

Vitamin D Deficiency and Nutritional Rickets - Paediatric Full Clinical Guideline - Derby & Burton

Reference no.: CH CLIN G 164

1. Introduction

Vitamin D is a fat soluble vitamin which is essential for skeletal growth and bone health. Severe deficiency can result in rickets (among children) and osteomalacia (among children and adults). It is estimated that almost a fifth of the UK population are deficient in vitamin D (< 25nmol/litre).

There are two physiological forms, which have similar structures and actions. ;

- vitamin D2 (ergocalciferol) and
- vitamin D3 (cholecalciferol)

Vitamin D3 is the most abundant in humans and is produced in the skin following exposure to sunlight. 85-90% of our daily vitamin D requirement is obtained by the action of UVB sunlight on the skin (only possible during April to September in the UK). 10 -15% is obtained through diet.

2. Aim and Purpose

Management of Vitamin D deficiency

3. Definitions, Keywords

Vitamin D Status	Vitamin D Severe Deficiency	Vitamin D deficiency:	Vitamin D Insufficiency:	Vitamin D sufficiency:	Risk of Vitamin D toxicity:
Serum 25 hydroxyvitamin D level	<12.5 nmol/l	< 25 nmol/l	25-50 nmol/l	> 50nmol/l	> 250 nmol/l

Nutritional vitamin D deficiency can lead to skeletal deformities (rickets), impaired growth, muscle weakness and seizures due to hypocalcaemia.

Rickets occurs when there is defective bone mineralisation in the growth plate in children. It can be secondary to Vitamin D, calcium or phosphate deficiency.

4. Main body of Guidelines

4.0 Identification of high risk groups

Age group	Poor exposure to sunlight	Dietary	Metabolic
<5 years Children & young people with family members with proven vitamin D deficiency	Pigmented skin Occlusive garments Use of sun creams Disabled & young people who spend very little time outdoors	Vegetarian or fish-free diet Malabsorption Cholestatic liver disease Breast fed infants Children & young people with diet low in calcium or with generally poor diet	Liver disease (reduced stores) Renal disease (reduced synthesis of active vitamin D) Obese (excess storage in fat) Drugs: Rifampicin, Antiretrovirals, anticonvulsants, cholestyramine, glucocorticoids

Prevention

Lifestyle advice for Vitamin D insufficiency (and deficiency)

4.1 Safe Sun exposure: Sun exposure is the main source of vitamin D and should be exploited! However, this should be balanced with the risks of excessive exposure. Time required in the sun to make sufficient vitamin D is generally short and less than the time needed for skin to burn. This should be adjusted on an individual basis and safe practices adopted. Little and often is best.

The New England Journal of Medicine (2007) advice includes “*Exposure of arms and legs for 5 to 30 minutes (depending on time of day, season, latitude, and skin pigmentation) between the hours of 10*

a.m. and 3 p.m. twice a week is often adequate”

4.2 Diet

10- 15% is obtained through diet. The following foods are rich in Vitamin D

- 4.2.1 Oily Fish – Salmon, Mackerel, Sardines, Herring, Pilchards, Fresh Tuna etc.
- 4.2.2 Cod liver oil & other fish oils
- 4.2.3 Red meat & Egg yolk
- 4.2.4 Infant formula milk
- 4.2.5 Fortified breakfast cereals
- 4.2.6 Soya products, fortified margarines, low fat spreads

4.3 Multivitamins containing vitamin D

The Department of Health recommends daily vitamin D supplements (given as a multivitamin preparation) to the following:

1. All children between 6 months and 5 years.
2. Breast fed infants from age of one month if their mother has not taken vitamin D supplements in pregnancy or if she is known to be vitamin D deficient or insufficient

Other indications for multivitamins:

Children previously shown to be vitamin D deficient or insufficient should take a multivitamin preparation containing vitamin D. This should be continued long-term unless there is a significant lifestyle change to improve Vitamin D levels.

Multivitamins should be considered in other high risk groups (Page 1)

5.0 Indications for testing vitamin D

The main indication is symptoms. It is not recommended to

- screen the general population for deficiency.
- routinely screen high risk population.

However, clinicians should have low threshold to screen high risk population with co-morbidities likely to affect intake/absorption of Vitamin D (such as cystic fibrosis, coeliac disease and IBD). High risk populations should also be offered advice in order to avoid vitamin D deficiency.

5.1 Symptoms and signs of rickets:

- Bowing of legs (genu varum) or knock knees (genu valgum) - Note that some varus or valgus "deformity" is normal in certain age groups.
- Anterior bowing of the femur
- Painful wrist swelling (distal radius)
- Prominent costochondral joints (rachitic rosary)
- Softening of the skull with frontal bossing, and delayed fontanelle closure
- Scoliosis
- Bone pain(>3 months)
- Dental deformities (delayed dentition, enamel hypoplasia)

5.2 Other symptoms or conditions associated with Vitamin D deficiency

- Longstanding (> 3 months) unexplained bony pains
- Muscular weakness e.g. difficulty climbing stairs, waddling gait, rising from chair or delayed walking
- Infantile cardiomyopathy
- Carpopedal spasm, tetany, seizures or irritability due to hypocalcaemia

5.3 Abnormal Investigations

- Low serum calcium or phosphate , unexplained high Alkaline phosphatase
- Xrays- osteopenia, rickets, pathological fractures

6.0 Investigations:

The following investigations should be performed on children with suspected rickets.
If no suspicion of rickets testing should be limited to the ones in bold.

