

요독증 환자의 양측 기저핵 병변에 의해 발생한 급성 파킨슨증 1예

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박강민 · 김상진

Acute Parkinsonism with Bilateral Basal Ganglia Lesions in A Patient with Uremia

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A 47-year-old male who had hypertension and diabetes mellitus for 7 years suddenly developed bradykinesia, generalized limb muscular rigidity, dysarthria and dysphagia. Uremia developed 5 months prior to this and he had been on hemodialysis. A T2-weighted brain MRI showed extensive hyperintensity over the bilateral basal ganglia, extending to the adjacent periventricular white matter. In T1-weighted images the lesions were hypointense. Supportive treatments were given and his symptoms improved. Exacerbation of glucose utilization failure or vasogenic edema is suggested as the etiology of basal ganglia lesions, but the exact underlying pathophysiology is unknown.

Key Words: Uremia, Basal ganglia, Parkinsonis

Uremic encephalopathy is usually associated with cortical involvement in patients with uremia, as evidenced by frequent seizures and abnormal findings on electroencephalography.^{1,2} Involuntary movements in uremic encephalopathy typically consist of asterixis and myoclonus, which reflect cortical involvement.^{1,2} Other symptoms of abnormal movement disorder including parkinsonism and evidence of basal ganglia pathology as a consequence of uremia have been reported very rarely.³ A case of acute parkinsonism with bilateral basal ganglia lesions in a patient with uremia

is examined here.

Case report

A 47-year-old male visited to our hospital in June 2005 because of general weakness and gait disturbance. He had hypertension and diabetes mellitus for 7 years. The diabetes had been treated with oral hypoglycemic agents. Uremia was diagnosed in January 2005, and hemodialysis started in March.

Neurologic examination revealed mild bradykinesia, generalized rigidity, dysarthria, dysphagia and masked face. His gait was short-stepped and shuffling with mild postural impairment. His cranial nerve and cerebellar functions were normal. The deep tendon reflexes were normoactive and pathologic reflexes were absent. Blood chemistry showed elevated BUN (56 mg/dL) and creatinine (6.1 mg/dL) levels. The blood glucose level was 136 mg/dL and electrolyte levels were within normal

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limits.

A T2-weighted brain MRI performed 5 days after symptoms onset showed extensive hyperintensity over the bilateral basal ganglia, extending to the adjacent periventricular white matter. In T1-weighted images the lesions were hypointense (Fig. 1).

Supportive treatments and hemodialysis were given. His symptoms gradually improved. One month after symptoms onset, he had mild dysarthria but was otherwise perfectly well.

Discussion

Acute movement disorders and bilateral basal ganglia lesions in patients with uremia are a rare

distinct clinical syndrome. The lesions are symmetrical and have unique imaging characteristics. They are high signal intensity on T2-weighted images and low signal intensity on T1-weighted images with mass effect and perifocal edema.^{3,4} Our patient had uremia and suffered from acute parkinsonism associated with symmetrical bilateral basal ganglia lesions. Our case also exhibited high signal intensity on T2-weighted images and low signal intensity on T1-weighted images.

Wang et al. first reported similar findings in 1998 and 2003.^{3,4} All of their patients had both uremia and diabetes mellitus. They proposed the following as a possible underlying pathophysiology. The basal ganglia cellular functions had already been compromised by long-term diabetes

