Sir,

We read with interest the recently published article entitled, “Prevalence of Vitamin D deficiency (VDD) and associated risk factors among children residing at high altitude in Shimla district, Himachal Pradesh, India” by Kapil et al.[1] and would like to make few important comments. The authors noted a high prevalence (93%) of VDD (serum 25[OH] D level <20 ng/ml) among school children aged 6–18 years residing at high altitude in Shimla. This study contributed significantly to the limited literature on VDD in apparently healthy children in India.[2-4]

The authors stated that children in the age group of 6–11 years were not included for the assessment of socioeconomic status (SES), physical activity, sunlight exposure, and dietary pattern as these children were unable to provide valid information on these parameters. By doing so, they had missed important information from 1/3rd of children which could be simply obtained from the parents when consent was obtained. It was not clear how sample size was calculated? Whether 25% was estimated prevalence of VDD or Vitamin D sufficiency? If it was the prevalence of VDD, how it was arrived upon as various studies from India demonstrated that the prevalence of VDD is in the range of 85%–98%? Authors mentioned that VDD was more common in females, children belonging to upper SES, those having so-called symptoms due to VDD, sedentary physical activity level, sunlight exposure <150 min, and vegetarians. However, on having a look at Table 1, it can be found that the pattern was similar in Vitamin D deficient and Vitamin D insufficient/sufficient groups with no statistically significant difference except the fact that VDD was more common in females (54.3% vs. 27.3%, P < 0.001). There was no mention about how many children were receiving Vitamin D and calcium supplements. It was not mentioned how Vitamin D deficient children were treated.

We conducted a study and demonstrated that the prevalence of VDD in apparently healthy children (n = 338), 3 months-12 years, belonging to upper SES in Chandigarh was 40.24% and 8.53% of them had clinical signs of VDD.[5] On univariate analysis, VDD was associated with relatively younger age group, female sex, failure to thrive, exclusive breastfeeding, inadequate sun exposure, and no Vitamin D supplements.[6]

The prevalence of VDD among healthy children is varied in different studies. This difference may be due to different populations studied, latitude of residence, sunlight exposure, skin color, sunscreen use, weather, environmental pollution, dietary intake, Vitamin D supplementation, different methods used for measuring 25(OH) D level, and different cutoff values considered.[6] A daily intake of 400 IU/day of Vitamin D for all infants, children, and adolescents is recommended by the American Academy of Pediatrics.[7] In India, there are no such guidelines for routine Vitamin D supplementations.

Vitamin D Deficiency among Healthy Children: An Undisputed and Booming Problem
Sir,

Hypogonadotropic hypogonadism (HH) is the most frequent endocrinopathy in transfused patients with thalassemia major (TM). Hypogonadism is likely to be caused by iron deposits in the gonads, pituitary gland, or both. The treatment of pubertal disorders in thalassaemia is a complex issue due to the frequent coexistence of other factors such as severity of iron overload, chronic liver disease, insulin-dependent diabetes, and/or the identification of a hypercoagulable state. In addition, splenectomy can contribute to, and increase, the risk of thrombosis.

As the current literature is very limited regarding the potential risks of venous thromboembolism and cardiovascular in TM patients with hypogonadism, the main aim of the present retrospective study was to investigate the incidence of venous thromboembolism (deep venous thrombosis and pulmonary embolism) in three cohorts of hypogonadal men with TM treated with depot testosterone, in Muscat (Oman), Doha (Qatar), and Ferrara (Italy).

The registry database included 424 male patients followed regularly or occasionally in Muscat (96 patients), in Doha (56 patients), and in Ferrara (272 patients). In the latter group, all patients were of Italian ethnic origin. Forty-one of 96 TM patients in Muscat (42.7%), 22 of 56 TM in Doha (43%), and 95 of 272 TM patients in Ferrara (34.9%) developed a pubertal disorder: delayed puberty (1.8%), arrested puberty (1.7%), HH (91.1%), or acquired HH (5.4%).

One of the coauthors (ATS) observed the development of left atrial thrombosis in a 19-year-old adolescent male with TM and diabetes mellitus, who had been on testosterone replacement therapy (100 mg testosterone enanthate, monthly) for 1 year. His laboratory and hormonal profile is reported in Table 1.

Diabetes mellitus (blood glucose at 2 h oral glucose tolerance test = 220 mg/dl) developed 7 months after starting testosterone therapy. He was on insulin therapy with HbA1c = 8%, and he did not show any of the side effects of testosterone therapy apart from this acute incidence. The hormone replacement therapy (HRT) with testosterone was stopped. Unfortunately, no further information was available after his admission to the Cardiac Intensive Care Unit.

No cases of thrombosis were reported in our thalassaemic patients with spontaneous pubertal development.

In conclusion, male hypogonadism and its treatment is a...