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The Syndrome of Acute Bilateral Basal Ganglia Lesions in a Patient with Diabetes Mellitus and Uremia

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Full Text

Sir,

Common neurological complications of chronic renal failure include uremic encephalopathy, stroke, neuropathy, and myopathy.[1],[2] Acute extrapyramidal movement disorders associated with basal ganglia pathology have rarely been reported in patients with diabetic uremic condition and this syndrome has been reported mostly in Asian patients. The clinical presentation of acute bilateral basal ganglia lesions syndrome is characterized by acute parkinsonism or involuntary movements.[2],[3],[4],[5],[6] The brain magnetic resonance imaging revealed a unique cytotoxic-type edema in the bilateral basal ganglia during the acute phase. The hallmarks of uniform lesions in basal ganglia on magnetic resonance imaging (MRI) are reversible. The main laboratory test abnormalities consist of elevated blood urea nitrogen, creatinine, and metabolic acidosis. Although the pathophysiology of the basal ganglia lesion giving rise to parkinsonism in diabetic uremic patients is unknown, uremic toxins, metabolic acidosis, and diabetic microangiopathy are the accused factors in the pathology of disease development.[3],[4],[5],[6] After supportive treatment, these changes regressed or disappeared during follow-up. A patient with diabetic uremia who develops parkinsonian symptoms will be discussed.

A 70-year-old man who had arterial hypertension, hypercholesterolemia, diabetes mellitus, and uremia had been receiving continuous hemodialysis twice a week for two years. His diabetes had been regularly treated with oral hypoglycemic agents. He was admitted with gait disturbance, decreased movement, and slurred speech which appeared two weeks earlier. His neurological examination revealed parkinsonism with low monotonous voice, bradykinesia, muscle rigidity, and postural instability. There was no tremor. His eye blinking and facial expression were decreased. His gait was markedly hypokinetic. His arm swing was decreased. His deep tendon reflexes were normoactive. His cranial nerve function, sensation, and cerebellar function were intact.

There was no recent history of hypoglycemia, hyponatremia, hypoxia, carbon monoxide intoxication, or hypotension. Blood urea nitrogen level was 99 mg/dL (5–25 mg/dL) and creatinine level was 6.6 mg/dL (0.51–0.95 mg/dL). Before developing these problems, his blood urea nitrogen and creatinine levels were kept stable between 50 and 70 mg/dL and 5 and 5.9 mg/dL.

His blood glucose was 165 mg/dL and his HbA1c was 7.6%. The pH value was normal. Electrolyte levels were within normal limits. T2-weighted and FLAİR cranial MRI showed hyperintensity over the basal ganglia bilaterally [Figure 1] and [Figure 2]. Levodopa/carbidopa treatment was initiated. Hemodialysis was continued. The frequency of hemodialysis was increased from two to three times weekly. The blood urea nitrogen and creatinine decreased to previous stable ranges, and the hypokinetic gait and bradykinesia improved considerably. Follow-up cranial MRI studies performed two months later revealed regression of the basal ganglia lesions [Figure 3]. His movement disorder, gait, and other symptoms improved gradually after 6 months. Levodopa/carbidopa treatment was reduced and then stopped.{Figure 1}{Figure 2}{Figure 3}

Chronic renal failure's neurological complications such as uremic encephalopathy, stroke, neuropathy, and myopathy are common. An acute extrapyramidal disorder in association with neuroimaging features of bilateral symmetrical basal ganglia changes in diabetics undergoing hemodialysis is rare.[1],[3],[4],[5],[6] Wang et al. first described this syndrome in 1998.[3],[4],[5],[6] Extrapyramidal disorder includes hypo- or hyperkinetic movement disorders.[4],[6]

Basal ganglia need high energy for motor control. If cerebral metabolism decreases, basal ganglia may be selectively damaged. Differential diagnosis of bilateral basal ganglia lesions includes carbon monoxide intoxication, cerebral hypoxia, and extrapontine myelinolysis. These lesions are irreversible, while bilateral basal ganglia lesion of diabetic uremia is reversible.[2] The basal ganglia seem particularly susceptible to a variety of toxins and metabolites. The etiology of basal ganglia lesions includes factors such as uremic toxins, diabetic microangiopathy, metabolic acidosis, and hypoglycemia.[2],[3],[4],[5] Elevated uremic or metabolic toxins may disconcert the regional cellular metabolism or give rise to a functional disturbance in smooth muscle cells of vessel in the basal ganglia, which may lead to vascular autoregulatory dysfunction and then to vasodilatation and focal hyperemia.[7] FH may be accounted for the the breakdown of the blood-brain barrier. Basal ganglia lesions may be the result of vasogenic edema attributable to focal hyperemia secondary to abnormal dilatation of small vessels.[5] Wang et al. reported that glucose metabolism in the basal ganglia was decreased in uremic patients with parkinsonism.[8] Lee et al. reported a case who did not suffer from diabetes mellitus. Thus, Lee et al. speculate that vascular or metabolic factors associated with diabetes mellitus do not contribute to the pathogenesis of basal ganglia lesions.[5]

The basal ganglia lesions show decreased density in the basal ganglia in the cranial computerized tomography and decreased signal intensity in the cranial MRI on the T1-weighted images and increased signal intensity on the T2-weighted images over the same areas reported in the literature.[6] Diffusion-weighted images (DWI) and the apparent diffusion coefficient (ADC) map show that the matching areas have increased signal intensities with relatively high intensity on DWI and low intensity on ADC map in the central lesion. It is considered that some region of the basal ganglia may experience an irreversible cytotoxic damage leading to ischemia. The SPECT images revealed notably elevated regional cerebral blood flow in the bilaterally basal ganglia. Magnetic resonance angiography showed eminent lenticulostriate arteries. The findings show that the basal ganglia lesions seen in conventional cerebral MRI may be the result of focal hyperemia secondary to the abnormal dilatation of small vessels.[5]

The role of hypoglycemia in the basal ganglia lesions was also suggested by DWI. The lesions have a high signal on DWI and low signal in the central part of the concerned regions corresponding to cytotoxic edema on ADC maps.[2] These lesions often regress or disappear.[6] But irreversible cystic degeneration can occur in the medial areas of basal ganglia.[3],[4],[5],[6] The treatment is symptomatic and hemodialysis intensification.[6] Clinic symptoms improve spontaneously and brain MRI shows almost complete resolution of the lesions.

The parkinsonism in our patient occurred during a descend of renal function and accompanied a basal ganglia lesion. As the renal function was improved by increased frequency of hemodialysis, the parkinsonian features improved remarkably with regression of the basal ganglia pathology. This finding suggests that the uremia itself contributed to the basal ganglia pathology. This article aims to emphasize a patient who has all the typical clinical and neuroimaging findings for bilateral basal ganglia lesion syndrome.

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Conflicts of interest

There are no conflicts of interest.

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