



Vitamin D deficiency and chronic autoimmune disease

Case—A 79-year-old Fijian woman, who had lived most of her life in Tokoroa (central North Island), was diagnosed with mixed connective tissue disease (MCTD) at the age of 39 years. Antinuclear antibody titre=2560 units; RNP extractible nuclear antigen=41 units ($n < 20$); ds DNA antibodies=2.1 IU/ml ($n: 0-4$). The patient had also been diagnosed with type 2 diabetes at 37 years, eventually receiving insulin.

Both disorders (MCTD and type 2 diabetes) were treated concurrently, and prednisolone 15 mg/day was required to suppress MCTD, thereby exacerbating her diabetic state. She developed retinal, renal, and ischaemic heart complications (requiring a pacemaker). Complications of MCTD included pulmonary fibrosis, pleurisy, pericarditis, and pulmonary artery hypertension. Osteopenia was noted radiologically, and considered to be steroid-related. There were no radiographic signs of osteomalacia.

Vitamin D deficiency was noted, with 25 hydroxy vitamin D ≤ 12.5 nmol/L ($n > 50$); parathyroid hormone (PTH)=65 pmol/L ($n: 1.2-6.2$); calcium=2.16 mmol/L ($n: 2.15-2.57$); corrected calcium=2.36 mmol/L; ionised calcium=0.99 mmol/L ($n: 1.13-1.32$). Creatinine=0.11 mmol/L.

After 5 weeks' treatment with calcium 1.25 g/day and high-dose vitamin D₂, serum 25 hydroxy vitamin D rose to 82.5 nmol/L, and PTH fell to 5.2 pmol/L. Ionised calcium became normal.

In a group of 45 chronic rheumatoid patients treated with slow-reacting agents by Doube in Waikato, 16 were found to be vitamin D deficient. Serum 25 hydroxy vitamin D (mean 23.75 nmol/L) was observed in the latter sub-group. No cause for vitamin D deficiency was found. Enquiries about sunshine exposure were not made. It was considered that vitamin D deficiency probably did not relate to the autoimmune disease process, or therapy.¹

The present patient had MCTD for 40 years and had been active socially. She had tended to remain home latterly, as she was poorly mobile. She was brown-skinned, habitually covered her body and arms with clothing, and wore a scarf on her head. She kept out of the sun. Her diet was poor. She had noticed that her limb muscles were wasting, and rising from a chair was difficult. Her weight was 64 kg and body mass index 22 kg/m².

Discussion—Inadequate sunlight exposure, poor skin response to sunlight, low vitamin D content in diet, decreased absorption or decreased hepatic 25-hydroxylation of vitamin D, or decreased action or increased clearance of renal 1,25-dihydroxy vitamin D may compromise vitamin D status in longstanding illness. Correction of vitamin D deficiency was achieved quickly in this patient, thus indicating swift absorption of vitamin D₂, with normal hepatic and renal hydroxylation.

Many chronic ailments, such as tuberculosis, rheumatoid disease, and hypertension, have been associated with vitamin D deficiency. There was no evidence in this case of chronic MCTD and diabetes of metabolic derangement of vitamin D status, and the

clinical explanation was low sunshine exposure. Low body weight, reduced mobility, and lack of sunlight exposure are particular risk factors for severe vitamin D deficiency in Auckland.²

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References:

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