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LETTER TO THE EDITOR

Another perspective on the cause of metaphyseal fractures

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Sir,

I read with interest the article by O'Connell and Donoghue [1] that describes three newborn infants with classic metaphyseal fractures (CMLs) following cesarean delivery. All of the CMLs were diagnosed radiographically or by clinical features on day 2 of life. Two of the infants were in the breech presentation, and in the third case the mother had poorly controlled gestational diabetes. While the authors attribute these fractures to twisting and pulling of the extremities during delivery, and possibly to large size (all three infants weighed >3.45 kg), there are many such infants born in a similar fashion who never show any evidence of bone fractures. The critical issue is whether these three infants may be predisposed to having lower newborn bone strength, and I would suggest that fetal immobilization is likely the common thread to understanding such a predisposition in these three infants.

Application of the Utah paradigm to the in utero development of fetal bone strength would suggest that bone loading through fetal movement is critical to the realization of normal bone strength at the time of birth [2]. Situations that diminish fetal movement will decrease bone strength and include the following: cephalopelvic disproportion, malpresentation (including breech presentation), twin pregnancy, prematurity, oligohydramnios, large maternal fibroids, and maternal use of medications that can cause decreased fetal movement [3, 4]. Since two of the three infants were in the breech presentation, it is likely that they may have been confined and moved less than normal while in utero.

It is known that infants of diabetic mothers have a lower bone mineral content at birth compared to controls as a result of increased bone resorption [5, 6]. It is also known that infants of diabetic mothers have decreased cyclic movements compared to normal controls, and this relative fetal immobilization would explain the osteoclast-mediated osteopenia in infants of diabetic mothers [7].

Case reports such as those in the article by O'Connell and Donoghue support the existence of a transient brittle bone state from fetal immobilization that can lead to the same types of fractures as seen in child abuse [1, 8]. CMLs associated with physical therapy in the treatment of clubfeet have also been described in this journal, and it has also been noted that some of these cases were also associated with fetal immobilization [9, 10]. The idea of decreased fetal bone loading leading to decreased bone strength has a scientific foundation based on experimental observations and is in accord with contemporary thinking of bone physiology [2, 8, 11, 12].

The authors imply that CMLs are specific for abuse unless there is a prior history of accidental injury. I disagree with this position. In the cases they present, what would have happened if the swelling and leg abnormalities were not noted until after the newborn infants had been discharged from the hospital and were in the care of the parents? Child abuse would have been alleged against the parents. There is a differential diagnosis for metaphyseal fractures that includes copper deficiency, Menkes disease, scurvy, hyperparathyroidism, osteogenesis imperfecta, temporary brittle bone disease from fetal immobilization, physical therapy for clubfoot, breech presentation with/without external version, the bone disease of prematurity, fetal exposure to magnesium, and child abuse [1, 13, 14].

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