Muscle pain as an indicator of vitamin D deficiency in an urban Australian Aboriginal population

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The prevalence of vitamin D deficiency among Aboriginal people in Australia is unknown. One of the possible sequelae of vitamin D deficiency, muscle pain, appears to have a higher prevalence in Aboriginal people.

A deficiency of vitamin D can cause osteoporosis, rickets in children, muscle pain and weakness. It is one of the main causes of undiagnosed muscle pain in adults. Such pain resolves rapidly with adequate doses of vitamin D.

Risk factors for vitamin D deficiency include darker skin pigmentation, urban lifestyle, veiling of women for cultural reasons, and intestinal malabsorption or a diet deficient in vitamin D.

Refugees from Africa and the Middle East are known to have a high risk of rickets and muscle pain caused by vitamin D deficiency. There is also research showing a deficiency in asymptomatic patients, both those at high risk as well as those with no obvious risk factors. This is important for the infants of women who are deficient in vitamin D, and hence at increased risk of both short- and long-term sequelae.

A study of a rural Aboriginal community found that 95% of the population had chronic non-specific musculoskeletal pain, compared with 30% in the general population. Traditional Aboriginal people spend much of their day outdoors, but most now have an urban lifestyle. They are less likely to spend enough hours in the sun or have a diet rich in vitamin D.

After noting a high prevalence of muscular pain among patients at our health service (Nunkuwarrin Yunti, an Aboriginal Community Controlled Health Service, at Elizabeth Downs in the northern suburbs of Adelaide), we conducted a case-control study to determine if muscle pain was associated with low vitamin D levels.

METHODS

Discussions about the study’s relevance with Elder Aboriginal Women in the community, and with the staff and Chief Executive Officer of Nunkuwarrin Yunti acknowledged its importance to individuals, families and the community. Ethics approval was obtained from the University of Adelaide and the Aboriginal Health Research Ethics Committee.

Results from Indigenous patients seen in our clinical practice before the study showed a serum vitamin D (25-hydroxyvitamin D) range of 35–55 nmol/L (standard deviation, 5 nmol/L). Anticipating a difference between cases (with muscle pain) and controls (without muscle pain) of 10 nmol/L, we calculated that a sample of six cases and six controls would be required, assuming a power of 0.8 and a significance level of 0.05.

Data were collected from eight patients in each group in October and November 2005, at the end of the Australian winter. Blood samples were collected from patients aged 18 years and older with muscle pain and from a sex- and age-matched control group without muscle pain. People with renal failure or who had recently taken vitamin D supplements were excluded. All patients had what would be classed as medium skin pigmentation.

The blood samples were sent to the local pathology service, where 25-hydroxyvitamin D was measured. Serum levels were tabulated and analysed using SPSS version 13.0 (SPSS Inc, Chicago, Ill, USA).

RESULTS

Our results are summarised in the Box. All patients with muscle pain had a vitamin D level below the normal value of 50 nmol/L. The mean vitamin D level was 40.88 nmol/L (SD, 3.52 nmol/L) for patients with muscle pain, and 58.25 nmol/L (SD, 15.90 nmol/L) for controls.

Data were normally distributed and equal variances could not be assumed. A t test showed a mean difference between cases and controls of −17.38 nmol/L (P = 0.017).

DISCUSSION

The eight Aboriginal patients with muscle pain had lower vitamin D levels than those without muscle pain. Vitamin D deficiency was not observed in asymptomatic patients except for one with mild deficiency. Despite being at a lower risk of osteoporosis, Aboriginal people may have an increased risk of muscular symptoms of vitamin D deficiency. We did not assess intercurrent illness, severity of symptoms, skin pigmentation, diet, time spent outdoors and success of treatment, and this limitation may affect the generalisability of our findings.

We found that muscle pain is an indicator of vitamin D deficiency in urban Aboriginal patients. General practitioners are well placed to screen those at high risk and may be able to improve the lifestyle and level of function of many previously undiagnosed patients with chronic muscle pain by having a high index of suspicion for vitamin D deficiency.

As more research reveals the sequelae of vitamin D deficiency, its importance to general health is likely to increase. A larger
study looking at the prevalence of muscle pain in the urban Aboriginal population, its effect on lifestyle, how that pain relates to vitamin D deficiency, and whether pain is reduced with treatment would clarify some of the issues. The potential for better quality of life resulting from successful treatment of muscle symptoms caused by vitamin D deficiency makes the clarification of this association a priority for Aboriginal health.

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