

## Afebrile Seizures as Initial Symptom of Hypocalcemia Secondary to Hypoparathyroidism

Sir,

I have two comments on the interesting case report by Gkampeta *et al.* on 7.5-year-old female child presented with afebrile seizures as an initial symptom of hypocalcemia secondary to hypoparathyroidism.<sup>[1]</sup>

First, the authors mentioned that the absence of cardiac defects, absence of distinct facial features, and unremarkable medical history during neonatal period helped them exclude the diagnosis of DiGeorge syndrome (DGS).<sup>[1]</sup> Nevertheless, I presume that DGS still represents an important differential diagnosis in the case in question necessitating appropriate workup. My assumption is based on the following point. It is obvious that DGS often presents with hypocalcemia during the neonatal period and it is associated with other characteristic features, including developmental delay, congenital heart anomalies, primary hypoparathyroidism, aplasia or hypoplasia of the thymus, and a dysmorphic face.<sup>[2]</sup> Truly, the clinical features of DGS are highly variable between individuals; some have subtle findings, whereas others are severely affected.<sup>[3]</sup>

Although the presentation of hypocalcemia due to DGS is mainly seen during neonatal period, presentation later in life has been reported usually precipitated by extreme stress.<sup>[4]</sup> Nearly 90% of the affected individuals have a deletion at the long arm of chromosome 22 and 80%–90% of those deletions are *de novo* mutations.<sup>[2,3]</sup> Genetic testing for that deletion is considered an important hallmark in the workup for DGS. Regrettably, it was not done in the case in question. I presume that if that genetic testing was accomplished and it revealed that chromosomal deletion, the case in question would be dually advantageous. On one hand, it could be considered a novel case report in Greece, since afebrile seizures presenting as an early manifestation of hypoparathyroidism in DGS patient has not been reported in the pediatric literature so far. On the other hand, it would support the notion that in a child with hypocalcemia, considering the hypothesis of DGS, even if there are no other manifestations, is important both for clinical diagnosis and management and for genetic counseling.<sup>[5]</sup>

Second, it would be also appropriate to seek out other causes for the studied patient, including mitochondrial disorders (several of these associated with parathyroid dysfunction) or mutations in the calcium-sensing receptor.

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There are no conflicts of interest.

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