, short stature, variable sclera	AD; AR (uncommon)
IV	Normal sclera, mild deformity, variable short stature, variable hearing loss	AD

* Further subclassification can be made within each OI type: subgroup A notation indicates no dentinogenesis imperfect; subgroup B notation indicates the presence of dentinogenesis imperfecta [26]; OI = osteogenesis imperfecta.

opposed to non-Type IV (I-III) forms. Furthermore, nine of these patients with Type IV (5.3%) versus only six (1.1%; $p < 0.001$) of other OI types led to legal proceedings [22].

The difficulty in differentiating CAN from bone disease is complicated by marked variability in the manifestations of "classic" findings. Three of our articles described patients with OI who had an atypical history and physical findings such as a lack of osteopenia (three of four) [29], lack of wormian bones (nine of 28) [22, 23, 29], and a lack of OI family history (21 of 33) [22, 23, 29]. Furthermore, the lack of dentinogenesis imperfecta has been suggested as a possible reason that milder forms of OI are mistaken for CAN [21]. However, the largest series in our review (with 802 total cases) [22] reported no difference between the groups of patient who were initially misdiagnosed with CAN as opposed to OI in terms of the presence of dentinogenesis imperfecta. Data were also pooled from these articles for the scleral color of these patients and family history (at the time of presentation with fracture) for the patients who were initially misclassified as CAN [22, 23, 29]: 72.7% (24 of 33) of patients had an abnormal scleral color (blue, pale blue, or gray) [22, 23, 29], 50% (three of six) of patients had a family history of multiple fractures [29], and 36.4% (12 of 33) of patients had a family history of OI yet were still misclassified as CAN victims [22, 23, 29]. Substantial data examined fracture location in patients with OI misdiagnosed as CAN. From those papers [22, 23], 18 of 27 patients with OI presented with femur fractures, nine with tibia fractures, eight with humerus fractures, four with clavicle fractures, and four with rib fractures. Patients with OI either had multiple fractures on presentation or had radiographic evidence of old fractures in 19 of the 27 patients [22, 23].

In addition to fracture location, bone quality was considered by a subset of papers in our study. Patients with "temporary" brittle bone were noted to have metaphyseal abnormalities 76% of the time, rib fractures in 72% of cases, and periosteal reaction without fracture in 49% of cases [21]. Patients in the metaphyseal dysplasia series had a variety of historical and radiographic signs such as bowing of long bones, diaphyseal sclerosis, and epiphyseal separation; these findings, along with notes that suggested trauma, led the authors to reclassify them as CAN [8].

Discussion

Child abuse is frequently encountered by orthopaedic surgeons, because fracture is the second most common presenting symptom [14]. Although extensive literature has described the injury patterns in abusive versus accidental trauma [7, 10, 12, 14–19, 24, 33], there has been a dearth of literature differentiating pediatric fractures caused by bone disease versus CAN; only one article [23] from this decade met our inclusion and exclusion criteria. The morbidity and mortality of a missed diagnosis of CAN has been well documented [6], although the emotional, psychologic, and financial burdens to both the family and child of false CAN accusations (in the context of underlying bone disease) can be presumed high as well. This systematic review sought to answer the following questions. First, what other conditions mimic CAN with enough regularity that they are represented in the literature to warrant a case series? Second, what types of OI are most frequently mistaken for CAN and why? Third, what specific features of underlying bone disease increase the likelihood of mistaken CAN?

The limitations of our study and the literature are not trivial. First, there is no gold standard test for diagnose CAN; it remains a synthesis of history, physical examination, and radiographic findings. In many instances, a physician's own instincts make the diagnosis. Therefore, any study of CAN is fraught with the danger of misclassification. Second, the literature directly comparing bone diseases with CAN is sparse. As such, it is difficult, if not impossible, to describe the data in quantitative terms. Third, the methodology of the papers reviewed does not provide controls for bias confounding or chance; articles were mainly descriptive in nature. As such, our study is simply an observational study of observational studies but does provide an overview of the literature available, which directly compares the two conditions. Weaknesses inherent to the member studies are not improved by aggregating them.

