An unusual cause of reversible parkinsonism

Sir,

A 57-year-old male known case of diabetic nephropathy since 4 years recently started on biweekly hemodialysis presented with subacute onset of difficulty in walking and slowness in daily activities of 3 months duration. Gradually, the gait disturbance progressed to the extent that he became dependent for his daily chores. There was no fever, headache, vomiting, focal neurological deficit, cognitive decline, or seizures. There was no history of hypotension during hemodialysis or rapid correction of hyponatremia or hypernatremia. On neurological examination, he had flapping tremors but was oriented to time, place, and person. He had mask-like faces, slow hypophonic speech, and symmetrical bradykinesia. Gait examination revealed slow, short steppage gait with stooped posture, bilaterally decreased arm swing with difficulty in initiation and *en bloc* turning with intermittent episodes of freezing of gait [Video 1]. Rest of the neurological and systemic examination was normal, except for the presence of diabetic peripheral neuropathy.

Investigations showed fasting blood glucose of 110 mg/dl, elevated creatinine (8.9 mg/dl) with severe metabolic acidosis, and normal sodium and potassium levels. Magnetic resonance imaging (MRI) of the brain revealed diffuse edematous symmetrical T2 and fluid-attenuated inversion recovery hyperintensities in bilateral caudate and lentiform nuclei with mild diffusion restriction [Figure 1a and b]. Cerebrospinal fluid analysis was normal. He was started on levodopa without much improvement. The patient was subjected to daily hemodialysis, following which there was significant improvement in his symptoms. His speech and speed of walking improved markedly and episodes of freezing almost disappeared [Video 2]. Repeat MRI of the brain 1½ month later demonstrated marked diminution in bilateral basal ganglia hyperintensities [Figure 1c and d].

Bilateral basal ganglia lesions resulting in parkinsonian syndromes are rarely reported in patients with end-stage renal failure.[1,2] We report a patient of chronic renal failure who presented with subacute parkinsonism which gradually improved with improvement in renal functions. This suggests uremia with metabolic acidosis itself contributed to pathology in basal ganglia. Increased levels of uremic toxins inflict toxic metabolic injury to basal ganglia already damaged by microangiopathic changes as a result of long-standing diabetes mellitus. Some authors report increased cerebral blood flow in bilateral basal ganglia on SPECT images, thus implicating

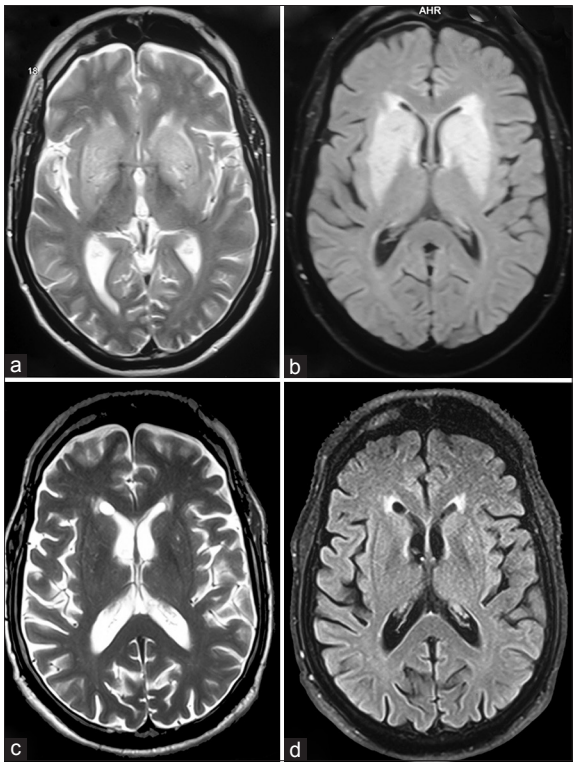

Figure 1: (a and b) T2-weighted and fluid-attenuated inversion recovery images showing bilateral symmetrical hyperintensities and swelling of basal ganglia. (c and d) Follow-up magnetic resonance imaging with T2-weighted and fluid-attenuated inversion recovery images showing resolution of hyperintensities in basal ganglia

vasogenic edema as the pathophysiologic correlate, while others have postulated cytotoxic edema as the cause based on diffusion restriction on MRI.[2,3]

Acute to subacute parkinsonism is rarely reported in uremia and is usually reversible with aggressive management of renal dysfunction.

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Conflicts of interest
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