Sir,

Vitamin D deficiency (VDD) is widely prevalent in Asian-Indians despite the presence of adequate sunshine.[1] Vitamin D toxicity (VDT) is being increasingly reported from our country because of overzealous correction of VDD with mega-doses of vitamin D by the general healthcare providers.[2–4] Nephrocalcinosis (NC) is a well-known but rare complication of VDT, which is usually irreversible.

We previously reported an infant with acute VDT and NC.[5] Briefly, the child presented to a local physician at the age of 8 months with symptoms of hypocalcemia and was prescribed one dose of intramuscular vitamin D containing cholecalciferol 600,000 units and oral calcium carbonate 500 mg daily. However, the parents continued the intramuscular injection for 3 consecutive weeks, and a week following the final injection, the child was brought to us with features of hypercalcemia. The child was diagnosed to have parathyroid hormone (PTH)–independent hypercalcemia due to VDT (serum total calcium 11.5 mg/dL, 25-hydroxyvitamin D >100 ng/mL, and undetectable intact PTH) and was treated with intravenous normal saline and subcutaneous calcitonin injections, with improvement in hypercalcaemic state. During the initial presentation, he was also found to have hypercalciuria (24-h urine calcium >4 mg/kg and elevated urine calcium: creatinine ratio of 0.83) and bilateral medullary NC. In this report, we present the long-term follow-up data of this child.

The child was followed up annually with serum total calcium value, urine calcium: creatinine ratio, and ultrasonography of bilateral kidneys. On serial follow-up visits, serum calcium level, urine calcium: creatinine ratio, and estimated glomerular filtration rate (eGFR) remained normal, and there was no reduction in NC. On a recent follow-up visit (14 years after the initial presentation), the child was growing normally with good scholastic performance. His total serum calcium value, urine calcium: creatinine ratio, and eGFR were normal at 9.4 mg/dL, 0.015, and 138 mL/min/1.73 m², respectively. Ultrasonography and computerized tomography of kidneys revealed persistent medullary NC with minimal reduction in size [Figure 1]. NC is defined as generalized deposition of calcium salts (calcium oxalate or phosphate) in the kidney, predominantly in the interstitium. It usually involves the renal medulla (>97% cases), and less commonly the cortex.[6] The common causes of medullary NC include primary hyperparathyroidism (PHPT), distal renal tubular acidosis (dRTA), primary hyperoxaluria, Barter’s syndrome, hereditary hypophosphatemic rickets with hypercalciuria, Dent’s disease, idiopathic hypercalciuria, medullary sponge kidney, Williams–Beuren syndrome, VDT, and treatment with active vitamin D for hereditary hypophosphatemic rickets.

In a pediatric series of 40 patients from North India with NC (median age at presentation 72 months), dRTA (50%), idiopathic hypercalciuria (7.5%), primary hyperoxaluria (7.5%), and VDT (5%) were reported as the most common causes.[7] At a median follow-up of 35 months, no patient showed resolution of NC while GFR declined significantly from 82 to 73 mL/min/1.73 m². In another study from the Netherlands, NC in preterm neonates was found to be associated with long-term adverse effects on glomerular and tubular function.[8] In a series of 41 patients from Italy (median age at presentation 15 months), renal tubulopathies (41%) and VDT (10%) were reported as the most common causes of NC.[9] The authors also reported the follow-up data (median 53 months) for 26 patients with NC. The degree of NC worsened in 16 (62%), remained stable in 8 (31%), and improved in 2 (8%) patients. The two children with improvement in NC on follow-up had VDT and unknown cause, respectively. The authors also concluded that progression of NC was not related to glomerular function, because GFR remained stable in 14 of 16 patients showing worsening of NC. Similarly, Lin et al. reported data of 16 children with NC from Taiwan [VDT (31%), dRTA (19%), and
Sir,

I read with interest, an article by Patwardhan et al. [1] published in your journal. It is commendable to note that the authors have put in effort to validate questionnaire to estimate sunlight exposure and then correlate the same with vitamin D status. But authors mention ‘Nevertheless, the association of UV exposure with serum 25(OH)D concentrations in Asian adults from tropical climates like India, where angle...

Financial support and sponsorship
Nil.

Conflicts of interest
There are no conflicts of interest.

Alpesh Goyal, Sk Hammadur Rahaman¹, Nishant Raizada³, Devasenathipathy Kandasamy¹, Ajay Prakash Mehta¹, Rajesh Khadgawat
Department of Endocrinology and Metabolism, All India Institute of Medical Sciences, New Delhi, ¹Department of Endocrinology and Metabolism, Medica Superspeciality Hospital, Kolkata, West Bengal, ²Department of Endocrinology and Metabolism, University College of Medical Sciences, ³Department of Radiodiagnosis, All India Institute of Medical Sciences, ⁴Department of Pediatrics, Max Hospital, Shalimar Bagh, New Delhi, India

References
1. Ritu G, Gupta A. Vitamin D deficiency in India: Prevalence, casualties and interventions. Nutrients. 2014;21:729-37.
2. Kaur P, Mishra SK, Mithal A. Vitamin D toxicity resulting from overzealous correction of vitamin D deficiency. Clin Endocrinol (Oxf). 2015;83:327-31.
3. Maji D. Vitamin D toxicity. Indian J Endocr Metab 2012;16:295-6.
4. Garg G, Khadgawat R, Khandelwal D, Das N. Vitamin D toxicity presenting as hypercalcemia and complete heart block: An interesting case report. Indian J Endocr Metab 2012;16:S423-5.
5. Khadgawat R, Goswami R, Gupta N, Seith A, Mehta AP. Acute vitamin D toxicity in an infant. Clin Pediatr Endocrinol 2007;16:89-93.
6. Shavit L, Jaeger P, Unwin RJ. What is nephrocalcinosis? Kidney Int 2015;88:35-43.
7. Mantan M, Bagga A, Virdi VS, Menon S, Hari P. Etiology of nephrocalcinosis in northern Indian children. Pediatr Nephrol 2007;22:829-33.
8. Kist-van Holthe JE, van Zwieten PH, Schell-Feith EA, Zonderland HM, Holscher HC, Wolterbeek R, et al. Is nephrocalcinosis in preterm neonates harmful for long-term blood pressure and renal function? Pediatrics 2007;119:468-75.
9. Ammenti A, Pelizzoni A, Cecconi M, Molinari PP, Montini G. Nephrocalcinosis in children: A retrospective multi-centre study. Acta Paediatrica 2009;98:1628-31.
10. Lin MT, Tsau YK, Tsai WY, Tsai WS, Lu FL, Hsiao PH, et al. Nephrocalcinosis in childhood. Acta Paediatr Taiwan 1999;40:27-30.

This is an open access journal, and articles are distributed under the terms of the Creative Commons Attribution-NonCommercial-ShareAlike 4.0 License, which allows others to remix, tweak, and build upon the work non-commercially, as long as appropriate credit is given and the new creations are licensed under the identical terms.

Access this article online

How to cite this article: Goyal A, Rahaman SH, Raizada N, Kandasamy D, Mehta AP, Khadgawat R. Bilateral medullary nephrocalcinosis secondary to Vitamin D toxicity: A 14-year follow-up report. Indian J Endocr Metab 2018;22:853-4.

© 2018 Indian Journal of Endocrinology and Metabolism | Published by Wolters Kluwer - Medknow