Blood	Urine	Xray
Calcium Phosphate Alkaline phosphatase Electrolytes Creatinine Liver Function PTH Magnesium 25 hydroxyvitamin D (save serum for possible 1,25 hydroxyvitamin D) Bicarbonate (to rule out renal tubular acidosis) Coeliac serology (consider)	Calcium Phosphate Creatinine + urinalysis for pH, protein and glucose	AP films of both wrists +/- limbs with deformity where present e.g. femur.

In nutritional rickets, biochemical findings usually include low serum calcium and phosphate levels with associated high PTH hormone concentration. Alkaline phosphatase levels are high and 25 hydroxyvitamin D levels are usually low unless there has been recent sunlight exposure.

If there is evidence of another chronic medical disorder such as liver disease or malabsorption further investigations may be needed along with referral to the relevant speciality

7.0 Management of Vitamin D insufficiency (Serum 25-hydroxyvitamin D 25-50 nmol/l)

Give advice on safe sun exposure

Advise multivitamin containing vitamin D 200-400units/day. This should be continued long-term unless there is a significant lifestyle change to improve vitamin D levels. Some children require supplementation until growth is completed

Re-testing is not normally required if the individual is asymptomatic and compliant.

8.0 Management of Vitamin D deficiency (<25nmol/l) and Nutritional Rickets

8.1 Colecalciferol (D3) and Ergocalciferol (D2)

Dose depends on age. Doses are equivalent

AGE	Colecalciferol/ Ergocalciferol (3000 IU/ml)
1-6months	1ml per day
6 months – 12 years	2ml per day
12- 18 years	3ml per day

Treatment is advised for 8-12 weeks for vitamin D deficiency and 3-6 months for Nutritional rickets provided dietary factors have improved.

Preparations for treatment:

Colecalciferol solution (3000units/ml) - **contains animal products and is not suitable for vegetarians.**

Ergocalciferol solution (3000 units/ml) – **may contain peanut oil. Unlicensed medicines**

Ergocalciferol tablets (10,000unit/ tablets)

8.2 Monitoring

Monitor calcium, phosphate and alkaline phosphatase levels after one month (**after one week if symptomatic hypocalcaemia**), then 3 monthly.

Treatment does not need to continue until limb deformity has resolved as this may take up to 2 years.

N.B. If the child's symptoms/signs have not improved despite a satisfactory vitamin D level they are unlikely to be related to vitamin D deficiency

9.0 Indications for secondary care Management

- Low calcium with or without symptoms (irritability, brisk reflexes, tetany, seizures or other neurological abnormalities)
- Children under 1 year
- Children with biochemical rickets or raised PTH
- Renal disease (CKD 4&5)
- Atypical biochemistry- including hypercalcaemia
- Failure to respond to treatment after 3 months despite good adherence
- Short stature / skeletal deformity
- Focal bone pain
- Unexplained severe deficiency
- Unexplained weight loss
- Children with repeated low serum calcium concentration with or without symptoms (irritability, brisk reflexes, tetany, seizures or other neurological abnormalities)

- symptomatic: requires immediate referral to Emergency department

- Asymptomatic: discuss treatment with paediatrician

Children with underlying complex medical disorders (e.g. liver disease, intestinal malabsorption)

5 References (including any links to NICE Guidance etc.)

1. Vitamin D deficiency in children <https://cks.nice.org.uk/vitamin-d-deficiency-in-children>
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7. British National Formulary for Children [accessed online 02/ 07/2020
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6 Documentation Controls (these go at the end of the document but before any appendices)

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7 Appendices

Appendix One - Only to be done within hospital setting

Management of Vitamin D Severe deficiency (Serum 25-hydroxyvitamin D <12.5 nmol/l) or Poor compliance

Single high dose Vitamin D therapy (STOSS therapy) In Hospital (oral treatment)

Invita D₃ (25,000 units/ml)

<6 months – 25,000 units Once a Week for 7 weeks

6 months – 12 years – 50,000 units once a week for 7 weeks

>12 years – 75,000units once a week for 7 weeks

Secondary care STOSS treatment – IM doses

1 month upto 6 months – Ergocalciferol 150,000 units as a single dose
6 months – 12 years – Ergocalfierol 300,000 units as a single dose

>12 years – Ergocaliferol 500,000 units as a single dose

Maintenance STOSS

Two IM doses of 100,000 units (1st dose at the beginning of Oct and second dose 3 months later (Jan))