The condition that was most frequently mistaken for CAN was OI, although one additional included article examined metaphyseal dysplasias. The most common form of OI mistaken for CAN was Silence Type IV. With the information as to what general condition (OI and a specific type) most commonly mimics CAN, it would seem logical that these readily distinguishable history, physical examination, and imaging characteristics can aid in making the proper diagnosis. However, patients with OI who were mistakenly considered victims of CAN had a wide range of history, physical examination, and radiographic findings, particularly for features considered "typical" for OI [22, 23, 29]. In fact, patients in our series with OI who were wrongly diagnosed with CAN lacked "classic" osteopenia and wormian bones. This could potentially explain why these patients' caretakers were wrongly accused of CAN.

The difficulty in diagnosing OI may stem from the fact that fracture patterns in the patients with OI did not differ from those in patients with CAN. From papers presenting fracture data in our review [22, 23], femur fractures were the most common (66.6%) in patients with OI followed by tibia (33.3%), humerus (29.6%), and rib fractures (14.8%). In addition, in 70.3% of cases, patients with OI had

multiple fractures or radiographic evidence of old fractures on presentation [22, 23]. This is similar to data presented in prior studies in patients younger than 18 months; the odds of rib, tibia/fibula, humerus, and femur fracture are higher in patients with CAN, and multiple fractures and fractures in multiple stages of healing are indicative of CAN [14, 19]. Furthermore, Steiner et al. observed no difference between patients with OI (33%) and those with CAN (39%) in terms of the presence of fractures with a "high" specificity [13] for abuse (ie, metaphyseal, posterior rib, scapular, spinous process, or sternal fractures) or for fractures with a "moderate" specificity for abuse as outlined by the authors [29].

The lack of dentinogenesis imperfecta has also been suggested as a possible reason some milder forms of OI can be mistaken for CAN [21]. However, the largest series in our review [22] found no difference between the groups of patients who were initially misdiagnosed in terms of the presence of dentinogenesis imperfecta. Furthermore, bone quality was examined by a subset of papers in our study. A similar problem existed for patients with metaphyseal dysplasia, in which mistaken diagnoses were made in the setting of heterogeneous historical and radiographic signs [8]. Clearly, these results challenge the notion that patients with OI are mistaken for victims of CAN as a result of the fact that they are "atypical" from the common presentation of OI.

In summary, there are no high-quality studies examining the differences between underlying bone disease and CAN. Based on this review, OI (in particular Sillence Type IV) is the most common condition mistaken for CAN. Patients with OI wrongly diagnosed with CAN have fracture patterns that are indistinguishable from patients with CAN, and they may or may not have "classic" findings of OI. Well-designed case-control studies might address some of the injury patterns observed in CAN and compare them directly with one or more bone diseases. Furthermore, currently, bone disease is a diagnosis of exclusion, and current recommendations are that the prevalence in the general population is too low for the test to be useful unless clinical suspicion exists. Ultimately, the clinician should have OI/metaphyseal dysplasia in the differential diagnosis of child abuse. Although clearly, examining dentition, radiographs of the skull for wormian bones, scleral examination, and personal or family history of easy fractures can be helpful, these conditions are inconsistently present and may miss cases of bone disease. Clinicians must keep child welfare as the top priority; because child abuse is more common and is a high morbidity and mortality condition, bone disease remains a diagnosis of exclusion.

