Letter to the Editor: Neonatal Hypocalcemia in Preterm Infants

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Romagnoli *et al.* demonstrated clearly as we have published (1) that administration of 25 (OH)D did not modify the course of serum calcium during the first week of life and did not prevent the early type of hypocalcemia. We are in perfect agreement that early calcium administration either intravenously (2) or orally (3) increases calcemia and prevents the decrease of serum calcium.

25 (OH)D per se does not play any role in intestinal Ca absorption and only its metabolite 1,25 (OH_2) D has an effect on this phenomena. We have published that in premature more than 32 wk of gestation, 25 (OH)D is well hydroxylated in the kidney and the serum level of 1,25 (OH_2) D depends on the serum level of 25 (OH)D (4). We agree that a defect of vitamin D metabolism is not the primary pathogenic factor of this type of hypocalcemia.

Administration over a long period of 25 (OH)D seems to us not to be warranted because of the half-life of this compound (21–25 days) and furthermore administration of vitamin D at a sufficient dosage is safer and cheaper.

As for the late neonatal hypocalcemia, it is a different problem. Hypoparathyroidism is the primary factor (5) and administration of 1,25 (OH_2) D is the most reliable treatment. In our experience administration of 25 (OH)D or vitamin D does not prevent this disorder.

REFERENCES AND NOTES

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