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Vitamin D Deficiency and Chronic Low Back Pain in Saudi Arabia

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Study Design. Initial assessment involved 360 patients (90% women and 10% men) attending spinal and internal medicine clinics over a 6-year period who had experienced low back pain that had no obvious cause for more than 6 months. The patients ranged in age from 15 to 52 years.

Objectives. To investigate the contribution of vitamin D deficiency as a cause for idiopathic chronic low back pain, to find a simple and sensitive test for screening patients with low back pain for vitamin D deficiency, and to determine the correlation between the vitamin deficiency and pain.

Methods. A biochemical assay of serum calcium, phosphate, alkaline phosphatase, and 25-hydroxy vitamin D level was performed before and after treatment with vitamin D supplements.

Results. Findings showed that 83% of the study patients (n = 299) had an abnormally low level of vitamin D before treatment with vitamin D supplements. After treatment, clinical improvement in symptoms was seen in all the groups that had a low level of vitamin D, and in 95% of all the patients (n = 341).

Conclusions. Vitamin D deficiency is a major contributor to chronic low back pain in areas where vitamin D deficiency is endemic. Screening for vitamin D deficiency and treatment with supplements should be mandatory in this setting. Measurement of serum 25-OH cholecalciferol is sensitive and specific for detection of vitamin D deficiency, and hence for presumed osteomalacia in patients with chronic low back pain. [Key words: chronic low back pain, 25-OH vitamin D level, oral vitamin D therapy, serum] **Spine 2003;28:177–179**

Although Saudi Arabia enjoys a sunny climate throughout the year, direct exposure to sunlight by the local population is hardly practical. This is partly because of the excessive heat, which makes sunbathing a risk for heat disorders, and partly attributable to cultural reasons whereby Muslim communities, especially the women, tend to avoid body exposure. This adds an extra burden to the calcium balance for the female population, together with that of pregnancy and lactation, throughout their reproductive life. Accordingly, vitamin D deficiency is expected to be a major problem in such a community, especially for women during their years of reproduction.[3,5,10]

The clinical manifestation of vitamin D deficiency is protean, and lower back pain alone is a well-recognized presentation of the disease.[3–5] This study was conducted to assess the contribution of vitamin D deficiency to lower back pain, a major symptom in the presentation of patients.

Methods

Patients seen initially were assessed clinically for pain and the presence or absence of neurologic manifestation using plain radiograph of the lumbosacral spine, complete blood count, and ESR. Patients who had a mechanical cause for the back pain were excluded from the study. Both CT scanning and MRI of lower spine were performed to exclude disc prolapse, spinal stenosis, and degenerative disease of the spine. These patients were labeled as having idiopathic low back pain after the aforementioned assessment. Patients with clinical features suggestive of neurologic involvement were excluded.

Patients with renal impairment or chronic liver disease also were excluded from the study. Venous sampling was done for serum calcium, phosphate, alkaline phosphatase, and 25-hydroxy vitamin D3. The latter was measured by radioimmunoassay (RIA) using kits supplied by Dia Sorin (Minnesota, USA). The normal range accepted in our laboratory was 22.5 to 93.8 nmol/L. Oral therapy with 25-OH cholecalciferol using a dosage of 5000 to 10,000 U/day was commenced for all patients after venous blood sampling. Patients whose weight was less than 50 kg were given 5000 U/day, and those with weight exceeding 50 kg were given 10,000 U/day. Three months after vitamin D therapy had begun, measurements of all the aforementioned parameters were repeated, with clinical assessment of pain.

Results

The results of this study are presented in Table 11 and Figure 1. From a total of 360 patients, 299 (83%) (24 men and 275 women)(83%) were found to have a low serum level of 25-OH vitamin D3. These patients were further subdivided into groups with mild (15–22.4 nmol/L), moderate (10–14.9 nmol/L), and severe deficiency (<10 nmol/L). Serum calcium phosphate and alkaline phosphatase both were at normal levels in all 360 patients. There was no measurement of 24-hour urinary excretion because of technical difficulties in clarifying instructions to patients. The measurements of serum calcium, phosphate, alkaline phosphatase, and 25-OH vitamin D3 all were repeated after 3 months of therapy with 25-OH cholecalciferol 5000–10000 U/day.

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