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Appendix 1

Summary of CAN Versus Bone Disease Articles

Horan and Beighton in 1980 reviewed material in Sir Thomas Fairbank's collection of metaphyseal dysplasias and dysostoses in the radiology museum of the Royal National Orthopaedic Hospital in London [8]. They were able to review long-term fracture followup information on 13 patients. Using criteria for "battered-child syndrome" established by Kempe et al., the authors set out to determine if the true diagnosis in these patients was CAN (child abuse and neglect) or metaphyseal dysplasia [8, 11]. The authors do not describe their specific criteria, and Kempe in his original article is nebulous, saying that "The battered-child syndrome may occur at any age, but, in general, the affected children are younger than 3 years. In some instances the clinical manifestations are limited to those resulting from a single episode of trauma, but more often the child's general health is below par, and he shows evidence of neglect including poor skin hygiene, multiple soft tissue injuries, and malnutrition. One often obtains a history of previous episodes suggestive of parental neglect or trauma. A marked discrepancy between clinical findings and historical data as supplied by the parents is a major diagnostic feature of the battered-child syndrome" [8, 11]. Using these criteria, the authors found a 53.8% (seven of 13) misdiagnosis rate. Whereas six of the patients had the correct diagnosis of metaphyseal dysplasia as their fracture etiology (including two with Jansen dysplasia, one with hyperphosphatasia, one with Schmidt-type chondrodysplasia, one with spondylometaphyseal dysplasia, and one with hypophosphatasia), seven were actually found to have sustained fractures resulting from CAN, not underlying bone disease.

Paterson and McAllion reviewed a series of 802 cases with the confirmed diagnosis of OI (osteogenesis imperfecta) [22]. The purpose of their study was to determine the extent to which these children initially were believed to have sustained nonaccidental trauma on presentation with a fracture. They found in 691 of 802 (86.1%) cases there were no difficulties with the diagnosis of OI, in 96 of 802 (11.9%) cases there was at least one accusation of CAN, which resulted in an investigation of less than 1 week (clearing the caretakers of abuse), and in 15 of 802 (1.8%) cases, CAN was again suspected yet it led to prolonged proceedings (case conferences, social work meetings,

police involvement, etc). The greatest percentage of these accusations of CAN were seen in patients with Type IV Sillence-type OI (23.3% [39 of 167]) [22].

In a second article, Paterson et al. examined a variant form of OI, which they termed "temporary brittle bone disease" [21]. In this study, they examined 39 patients with fractures that transiently occurred in the first year of life (with cessation of fractures after this time period). In seven of 39 (17.9%) of the patients, the initial diagnosis was OI, whereas the rest were diagnosed incorrectly with CAN. These patients with temporary OI shared radiographic characteristics such as metaphyseal abnormalities (76%), rib fractures (72%), diaphyseal fractures (57%), periosteal reaction without fracture (49%), delay in bone age (35%), expanded costochondral junctions (34%), and overt osteopenia (31%). In addition, many of these patients clinically had vomiting (71%), diarrhea (50%), apnea attacks (32%), hepatomegaly (35%), and hemoglobin less than 10 g/dL (40%).

A third article by Paterson et al. examined 12 patients with OI who were initially diagnosed with CAN when in fact the true cause of their fractures was underlying bone disease [23]. The purpose of this study was to identify the clinical and radiographic factors that led to the initial incorrect diagnosis of CAN (and the removal of seven of 12 children from their homes). The presence of unexplained fractures in different stages of healing, apparently normal bone density on radiographs, negative family history, non-diaphyseal fractures, lack of wormian bones, normal anterior fontanelles, and normal scleral color were all factors found in these patients, which were atypical of patients with OI. They concluded OI should always be considered in the differential of suspected cases of CAN even with the lack of "typical" features; it is in the absence of these typical features that a diagnosis can be missed.

Finally, an article by Steiner et al. aimed to determine whether analysis of collagen synthesized by dermal fibroblasts could help to correctly identify children with OI among a group of children suspected to have been the victims of CAN [29]. The authors examined 48 children who had been referred to them to specifically distinguish OI from child abuse. Examining for the COL1A1 gene, the authors found an OI rate of 12.5% (six of 48) in children with suspected CAN in whom the diagnosis of OI was entertained. In 83.3% (five of six) of the children with biochemically proven OI, the diagnosis of OI was strongly suspected on clinical grounds before the diagnosis. These clinical findings included blue sclera (83%) and a family history of fracture (50%), whereas osteopenia (25%), wormian bones (0%), and a family history of OI (0%) were not as clinically reliable. The authors concluded routine biochemical evaluation of all patients with suspected abuse for OI is not warranted because the determination can be largely made based on history and physical examination. In cases in which diagnostic uncertainty remains, biochemical studies may be a useful adjunct.

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