Severe vitamin D deficiency: A persistent yet preventable problem among Canadian youth

Leanne M Ward MD FRCP1, Moyez Ladhani MD FRCP2, Stanley Zlotkin MD FRCP3

1Children's Hospital of Eastern Ontario, University of Ottawa, Ottawa, Ontario; 2Hamilton Health Sciences Centre, McMaster University, Hamilton, Ontario; 3The Hospital for Sick Children, University of Toronto, Toronto, Ontario

Correspondence: Leanne M Ward, Children's Hospital of Eastern Ontario, University of Ottawa, 401 Smyth Rd, Ottawa, Ontario K1H 8L1. Telephone 613-737-2253, fax 613-738 4211, e-mail Lward@cheo.on.ca

CASE 1

A 6-month-old Inuit, formula-fed infant was taken to the emergency room by ambulance following a generalized tonic–clonic seizure. On examination, the infant was fussy and hypotonic. Biochemistry was consistent with severe vitamin D deficiency (undetectable 25-hydroxyvitamin D3 and urinary calcium to creatinine ratio, low serum ionized calcium, normal serum phosphate and elevated parathyroid hormone [PTH] and alkaline phosphatase). A hand x-ray showed no signs of rickets. Risk factors for vitamin D deficiency were determined to be severe maternal vitamin D deficiency and absence of infant postnatal vitamin D supplementation.

Given the history of a seizure, the infant was treated with intravenous calcium with oral vitamin D, 4000 IU/day and elemental calcium 50 mg/kg/day in four divided doses. Once the ionized calcium was >1.0 mmol/L (after 72 hours), the intravenous calcium was discontinued. The patient remained on vitamin D, 4000 IU/day and supplemental calcium until the biochemistry normalized (total duration of high-dose therapy was 5 months). The infant was then transitioned to vitamin D, 800 IU/day as maintenance therapy, calcium supplementation was discontinued and adequate calcium intake was encouraged through dairy products to meet the recommended intake for age (260 mg elemental calcium daily).

CASE 2

A 6-month-old dark-skinned, breastfed infant presented with lethargy and rapid breathing. On physical examination, the infant manifested growth failure, pallor, delayed developmental milestones, tachycardia, and tachypnoea with inter- and subcostal retraction. Hepatomegaly and a 2/6 systolic murmur were also present. Biochemistry was consistent with severe vitamin D deficiency (undetectable 25-hydroxyvitamin D3, low serum ionized calcium and phosphate, elevated PTH and alkaline phosphatase). A hand x-ray showed rickets and a chest x-ray was consistent with congestive heart failure. An echocardiogram confirmed a dilated cardiomyopathy. Risk factors for vitamin D deficiency were determined to be severe maternal vitamin D deficiency and lack of infant postnatal vitamin D supplementation. No causes of cardiomyopathy other than vitamin D deficiency were found. The patient was treated for 6 months with calcium and vitamin D, as for Case 1, along with cardiac and anticongestive therapy, at which time there was normalization of biochemistry and cardiac function.

LEARNING POINTS

• Vitamin D deficiency is persistent in Canada despite clear guidelines for its prevention that include maternal prenatal and infant postnatal vitamin D supplementation (1–4).
• Vitamin D deficiency has been linked to serious health outcomes, including hypocalcaemic seizures, fractures, delayed developmental milestones and dentition, cardiorespiratory failure and even death (2,5–8).
• Symptomatic vitamin D deficiency can occur not only in breastfed but also in high-risk formula-fed infants, because of severe maternal vitamin D deficiency that is incompletely rescued by the 400 IU of vitamin D per litre in standard infant formula (2,9).
• While darker-skinned infants are at greater risk for vitamin D deficiency, fair-skinned infants and infants born to fair-skinned mothers are not exempt (2,5).
• The radiographic features of all forms of rickets arise from hypophosphataemia (10). In cases where hypophosphataemia is not a feature, radiographic evidence of rickets may be absent. In such situations, the diagnosis is vitamin D deficiency without rachitic features.
• The optimal method for preventing vitamin D deficiency is to ensure maternal vitamin D adequacy through vitamin D supplementation during pregnancy (600 to 4000 IU/day) followed by infant supplementation starting shortly after birth (400 to 600 IU/day; 800 IU/day for high-risk groups such as those at Northern latitudes) (1,3,4,11).
• Vitamin D supplementation is required beyond infancy in high-risk groups, such as children living at Northern latitudes, as well as those with prolonged breastfeeding, darker skin, dietary restriction, lack of sun exposure and disorders or medications that interfere with normal vitamin D metabolism (renal, hepatic and gastrointestinal diseases, antiepileptic drugs and glucocorticoids) (2,4).
• The failure to eradicate severe vitamin D deficiency in Canada, despite evidence for serious health consequences, raises the need for an approach that places prevention in the hands of mandated public health policy. Strategies that merit consideration include intermittent vitamin D supplementation at the time of regular visits to health care providers in high-risk groups (2).

Acknowledgement

All authors declare that they have no conflict of interest to disclose.
References


