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LETTER TO THE EDITOR

Commentary on "congenital rickets" article

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Sir,

I am concerned that the clinical and radiologic details reported by Drs. Kathy Keller and Patrick Barnes [1] in their discussion of "congenital rickets" ignore important clinical and radiologic findings of the cases, while reporting radiologic findings that none of the radiologists I have consulted can see.

I have personal knowledge of three of the article's cases. In case 1, I provided the clinical child abuse evaluation and subsequently was called to testify in the child's dependency case. In case 4, I was consulted by the attorney general who was handling the child's dependency and subsequently by the prosecutor pursuing the criminal matter. I also assisted our neurosurgeons in the clinical management of this child, after she was referred to our institution for follow-up care. In case 2, both Dr. Keller and I were hired by the family's defense attorney to evaluate and possibly testify in the dependency. Because of my relationship with this case, through the family's defense attorney, I am not at liberty to share the case material. However, Dr. Keller and I disagreed about that child's injuries and only she was used by the attorney in court. I was personally paid for my legal consultations in case 4. I was partially paid through an initial retainer in case 2 and my hospital's practice group was paid in case 1. I do not have personal knowledge about case 3, but the physicians at Cincinnati Children's Hospital who cared for that child have informed me that that child's findings were also misrepresented.

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Some specifics of their omissions and commissions include case 1. This child was born in mid-July to a Caucasian mother who had been taking prenatal vitamins. The child sustained abusive fractures at 3 months of age. They reported that the child's mother's vitamin D level was 8.7 ng/ml. However, they failed to disclose that the mother's vitamin D level was not determined until late November. Dr. Keller extensively used a paper by Bodnar et al. [2] at this child's dependency trial to support the existence of vitamin D deficiency as a common problem in mothers at term. Intrapartum vitamin D deficiency is a well-established condition, as documented by such current literature. Dr. Keller, however, did not disclose either in court or in the paper that this same article points out that mothers' vitamin D levels in the summer are on the average significantly greater than mid-winter levels (23.2 nmol/ml higher on average for Caucasian mothers), which would have put this mother's vitamin D level at the time of her infant's birth in the normal range (roughly 50 nmol/l). Also not mentioned is that Bodner et al. report that vitamin D levels in Caucasian mothers delivering during the summer, after taking prenatal vitamins, are rarely deficient (none <37.5 nmol/l) but are fairly often insufficient (40% between 37.5 and 80 nmol/l). More commonly, 30 nmol/l is used as the threshold for vitamin D deficiency.

Infant 1 had been evaluated for a swollen right lower leg. She had been primarily breast-fed and was growing subnormally. Her physical examination was notable for multiple palpable healing fractures, but no bruises. Father had noted her right shoulder popped when he had picked her up 1 month previously; she had subsequently favored that arm. Her calcium (10.1 mg/dl) and phosphate (6.3 mg/dl) were normal, but her alkaline phosphatase was mildly elevated at 483 U/l. Her cranial CT scan and eye examination were normal.

This child had multiple fractures, including: a healing right spiral/comminuted midshaft tibia fracture, healing right transverse distal radius and ulna diaphyseal fractures, periosteal layering of the distal right humerus (indicating a healing supracondylar injury), a sclerotic band of the distal left radius (reflecting a more remote fracture), possible proximal and distal left femur metaphyseal fractures, a proximal right first metacarpal fracture, a transverse fracture of the proximal right second phalanx, a fracture of the right proximal third phalanx, a fracture of the base of the proximal phalanx of the left third and fifth fingers, sclerosis of the mid-portion of the proximal phalanx of the left second toe, classic metaphyseal fractures (CMLs) of the proximal right tibia and distal left tibia, posterior rib fractures of the left ninth and tenth and right tenth ribs, anterior-lateral right fifth and sixth rib fractures, a heavily callused proximal right clavicle fracture and anterior wedging of the vertebral body of T8. The child's mineralization looked normal, as did her metaphyseal mineralization, although her bones seemed somewhat denser on a follow-up skeletal series. Other abnormalities Barnes and Keller describe in the figures including abnormal sutures and costochondral junctions were considered normal on review by the radiologists I consulted. The distal forearm changes they describe simply reflected fractures in various stages of healing. They were correct in stating that the child's injuries were "interpreted as … suspicious for inflicted injury." In fact, we thought they were diagnostic of abuse.

The child was subsequently evaluated in our metabolic and genetic bone disease programs, and it was thought there was no indication of osteogenesis imperfecta or metabolic bone disease. Her skin biopsy for osteogenesis imperfecta was normal.

Mother's bone mineral density that November was 2.3 standard deviations below normal. Her 1,25-dihydroxyvitamin D was normal (25.7 nmol/l) in the end of November. Her calcium was normal (9.9 mg/dl).

