Bilateral dentate nuclei hyperintensity due to isoniazid toxicity

Somarajan Anandan[1], Sajeesh S Rajendran[2], Jyothish P Kumar[3], Divine S Shajee[3]

From [1]Consultant Neurologist, [3]Resident, Department of Neurology, St Joseph Hospital, Anchal, [2]Consultant Neurologist, Department of Neurology, Welcare Hospital, Kochi, Kerala, India

A 57-year-old lady with an 8-year history of diabetes mellitus and a 3-year history of chronic kidney disease presented with a 3-day history of gait ataxia and dysarthria. There was no history of any other cranial nerve symptoms, weakness, or sensory symptoms. There was no history of fever, headache, or vomiting. On examination, she had scanning dysarthria, bilateral finger nose in coordination, and gait ataxia. There was no nystagmus. Motor system examination showed normal power and generalized hyporeflexia. Sensory system examination showed mild vibration loss in the toes bilaterally. She had a history of chronic cough and was started on anti-tuberculous therapy 1 month back (Isoniazid [INH] 300 mg/day, Rifampicin, Ethambutol, and Pyrazinamide) from a tertiary care hospital even though work up for tuberculosis was negative. She was not on pyridoxine. The evaluation showed S. creatinine 2.3 mg% and blood urea 70 mg%. Computerized tomography head was normal. Magnetic resonance imaging (MRI) brain showed symmetrical T2, fluid-attenuated inversion recovery (FLAIR), and diffusion-weighted image (DWI) hyperintensity in bilateral dentate nuclei. ADC did not show any signal changes (Figs. 1-3). Her INH was stopped and was started on pyridoxine 100 mg/day. Follow-up images could not be taken due to financial issues.

INH is considered safe in patients with kidney disease. Neurotoxicity due to INH usually presents with seizures, encephalopathy, or neuropathy. Cerebellar ataxia due to INH toxicity is rare. Toxicity was initially reported only in those patients on dialysis, receiving pyridoxine supplements of <100 mg/day. To prevent the neurotoxicity associated with INH therapy, pyridoxine 100 mg/day has been suggested as a supplement to hemodialysis patients requiring INH therapy [1]. Although metronidazole is the most common drug associated with such MRI signal changes in the dentate nucleus, the uncommon association with INH has been described in the literature, especially in patients with renal function impairment [2]. On MRI, INH toxicity causes symmetrical T2, and FLAIR hyperintensities involving dentate nuclei due to toxicity-induced edema. Bilateral dentate hyperintensities usually resolve by stopping INH and with pyridoxine supplementation. Bilateral dentate involvement may also occur in methyl bromide toxicity, enteroviral infections, maple syrup urine disease, canavan disease, glutaric aciduria Type 1, and atypical Wernicke's encephalopathy [3-5]. Similar changes

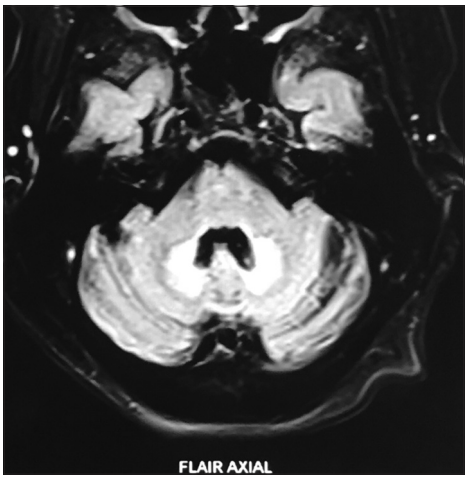

Figure 1: Magnetic resonance imaging brain axial fluid-attenuated inversion recovery image showing bilateral dentate nucleus hyperintensity

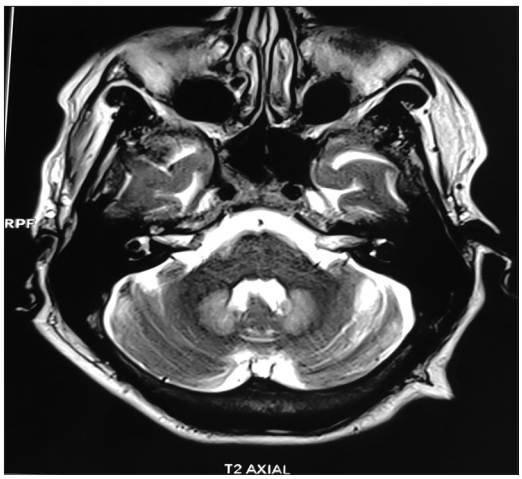

Figure 2: Magnetic resonance imaging brain T2-axial image showing bilateral dentate nucleus hyperintensity

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Correspondence to: Dr. Somarajan Anandan, Department of Neurology, St Joseph Hospital, Anchal 691306, Kerala, India. E-mail: drsomarajan@yahoo.co.in

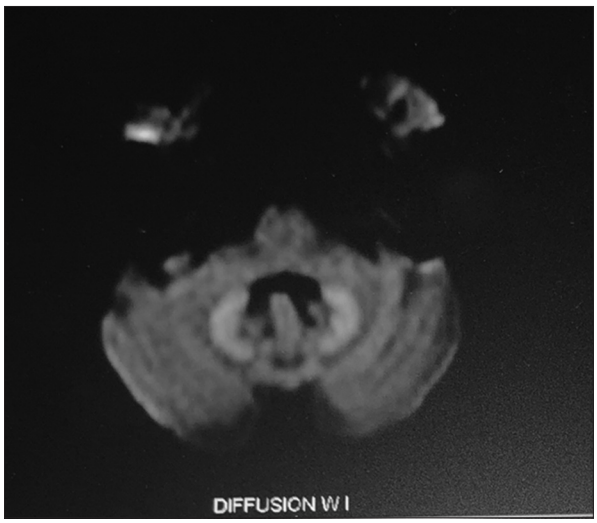

Figure 3: Magnetic resonance imaging brain axial diffusion-weighted image showing bilateral dentate nucleus hyperintensity. Corresponding ADC did not show any signal changes

have been described in cycloserine toxicity in tuberculous patients [6].

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