Vitamin D and Muscle Weakness

JK Mokta, Kiran Mokta, Asha Ranjan, A Muruganathan

Professor, Asst. Professor, Senior Resident, Dept. of Medicine, IGMC, Shimla, Himachal Pradesh; Chairman, Shristi AG Hospital, Tirupur, Tamil Nadu

Sir,

Although ubiquitous, vitamin-D deficiency is still unrecognized and under-diagnosed. Proximal muscle weakness is a common symptom of vitamin-D deficiency and occurs before biochemical signs of its deficiency appears. Therefore, vitamin-D deficiency should be kept in the differential diagnosis for the muscle weakness, as it is reversible and easily treated with vitamin-D supplementation.

A 53 years old woman and a 46 years old male presented in our out-door clinic in May and July 2014 because of difficulty in getting up from squatting position. About 15 month before presentation, the family members noticed the difficulty in getting up from the sitting position, which was progressive. For last 6 months, they had to use hands and or needed help of the others to get up. They denied generalized aches, other symptom or any drug intake. On physical examination, muscle bulk and muscle tone were normal, muscle power was 3/5 in the flexor and extensor of the hip and they were unable to get up from squatting position unaided.

Their complete hemogram, fasting plasma glucose, liver function tests, renal function tests included electrolytes, vitamin B12, thyroid function tests, rheumatoid factor, anti-nuclear anti-body, muscle creatinine phosphokinase (CPK) and CXR were normal. Corrected serum calcium was 7.8 mg/dl and 8.4 mg/dl respectively with normal alkaline phosphatase levels. Their serum 25-hydroxyvitamin-D levels were extremely low (4.5 ng/dl and 5.8 ng/dl). Keeping in view their risk factors: dark skin color, wearing whole body clothes, low sun exposure and poor dietary intake, a diagnosis of proximal muscle weakness due to severe vitamin-D deficiency was made. Treatment with oral cholecalciferol 60,000 IU weekly for eight weeks followed by 60,000 IU monthly was initiated. They were able to stand up unaided now and their serum 25-hydroxyvitamin-D levels were 33 ng/dl and 31.5 ng/dl at 3 month follow-up.

About 30% of patients with hypovitaminosis-D presents with proximal muscle weakness and is often unrecognized because patients don’t complain of muscle weakness until they are unable to rise from sitting position. A serum 25-hydroxyvitamin-D <20 ng/dl causes increased body sway and level <10 ng/dl leads to difficulty in rising from sitting position and inability to ascend stairs. The powerful type II muscle fibers that are essential for muscle strength are atrophied in vitamin-D deficiency. Skeletal muscle contains vitamin-D receptors that modulate various transcription factors in muscle cells, mediating muscle cell proliferation and differentiation into mature type II muscle fibers. Only in minority of patients with vitamin-D related muscle weakness has raised CPK and muscle biopsy shows non-specific muscle fiber atrophy and no sign of inflammation. Ultimate evidence of the diagnosis rests on the response to therapy. Muscle strength strikingly improves when 25-hydroxyvitamin-D level increases from 4 ng/dl to 16ng/dl and continues to improve as the level increases to more than 40 ng/dl as seen in our patients.

We conclude that in patients with proximal muscle weakness and finding of typical biochemical alterations in high risk individuals should limit exhaustive and costly neuromuscular work up. In such patient, a therapeutic trial of vitamin-D is warranted.

References