In case 4, their statement that the child presented without evidence of increased intracranial pressure is true but ignores other significant cranial clinical and imaging findings. The child's presenting symptoms included a single reported low-grade fever at home (not confirmed or repeated), decreased feeding and activity, decreased use of her arms, sleepiness and congested/grunting respiration. There was a history of a fall 3 days before off father's chest, when he was resting on a couch, to the adjacent floor. She was initially thought to have a respiratory infection or sepsis, but after healing rib fractures were noted on chest radiographs, a CT, MRI and skeletal survey were done. Her lethargy and respiratory abnormalities were then recognized to be symptoms of an acute brain injury and she was found to have arm paralysis from a cervical spinal cord injury (central cord syndrome).

Keller and Barnes did not mention the child's acute cervical cord hemorrhage/injury on MRI, which caused the arm paralysis. They inferred that the child had an isolated cervical vertebral body compression fracture, secondary to a weak vertebral body sustaining normal forces. They failed to report that this vertebral compression fracture was associated with retrolithiasis of C4 on C5 and acute central cervical cord hemorrhage, extending well up into the cerebellar peduncle. These findings indicate significant trauma, not simple vertebral collapse. The child's old subdural hemorrhage, which they minimize by noting its size was small, was clinically and diagnostically significant for prior inflicted head injury. They did not report the child's acute subdural hemorrhage. Though they reported that she had skull and cervical spine fractures, they did not point out that the skull fractures were independent, involving both parietal bones. One had and one lacked soft-tissue swelling, strongly suggesting recent and older skull trauma. The child's skeletal injuries were also highly indicative of abuse. Included were CMLs of the left tibia, distal right radius, proximal right humerus, distal right acromion, distal right and left femurs, and distal left tibia. There were posterior rib neck fractures of the right second and left third and left eighth ribs, postaxillary line fractures of the left sixth and seventh ribs, and possibly a left fifth costochondral fracture. All rib fractures showed evidence of healing. The distal third to fifth right metatarsals were irregular compared to the left; although, in the end our radiologists thought these had not been fractured. This child has been reported elsewhere in detail (as case 1) [3].

In cases 1, 3 and 4 they reported metaphyseal changes they considered significant for rickets, "Looser zones," frayed distal radial metaphysis and indistinct distal ulnar metaphyses. I have submitted films of children 1 and 4 to several pediatric radiologists, who confirmed the absence of such changes. In fact, the editors of *Pediatric Radiology*, in an accompanying editorial, opined, "The fractures are mainly that – fractures. The areas in which one expects to see signs of rickets in this age group are normal" [4]. Likewise, Barnes and Keller report metabolic skull changes that are not confirmed by other pediatric radiologists. They failed to note that classical metaphyseal lesions are the result of traction and torque forces, atypical of forces that normal infants experience, and that these fractures are not of the character that occur with weak bones, when failing to resist normal forces. They failed to note the high reported specificity for abuse of CMLs and fractures of the posterior rib neck, acromion, proximal humerus, proximal clavicle and digits in infants [5].

Neither Dr. Keller nor Dr. Barnes provided clinical care to these children. All the cases came to them as defense witnesses in the legal system. It is a serious breach of research bias for them to not disclose the source of their

case material in their article. Likewise, both physicians are active as defense witnesses in child abuse cases. It is a serious breach of conflict of interest to not disclose in their article that they profit personally from promoting the existence of congenital rickets as legitimate disease and as an explanation for multiple fractures in young infants.

In case 2 the dependency judge ruled that the child had been abused and opined that there was sufficient evidence for a criminal conviction. In case 4 the child was ruled in dependency court to have suffered abuse and in criminal court the child's father was convicted of the abuse. Case 3 was placed in a relative's care under court-ordered dependency. In case 1 the dependency judge ruled in favor of the parents, largely on the basis of Dr. Keller's testimony and quirks of how the trial proceeded. A different judge heard the defense's case about a half-year after the state's case was presented. There was no opportunity to present rebuttal of Dr. Keller's testimony. In any case, it is a serious breech of ethical disclosure, even if Keller and Barnes disagreed with those outcomes, to assert in their article that these cases prove the existence of congenital rickets as a cause of infant fractures, without disclosing that bodies as significant as the trial courts deemed their opinions to be incorrect.

I am concerned that Drs. Keller and Barnes' congenital rickets paper, if used in court without rebuttal, promotes a rare condition as a cause of multiple fractures in early infancy. In order for maternal vitamin D deficiency to really explain infant injuries, there must be an unbroken chain of causation from their vitamin D-deficient mothers to vitamin D deficiency explaining all of the child's injuries. In the cases Keller and Barnes reported, this chain of evidence was clearly lacking. Use of their unsupported theory in court is likely to put many children in harm's way.

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