Correspondence

Acute life threatening events associated with hypocalcemia and vitamin D deficiency in early infancy. *A single center experience from the Kingdom of Saudi Arabia*

To the Editor

I have 2 comments on the interesting study by Mosalli et al[1] on the acute life threatening events (ALTE) associated with hypocalcemia and vitamin D deficiency in early infancy.

First, stridor is an acute emergency state that is well-known to be caused by hypocalcemia.[2] It should be considered among the inclusive criteria of ALTE associated with hypocalcemia and vitamin D deficiency addressed in Mosalli et al's study.[1]

Second, astute pediatricians working in the emergency departments must suspect occult hypocalcemia and vitamin D deficiency as possible causes of ALTE through considering certain risk factors, namely history of exclusive breastfeeding, time of presentation in late winter or early spring, and a physical examination that reveals pigmented skin.[3]

Mahmood D. Al-Mendalawi
Department of Pediatrics
Al-Kindy College of Medicine
Baghdad University, Baghdad, Iraq

Reply from the Author

We appreciate the response by Prof. Mahmood D. Al-Mendalawi on our brief article describing acute life threatening events associated with hypocalcemia and Vitamin D deficiency in early infancy. Our cohort was assembled retrospectively from January 2007 to January 2009 and comprised 5 infants that were diagnosed with ALTE. None of the cases presented with stridor, but we did address this feature as part of the symptoms that may manifest during the serious hypocalcemic phase of nutritional rickets as reported by Ladhani et al[4] and perhaps several other authors in the literature.[5-7] It is important to note that the case discussed by Halterman et al[2] did not present as an ALTE, but was described as "being anxious and in significant respiratory distress in a sitting position." Moreover, among the described cases[5-7] only Train et al[6] describe one case of a 5-month old child who had an ALTE and following resuscitation became stridulous and was initially thought to have upper airway obstruction secondary to enlarged tonsils. Subsequently, the infant was found to be hypocalcemic and had florid rickets on x-ray examination unlike our cases in which none had skeletal or radiological deformities as a cue to the diagnosis of Vitamin D deficiency.

We agree that astute physicians in emergency departments should have a high diagnostic suspicion bias for Vitamin D deficiency in the presence of occult hypocalcemia and specific risk factors but piecing the puzzle together and arriving at a correct diagnosis may be overshadowed by the broad etiology of ALTE which sometimes prevails as the primary focus. We hope that our article serves sufficient notice that Vitamin D deficiency should be considered as one of the possible causes of ALTE in infancy.

Rafat Mosalli
Umm Al-Qura University
Department of Pediatrics

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