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Hiding from the sun

Vitamin D deficiency in refugees

BACKGROUND

It has been well established that women who wear a veil for cultural reasons and dark skinned migrants from Africa have an increased prevalence of vitamin D deficiency. Many refugee patients also come from countries where their skin is covered or they are indoors for most of the day.

OBJECTIVE

This article explores the risk, diagnosis and management of vitamin D deficiency in the Australian refugee population.

DISCUSSION

In 2004–2005, 75% of the 7000 refugees settling in Australia were from African countries and 20% were from the Middle East. Refugees may be exposed to less sunlight in Australia than in their country of origin because of an indoor lifestyle or an increased latitude. Refugee health centres confirm that vitamin D deficiency is present in 40–80% of refugee patients. Importantly, this is often asymptomatic. General practitioners are encouraged to test for vitamin D deficiency in refugees, especially as part of the initial health assessment.

No one would contest that Australia is a country that has plenty of sun. We pay for this with the highest prevalence of skin cancer in the world with two out of three Australians being treated for skin cancer in their lifetime.¹ However, public health messages to reduce sun exposure must be balanced with maintaining adequate vitamin D levels,² especially given Australia's multicultural and aging population. People who come to Australia as refugees are particularly at risk, with approximately 40–80% prevalence of vitamin D deficiency in refugee health centres across Australia.³

There is a growing body of Australian research on asymptomatic vitamin D deficiency in the elderly, women who wear a veil for cultural reasons, and migrants from Africa as well as in healthy children who spend more time indoors.^{4,5} One study of veiled and dark skinned pregnant women in an antenatal clinic in Melbourne, Victoria, found that 80% were deficient in vitamin D.⁶ For reasons including fear of physical harm, many refugees have not been outdoors for a long time and so have had very little sun exposure. This seems to be particularly common in refugees from East Africa who have spent time in Egypt where the prevalence of vitamin D deficiency has been found to be 100%.³

Vitamin D is not really a vitamin but a neurohormone that is important for calcium metabolism, bone growth, immune function, gene stability, muscle function and brain development. It is derived predominantly through the action of ultraviolet (UV) light on precursors in the skin but may

also be absorbed through the gut from oily fish and eggs. It is then further metabolised in the liver and kidney. The two forms of vitamin D are cholecalciferol (vitamin D3) and ergocalciferol (vitamin D2). Levels of serum 25-OH vitamin D3 are used as the indicator of vitamin D status.⁷ Lips classifies a level of 25-OH vitamin D above 50 nmol/L (20 ng/mL) as normal, 26–50 nmol/L (10–20 ng/mL) as a mild deficiency, 12.5–25 nmol/L (5–10 ng/mL) as moderate and <12.5 nmol/L (<5 ng/mL) as a severe deficiency.⁸

People with darker pigmentation or keratinisation causing darker coloured skin have decreased sunlight penetrating to the deeper layer of the skin where vitamin D is produced.^{9–13} Other populations at risk of deficiency include: those with decreased sun exposure because of lack of time spent outdoors (an increasingly common situation in developed settings), those covered extensively by clothing, as seen most profoundly in women observing purdah, and those with gut absorption problems or who have a diet deficient in vitamin D.^{6,7,14–17} Up to 2.5 hours of sun exposure per day may be needed to supply enough vitamin D for people with darker skin.¹⁷ Most commentators also agree that the current western diet is inadequate with respect to vitamin D intake, especially in countries such as Australia where most food is no longer fortified with vitamin D.^{2,7,16–19}

Presentation and diagnosis

Vitamin D deficiency most commonly presents asymptotically. However, it may also present as osteoporosis or its complications, particularly in

postmenopausal women and the elderly, and as rickets, delayed walking, leg bowing, seizures or failure to thrive in children.^{2,16,17,20} Infants of women who are vitamin D deficient during pregnancy will usually also be vitamin D deficient and hence at an increased risk of both short and long term sequelae.^{2,6,21} In addition to the well established dysfunction of bone and muscle metabolism, vitamin D has been linked with a wide range of other illnesses. These include diabetes, schizophrenia, prostate cancer, multiple sclerosis and autoimmune disorders.^{15,22–25}

