Reversible Parkinsonism Due to Vitamin D Toxicity

Sir,
A 72-year-old retired man presented to the medical emergency with a history of difficulty in walking, tremors of hands and forgetfulness for the past year. There was associated nausea, loss of appetite, and significant loss of weight in the past 6 months. No history of fever or other constitutional symptoms was present. Family members denied any episodes of seizures or recent trauma. There was no history of falls, visual complaints, and hallucinations in the past. The patient was a known case of hypertension for the past 5 years and was on tablet amlodipine 10 mg once daily. His family members revealed that he was in the habit of taking over the counter vitamins and health supplements from the pharmacy 2 to 3 times a week.

On examination, the patient was conscious but not oriented to time place or person. Mild dehydration was present. Glasgow coma scale was E 4 V 3 M 4. Pulse rate was 86 beats per minute, regular, normal, and character. Blood pressure was 136/82 mm of Hg in the upper limb in supine position.

There was no pallor, icterus, cyanosis, clubbing, lymphadenopathy, or edema. Higher mental functions could not be tested. Resting pin rolling tremor was present. There was hypertonia in all four limbs with cogwheel rigidity. He was moving all four limbs equally. All deep tendon reflexes were exaggerated. Plantars were flexor. The clinical impression was of a parkinsonian disorder, likely primary Parkinson’s disease, given the age at initial presentation and the representative neurological findings. The altered mentation was, however, unexplained. Routine investigations revealed a corrected serum calcium of 13.2, a serum phosphorus level of 6.1 other electrolytes were within normal range. Serum parathyroid hormone (PTH) levels were done and were significantly low for age at 4.16 ng/ml. There was no derangement in renal function. Serum Vitamin D3 levels were studied and found to be raised at 142.7 ng/ml. Urinary calcium was found to be significantly raised at 386 mg per 24 h. The possibility of hyperparathyroidism was ruled out as serum PTH was suppressed. On carefully revising the history, family members revealed he had been taking Vitamin D3 sachets 60,000 iu/week for the past 4 years as over the counter vitamin supplements. A diagnosis of Vitamin D toxicity was made. The patient was hydrated adequately and started on loop diuretics. The patient showed clinical improvement over the next week with near complete resolution of symptoms. At discharge, the corrected calcium level was found to be 9, with an ionic component of 5.8. On 6 months follow-up, the patient did not demonstrate any relapse of similar symptoms, his relatives having been apprised of the need to restrict unwarranted nutritional supplementation.

Parkinson’s disease has been repeatedly linked in existing literature Vitamin D insufficiency.[1-3] Dietary Vitamin D, in addition to endogenous Vitamin D have been found to be inversely associated with the prevalence of Parkinson’s disease.[4] Vitamin D has been found to be a potential modulator of neurodegenerative disorders due to the widespread presence of Vitamin D receptors in the human brain along with 1-alpha-hydroxylase, the enzyme responsible for activation of Vitamin D.[5]

There have been reported cases of hypercalcemia secondary to hyperparathyroidism causing parkinsonian symptoms. Our case was a rare event of hypervitaminosis D-induced parkinsonism that may mimic a state similar to hyperparathyroidism. Vitamin D supplementation leading to parkinsonian features in not widely reported in literature.

A meta-analysis to find out the role of Vitamin D and Parkinson’s disease concluded that supplementation of the same may have a role in the prevention of Parkinson’s disease.[6] However, one must always in life watch for a streak of zealotry in our endeavors, even regarding vitamin supplements supported by evidence.

The over the counter sale of vitamin supplements bears potential risks of such instances of abuse that may lead to serious medical conditions.[6] In a country like India, where even prescription medicine is routinely dispensed at abandon on no recommendation from the medical community, the regulation of over the counter vitamins is nigh impossible.

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Conflicts of interest
There are no conflicts of interest.

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Sir,

A 56‑year‑old woman, with alleged history of hanging 10 days back, was rescued and taken to a local hospital. She was unconscious, intubated, and managed conservatively. At the time of admission to our hospital, her Glasgow Coma Scale was E₂V₃M₁, with quadriplegia, bilateral plantar reflex showing extensor response. Pupils were equal bilaterally, measuring 2 mm and responding to light. Initial magnetic resonance imaging (MRI) of the brain performed at an outside center [Figure 1] was reported as normal.

Repeat MRI of the brain obtained in our institute showed abnormal T2/fluid‑attenuated inversion recovery (FLAIR) hyperintensity of bilateral caudate nucleus and putamen, multiple cortices in bilateral temporo‑parieto‑occipital lobes [Figure 2a‑c], with effaced adjacent sulci. Subtle abnormal T2/FLAIR hyperintensity was seen in corpus callosum [Figure 2c], bilateral temporooccipital and parietal lobe white matter, with preservation of normal white matter in frontal lobes [Figure 2a and b]. Diffusion‑weighted imaging images showed abnormal hyperintensity in bilateral temporooccipital and parietal lobe white matter and corpus callosum [Figure 2d], corresponding region hypointensity in apparent diffusion coefficient [Figure 2e and f], suggestive of diffusion restriction. Based on MRI images, severe hypoxic brain injury was diagnosed.

Serial changes are seen in conventional MRI in Wallerian degeneration. No abnormal white matter T2 signal intensity changes will be seen during initial 4 weeks, T2‑weighted/FLAIR hypointensity seen during 4–10 weeks, and hyperintensity from 10 to 12 weeks onward. [1] Initial normal T2/FLAIR hyperintensity is due to intact distal axons, T2/FLAIR hypointensity during second phase is due to disintegration of axons and myelin along with alteration in protein‑lipid‑water ratio, while in late stage, T2/FLAIR hyperintensity is to gliosis. Adult diffuse hypoxic injury of prolonged duration causes extensive cortical damage, producing gyriform diffusion restriction in acute stage. Wallerian degeneration producing diffusion restrictions in subacute stage limited to projection fibers (pontocerebellar fibers, [2]corticospinal tract, [3]or subcortical U‑fibers only) [4] is described. Few atypical MRI findings of adult diffuse hypoxic injury such as (i) sparing of posterior circulation, [5] (ii) only basal ganglia involvement, (iii) selective occipital and perirolandic subcortical white matter involvement, (iv) motor cortex involvement, and (v) basal ganglia and visual cortex involvement [6] are also described. In the present case, extensive diffusion restriction and T2/FLAIR hyperintensity were noted involving cortex and white matter, with peculiar sparing of frontal lobe gray Figure 1: (a and b) Axial T2 and fluid‑attenuated inversion recovery magnetic resonance images showing normal gray and white matter, basal ganglia, and thalami, (c) sagittal T2 image showing normal signal intensity in corpus callosum.