

Vitamin D deficiency and youth-onset diabetes in North India

A deficiência de vitamina D no diabetes de início de juventude no Norte da Índia

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We are thankful to Kurt, O. for his keen interest in the study “*High prevalence of vitamin D deficiency among newly diagnosed youth-onset diabetes mellitus in north India*” published recently in *Arquivos Brasileiros de Endocrinologia & Metabologia* (1). It is also appreciated that the researcher has gone into the details of the study. The authors have rightly pointed out that vitamin D deficiency was more common in youth-onset diabetes (age less than 25 years) when compared with age, body mass index (BMI) and gender-matched healthy control population.

Vitamin D deficiency was also found in healthy controls, but mean vitamin D was significantly lower in people with diabetes. We found that mean vitamin D levels were significantly lower when compared with healthy controls, but we did not demonstrate the relationship between vitamin D deficiency and development of diabetes, as it was not the mandate of the study.

The author has rightly pointed out the difficulties in measuring vitamin D in blood samples. Compounds such as vitamin D, which are extremely hydrophobic, show many inherent difficulties for their measurement, also because there are two forms: viz 25(OH)D₂ and 25(OH)D₃. Currently, there are three different methods of assay, viz competitive protein binding, immunochemical and chromatographic procedures. At present, no single method seems to be the gold standard, but chromatographic methods such as gas chromatography/mass spectrometry and liquid chromatography-tandem mass spectroscopy are a possible candidates (2). A good agreement was found between high performance liquid chromatography, liquid chromatography-tandem mass spectroscopy, and radioimmunoassay. A better agreement was found especially at lower serum concentrations of vitamin D (3,4).

The author has pointed out the conditions that would affect the vitamin D concentration in blood, such as liver and kidney diseases, which were excluded both in patients and controls. Similarly, people on treatment with drugs such as rifampicin and anticonvulsants, which would alter the metabolism of vitamin D, were also excluded from the study.

We agree with the author that vitamin D deficiency has assumed pandemic proportions and diabetes is also assuming epidemic proportions, especially in developing countries. The role of vitamin D in the prevention of type 2 diabetes is not as clear as it is in type 1 diabetes. A large number of cross-sectional and longitudinal studies have reported an inverse association between vitamin D status and type 2 diabetes. Studies have also shown that replacement with vitamin D rectifies the abnormalities of impaired insulin secretion and glucose tolerance (5,6). In patients with type 2 diabetes or impaired glucose tolerance, vitamin D supplementation was reported to have inconsistent results in people with normal vitamin D status. Some, but not all studies, have reported an improvement in metabolic control in response to vitamin D supplementation (7,8).

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There have been few limited trials where the combined effect of supplementation of calcium and vitamin D was used. In one study, supplementation with 700 IU of vitamin D3 and 500 mgs of elemental calcium prevented the rise in insulin resistance and hyperglycemia in people with impaired glucose tolerance at baseline (but not in people with normal glucose tolerance at baseline) (9). Similar results have not been shown with the supplementation of a lower dose of calcium and vitamin D (10).

More prospective studies are needed to understand the importance of the prevention of type 2 diabetes with vitamin D supplementation.

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