NEONATAL MANAGEMENT FOR EXISTING MATERNAL CONDITIONS

5. MATERNAL VITAMIN D DEFICIENCY

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5 Maternal vitamin D deficiency
Neonatal Postnatal
Clinical Guidelines
King Edward Memorial Hospital
Perth Western Australia

BACKGROUND INFORMATION

See Clinical Guideline Section B 1.1.9 Screening and treatment of vitamin D deficiency in pregnancy for detailed information about vitamin D deficiency:

- risk factors
- screening tests
- treatment in pregnancy
- follow-up in pregnancy
- education

KEY POINTS

1. Breastfeeding neonates should be supplemented with 400 IU of vitamin D daily until 12 months of age if:
    - The mother is dark skinned, veiled, or high risk for vitamin D deficiency
    - The mother has been treated for vitamin D deficiency in pregnancy
2. If a breastfeeding mother is at 'high risk' for Vitamin D deficiency and has not been screened in pregnancy she should be advised to arrange screening for herself and the neonate at the 6 week postnatal check. The GP should be advised of this recommendation in the discharge information.
3. Neonates of vitamin D deficient mothers who are formula feeding will probably not require supplementation as most formulas in Australia contain 400 IU of vitamin D3 per litre. Mothers must be advised to ensure the brand contains the recommended supplementation.[1]
4. If a mother has vitamin D deficiency she should be advised to arrange screening for other family members (particularly children).
5. Vitamin D deficient women once treated, and vitamin D levels are within a normal range, should continue maintenance supplementation until cessation of lactation.
6. Vitamin D stores in the neonate from the vitamin D-replete mother last for at least 8 weeks.
7. The American Academy of Paediatrics recommends infants have a minimum intake of 200 IU of Vitamin D a day. The vitamin D content obtained by average consumption when breastfeeding (750mL/) will only provide up to 38 IU/day.[2] The neonate is at high risk of rickets with maternal vitamin D deficiency.

BACKGROUND

Vitamin D has long been recognised as a major factor responsible for skeletal mineralisation through its role in maintaining calcium homeostasis under the influence of parathyroid hormone (PTH). An ever increasing range of physiological and metabolic functions for vitamin D and its metabolites have been identified, with diverse roles in immune-regulation, cardiovascular function and neurodevelopment (Marshall). Deficiency of this important hormone has been linked with increased risk of Type 1 Diabetes, respiratory infections, malignancy and cardiovascular disease, in addition to the its traditionally recognised role in the development of rickets (Perrine)

The term born infant skeleton contains approximately 30 gm of calcium, the large majority of which is accreted during the third trimester (Kovacs). A significant proportion of this calcium is obtained from

the maternal diet, as intestinal absorption is increased up to two-fold from early in pregnancy. The impact of both maternal deficiency of Vitamin D during pregnancy, and its correction by oral supplementation on both the fetus and the lactating infant remains equivocal (Kovacs). Several studies have failed to identify a significant relationship between measures of maternal 25(OH) Vit D and neonatal indices such as birthweight, head circumference, length or cord calcium levels. Anthropometric parameters from infants aged 9 months also showed no relationship with maternal levels, whilst children aged 9 years demonstrated a lower bone mineral content in cases of maternal 25(OH) Vit D <27.5 nmol/L during pregnancy. The authors postulated early programming of childhood bone mass during *in utero* life as a possible mechanism (33 in Kovacs). A study recently published from researchers in Perth, using Raine Study data collected during 1989-1991 suggested that infants of mothers with vitamin D deficiency during pregnancy have a two-fold increase risk in delayed language development. It is worth bearing in mind that many factors likely to alter the prevalence of Vitamin D deficiency in women at KEMH have occurred since that time, including altered sun exposure and skin protection behaviours, as well as a population demographic with dark-skinned and veiled women recognised as being at higher risk for Vitamin D deficiency.

MANAGEMENT

MILD MATERNAL VITAMIN D DEFICIENCY (25-50 NMOL/L)

Women diagnosed with mild Vitamin D deficiency will hopefully have been identified either prior to pregnancy or at the time of having 'booking bloods' obtained. In most cases, supplementation will have been commenced and Vitamin D levels normalised on follow-up screening. Although there is the potential for the fetus to experience compromised transport of 25(OH) Vit D3 in such cases, most fetal demands are met via maternal stores and are unlikely to be compromised unless the maternal level of deficiency is significant. Most skeletal mineralisation with calcium in the fetus occurs in the third trimester, at which point it is hoped that most cases of deficiency will have been identified through screening blood tests and supplementation commenced. There is little available data to indicate that treatment of infants of mildly Vitamin D deficient mothers with supplementation has a significant impact on medium or long-term outcomes such as neuro-cognitive development or Rickets.

Until further evidence is available, mildly deficient mothers should be recommended to commence Vitamin D supplementation and their infants monitored for appropriate growth and development. This is particularly important for those of high risk mothers (e.g. dark-skinned or veiled women) whose exposure to natural sunlight may be limited. Other family members, including other children, should also be monitored by the GP for clinical evidence of Vitamin D deficiency.

MODERATE (<25) – SEVERE (<12.5) MATERNAL VITAMIN D DEFICIENCY

Infants of mothers with moderate to severe Vitamin D deficiency (<25 nmol/L) should be treated with cholecalciferol. KEMH uses Bio-Logical ® solution, 1000 I.U. / 0.2 mL with a dose of 0.2 mL given orally daily, for 3 months. In severe cases, particularly in high risk families or in cases of persistent severe Vitamin D deficiency in the breast-feeding mother, consideration may be given to assessing the infant's metabolic status after three months with serum Ca, PO4, ALP and Vit D levels. Ongoing requirement for Vitamin D supplementation may be considered until establishment of solids and should be discussed with a Paediatric Endocrinologist.

Infants born at KEMH, to mothers who are or have been moderate to severely deficient in Vitamin D during pregnancy, should be prescribed Cholecalciferol supplementation as above. Biochemical testing prior to commencement is not necessary. This is particularly important in infants who are breast, rather than formula fed, as formula contains significantly higher Vitamin D levels than breast milk. The mother and infant should be followed up by their GP. A referral letter is provided under **Forms** in the Post Natal Ward Guideline.

MOTHER IS HIGH RISK FOR DEFICIENCY BUT HAS NOT BEEN TESTED.

Infants of veiled or dark-skinned women are at risk of Vitamin D deficiency, as are other family members (e.g. siblings). Women at risk should be counselled to visit their GP for testing of serum levels if this has not already been performed, and should be commenced on oral Vitamin D supplementation if considered appropriate. Infants of breast-fed mothers are particularly at risk and should be monitored closely for evidence of deficiency during the first year of life. Supplementation of Vitamin D intake in the infant is limited to either Pentavite (0.45 mL daily contains 400 IU) or cholecalciferol solution (e.g. Bio-Logical solution, 1000 IU / 0.2 mL daily). Formula fed infants are unlikely to require supplementation, but should be monitored for clinical symptoms of deficiency during the first year also.

See Clinical Guideline Section B 1.1.9 Screen for and treatment of vitamin D deficiency in pregnancy for other women who are at risk for deficiency. The neonate may need a supplement depending on maternal history.

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