

Redefining vitamin D deficiency: Reply to comments

Sir,

Thank you very much for showing keen interest in our editorial. Subclinical VDD still remains a grey area with questionable identity, with research work on this subject still in infancy. Our concern was “How much should we rely on a single value of 25OHD to diagnose VDD? As the authors have pointed out, we have not discussed 1,25-dihydroxy vitamin D (1,25-DOHD) because there is no standardization of activated hormone assay, and its value remains normal even when serum 25OHD falls to a level of 4 ng/mL.^[1] Similar concerns have also been raised about 25OHD assays.^[1] As acknowledged by the authors, we have also highlighted limitations of measurements of parathormone (PTH) in our editorial. With the availability of third generation PTH assay, the problem of interference from inactive PTH fragments has been largely resolved.^[2] However, the age-, sex-, and ethnicity-based reference range remains an area of active research. The author(s) suggest that serum 25OHD is only a screening test for VDD and is not scientific, because VDD has been defined by the serum 25OHD value. VDD can remain clinically asymptomatic; hence, the clinical pathway will not be largely helpful in deciding the presence of VDD.

Other surrogate markers have also been discussed by us including bone mineral density and bone markers, which were not very helpful in early stages of VDD.^[3] With the concern raised by the author(s), we have also acknowledged, “Till better and simple indicator of VDD is available, serum 25OHD levels should be interpreted with PTH levels, rather than in isolation.” Serum 25OHD levels can vary with genetic polymorphism of not only vitamin D receptor, but also with genetic polymorphism of various

genes involved in synthesis and metabolism of vitamin D, and carrier protein (vitamin D binding protein).^[1,4] This will further complicate the interpretation of isolated serum 25OHD levels to define VDD. Hence, we suggest that vitamin D should be interpreted with PTH and it is logical in the present scenario.

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REFERENCES

1. Romagnoli E, Pepe J, Piemonte S, Cipriani C, Minisola S. Management of endocrine disease: Value and limitations of assessing vitamin D nutritional status and advised levels of vitamin D supplementation. *Eur J Endocrinol* 2013;169:R59-69.
2. John MR, Goodman WG, Gao P, Cantor TL, Salusky IB, Jüppner H. A novel immunoradiometric assay detects full-length human PTH but not amino-terminally truncated fragments: Implications for PTH measurements in renal failure. *J Clin Endocrinol Metab* 1999;84:4287-90.
3. Garg MK, Kalra S, Mahalle N. Defining vitamin D deficiency, using surrogate markers. *Indian J Endocrinol Metab* 2013;17:784-6.
4. Garg MK, Kalra S. Rickets: Twist and turns in gordian knot. *Indian J Endocrinol Metab* 2013;17:1-4.

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Quick Response Code:	Website: www.ijem.in
	DOI: 10.4103/2230-8210.129125