Less well known, but well established in the literature, is the prevalence of muscle pain and weakness as a result of vitamin D deficiency.^{14,16,17,26,27} In Australia, muscle pain in particular appears to be a problem in refugee and immigrant communities, especially those from Africa and the Middle East.^{7,14,16,17,19,21} The diagnosis of vitamin D deficiency associated with muscle pain, however, is often missed. One study reported an average duration of 59 months before the correct diagnosis of vitamin D deficiency was made in a group of patients with musculoskeletal symptoms, with a range of ‘serious therapeutic consequences’ for some.²⁸

Treatment

Up to 500 000 IU of vitamin D may be needed to replenish stores in those who are deficient.^{2,29} Recent Australian studies on the efficacy and safety of ‘stoss therapy’ – which involves megadoses of cholecalciferol given intramuscularly or orally according to a standard protocol – to treat vitamin D deficiency concluded that this is safe, effective and cost beneficial compared to the oral ergocalciferol currently available.^{2,29,30} Treatment protocols are also available for infants and pregnant women who are vitamin D deficient using higher doses of vitamin D.^{2,21} Other studies have shown that complete resolution of symptoms, including muscle pain and weakness, can be rapid when adequate doses are given to those with moderate to severe deficiency.²⁶ Both in this author’s clinical experience and in case reports from the literature, patients have ‘left their wheelchair’ after only 3–6 weeks of treatment of their muscular weakness with high dose cholecalciferol (see *Case study*). However, in Australia, vitamin D is currently only available over-the-counter as up to 1000 IU of ergocalciferol or

cholecalciferol and is not available at a subsidised rate through the Pharmaceutical Benefits Scheme. Some hospitals and compounding pharmacies are able to supply higher dose preparations of vitamin D.

Conclusion

Some refugees are at substantially increased risk of developing vitamin D deficiency and its short and long term sequelae. Often the deficiency is asymptomatic or presents with symptoms

such as muscle pain, therefore it is important that general practitioners consider the diagnosis based on risk factors and maintain a high index of suspicion. In particular, serum vitamin D levels should be part of the initial health assessment of all refugees and health providers should be prepared to provide adequate long term treatment and follow up in patients with vitamin D deficiency (*Table 1*).

Conflict of interest: none declared.

Case study

A 38 year old Middle Eastern refugee with severe postnatal depression spent 12 months in and out of psychiatric hospitals and immigration detention centres. She had been immobile in bed or in a wheelchair for 8 months with severe pain, weakness and allodynia (extreme sensitivity to touch) such that it was difficult to distinguish if her condition was physical or psychological. Her 25-OH vitamin D was 15 nmol/L (60–160), alkaline phosphatase 342 U/L (30–110), and parathyroid hormone 34.3 pmol/L (0.8–5.5). She was treated with Ostelin 1000 (ergocalciferol) twice per day, and because she had pseudofractures, she was also given calcium carbonate 1500 mg (Caltrate, equivalent to 600 mg calcium) twice per day and alendronate 70 mg (Fosamax) weekly.

Six months after beginning this regimen she was still wheelchair bound and was taking morphine in the form of MS Contin 30 mg twice per day for her severe leg pain. She had not left the house and was being visited at home by her psychiatrist and physiotherapist. She was given cholecalciferol powder 50 000 IU from the hospital for three doses at two weekly intervals for her inadequately treated vitamin D deficiency. Her improvement was dramatic and 6 weeks later she had stopped her MS Contin and was walking unaided.

Twelve months later, she has had another child and has remained well, with a normal vitamin D level of 59 nmol/L, ALP 188 U/L, and PTH 6.1 pmol/L.

Table 1. Vitamin D deficiency screening and follow up

Measure serum 25-OH vitamin D/ALP/Ca/phosphate

25-OH vitamin D >50 nmol/L

No treatment necessary

25-OH vitamin D 25–50 nmol/L

100 000 IU–150 000 IU stat vitamin D orally or IMI if available^{2,29,30}

OR

500–1000 IU/day oral cholecalciferol or ergocalciferol (this dose is also safe for newborns and pregnant women)^{2,21}

Repeat vitamin D level/ALP/PTH after 2–3 months and repeat dose as necessary until blood tests are normal

25-OH vitamin D <25 nmol/L

Treatment and follow up as above, plus:

- If over 40 years of age or any suspicion of pathological fractures do bone density studies
- If less than 15 years of age or any evidence of rickets or abnormal bone growth do hand/wrist/knee X-rays and refer to a children’s hospital

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