New Zealand Paediatric Surveillance Unit

VITAMIN D DEFICIENCY RICKETS

Summary

In June-2024 there have been significant changes to national NZ guidelines on Vitamin D. The current recommendations are to prescribe Vitamin D supplementation to all partially or exclusively breastfed infants until 1 year of age, irrespective of any risk factors. Additionally, pregnant people are recommended to take Vitamin D supplementation if any risk factors present (such as dark skin, high latitude, limited sun exposure).

The questions now are: How well has this new guideline been implemented and communicated? And has this led to reductions in rickets presentations particularly in the high risk <3 year age group?

Objectives

1. To ascertain the incidence of simple vitamin D deficiency rickets (also known as nutritional rickets) in NZ in children aged under 16 years diagnosed by specialist paediatricians over a two-year period
2. To obtain information of the demographics and clinical characteristics of Vitamin D deficiency rickets which will assist in the:
    a. Ongoing identification of risk factors for development of the disease in Aotearoa
    b. Evaluation of recently updated preventive strategies (ie, did cases and/or their mothers receive appropriate Vitamin D supplementation prior to diagnosis as per national guidelines, and have the recently updated national guidelines influenced the incidence and demographics)
3. To supply data that will continue to help develop public health policies to prevent vitamin D deficiency rickets among children living in Aotearoa

CASE DEFINITION AND REPORTING INSTRUCTIONS

Children up to and including 15 years of age with rickets secondary to simple Vitamin D deficiency (also known as nutritional rickets) confirmed biochemically and/or radiographically.

Inclusion criteria (Biochemical)

1. Low serum 25-hydroxyvitamin D (25OHD) (<50nmol/L)
2. Elevated serum alkaline phosphatase (ALP) (age specific)

Exclusion criteria

1. Vitamin D deficiency rickets associated with underlying disease, such as fat malabsorption, liver disease, renal insufficiency or metabolic bone disease of prematurity. Patients receiving total parenteral nutrition are also excluded.
2. Vitamin D deficiency secondary to heritable disorders of vitamin D metabolism, including:
    - 1a-hydroxylase deficiency (pseudo-vitamin D deficiency rickets)
    - Vitamin D receptor defects (hypocalcemic vitamin D resistant rickets)
3. Phosphopaenic rickets of any aetiology (where hypophosphatemia is the primary cause of the rickets, and not due to calcipenic rickets with secondary hyperparathyroidism).

Please report any new diagnosed patients, who you have seen this month, to NZPSU through our monthly reporting surveys or via email to nzpsu@otago.ac.nz.

Supplemental data ideally to be obtained prior to treatment*
(*the typical pattern of results obtained in VDDR is described in brackets*):

1. Serum calcium and albumin (normal or low), or Ionised calcium level (low)
2. Serum phosphate (normal or low)
3. Serum PTH (elevated)
4. X-ray confirmation of rickets at the distal ulnar or femoral epiphysis‡
5. Haemoglobin, MCV and serum ferritin (commonly low)

* These results are not essential for reporting.

‡In rare instances, the x-ray features of rickets may not be present at diagnosis e.g. if linear growth is arrested (and growth plate activity is blunted) or in the very early phase of the disease when x-ray changes at the growth plate are not yet visible. For this reason, x-ray confirmation of rickets is not a strict inclusion criterion but should be obtained during the initial patient evaluation

Follow-up of positive returns:
A questionnaire requesting further details will be forwarded to practitioners who report a case.

Study Background:
Vitamin D is critical for calcium homeostasis and for mineralization of the skeleton, especially during periods of growth. The most severe consequence of vitamin D deficiency during childhood is rickets (a mineralization defect at the epiphyseal growth plates). Rickets is associated with pain, fractures, skeletal deformity, growth restriction, dental defects, delayed developmental milestones and, in severe cases, hypocalcaemic seizures. Deficiency of vitamin D in children, without overt rickets, is also associated with low bone mineral density which has potential long-term implications particularly in adulthood. Of added concern, it has been recognised that Vitamin D has a major role to play in the developing and developed immune system. Low levels during foetal life, early infancy and into adulthood potentially have a major effect on the development of infection, autoimmune and cardiovascular disease, and possibly cancer.

Vitamin D is unique among vitamins as its main source is not dietary, but direct synthesis in the skin following exposure to UVB radiation from sunshine. UVB radiation exposure varies based on latitude, skin colour, sunscreen use and clothing. Changes in human lifestyle including sun avoidance practices, indoor occupations and recreation, added to New Zealand's geographical location at 35ºS to 47ºS mean that children and adults cannot depend on adequate skin exposure to sunlight for vitamin D synthesis, especially during winter months. Dietary intake is a secondary source, but for a few exceptions, without additional supplementation there is little in the foods humans normally ingest. Compounding this issue, human milk, is often not rich in vitamin D.

A prior NZPSU prospective surveillance study of NZ Vitamin D deficiency rickets (2015) found the incidence in children ≤15 years of age was 2.2 per 100 000 children. A total of 58 children were found to have VDRR, with the median age 1.4 years. For the younger children under 3 years, the incidence was increased to 10.5 per 100 000. Furthermore, the highest incidence was seen in children under 3 years living in Otago/Southland regions (highest latitudes of NZ), at 30.6 per 100 000. Other demographics associated with higher incidences of VDRR included darker skin, ethnicity (Middle Eastern, South Asian, African), exclusive breastfeeding, and season (winter/spring), consistent with known risk factors . This study also highlighted that despite national guidelines at the time recommending Vitamin D supplementation for at risk mothers and children, most cases of VDRR identified in the study had not received any prior to diagnosis.

Principal Investigators
Professor Ben Wheeler, Paediatric Endocrinologist, Department of Women's and Children's Health, Dunedin School of Medicine, University of Otago.

Associate Professor Craig Jeffries, Paediatric Endocrinologist, Liggins Institute - University of Auckland.

New Zealand Paediatric Surveillance Unit
Dunedin School of Medicine, P.O. Box 913, Dunedin
Tel: 03 556 6024
Email: nzpsu@otago.ac.nz

THANK YOU FOR YOUR HELP AND SUPPORT

THE RESULTS OF THIS SURVEILLANCE WILL BE INCLUDED IN THE ANNUAL REPORT OF THE NZPSU WHICH WILL BE AVAILABLE ON THE NZPSU WEBSITE AND CAN BE REQUESTED DIRECTLY FROM NZPSU

.