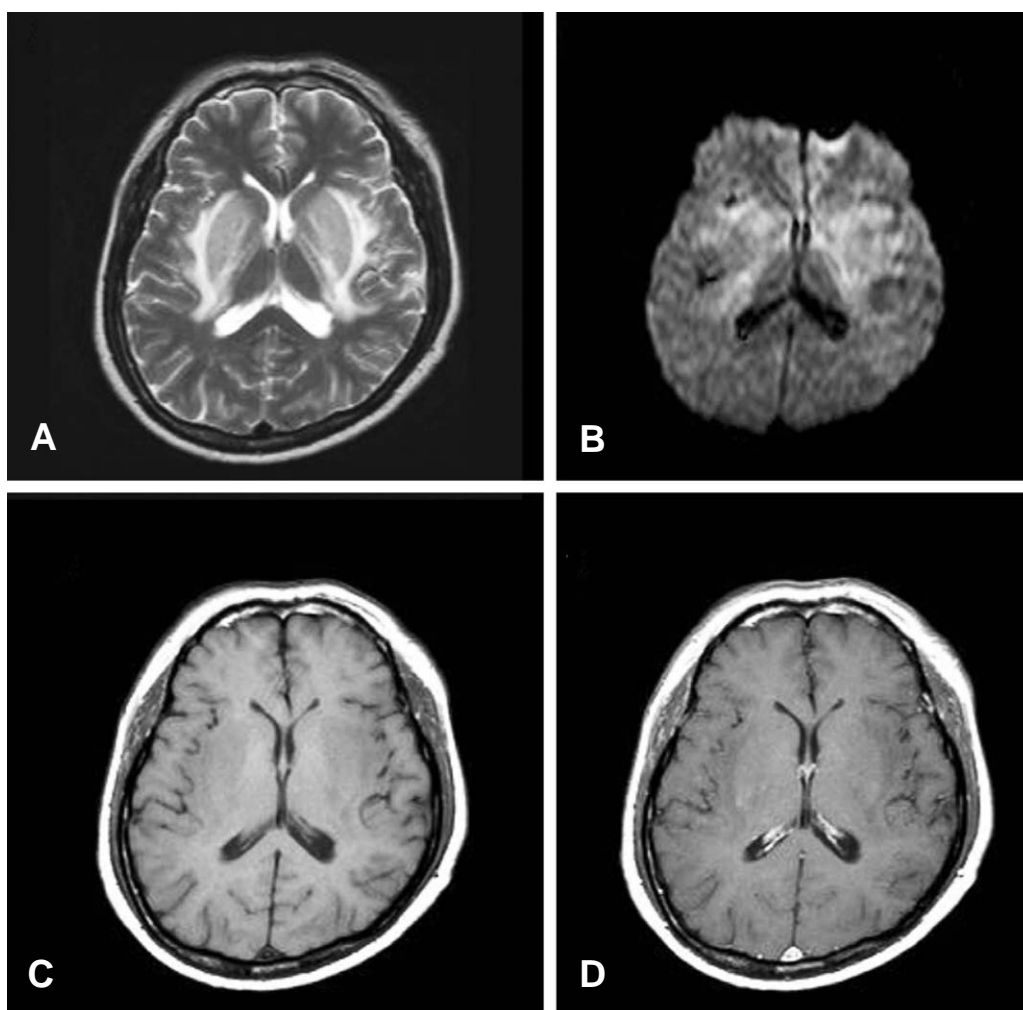


Figure 1. (A) T2 weighted MRI images show extensive hyperintensity over basal ganglia bilaterally, extending to the adjacent periventricular white matter. (B) On diffusion MRI images the lesions are slightly hyperintense. (C) T1 weighted MRI images show hypointensity on bilateral basal ganglia. (D) Gadolinium-enhanced T1 weighted images show slight enhancement of lesions.

mellitus through microangiopathic changes and energy utilization failure. When they were further exposed to markedly elevated levels of uremic toxins, their regional cellular metabolism and/or vascular autoregulation collapsed, and tissue was damaged.^{3,4} Wang et al. also reported a FDG-PET study.⁵ The two cases showed markedly reduced glucose metabolism in the basal ganglia, especially in the bilateral putamens where glucose uptake was nearly absent. They described that focal deficit of glucose utilization in the bilateral basal ganglia regions could explain these findings of FDG-PET study.⁵

But Lee et al. suggested vasogenic edema as the pathophysiology when examining diffusion weighted images and apparent diffusion coefficient maps in their case with uremia only.⁶ The exact etiology underlying these lesions is still unknown. Probably, pathophysiology of basal ganglia lesions is multifactorial including exacerbation of glucose utilization failure and vasogenic edema.

The prognosis for our patient was good, but most patients of Wang et al. died or required prolonged intensive care due to complications. Therefore, acute parkinsonism in uremic patients may require more careful observation and follow-

up. The definitive treatment of these patients is uncertain, but it is advisable to remove uremic toxins as soon as possible because of the proposed pathogenesis.

In conclusion, We have reported a rare distinct clinical syndrome of acute parkinsonism with bilateral basal ganglia lesions in a patient with uremia.

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