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## **Uremic Encephalopathy with Basal Ganglia Lesions in a Diabetic Predialysis Patient**

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### **Authors' contributions**

*This work was carried out in collaboration between all authors. Authors YNK and HSS wrote the protocol, and wrote the first draft of the manuscript. Authors YSJ and HR managed literature searches. All authors read and approved the final manuscript.*

**Case Study**

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### **ABSTRACT**

Syndromes associated with acute bilateral lesions of the basal ganglia in diabetic uremic patients are uncommon, and usually have reversible clinical and imaging findings. Such syndromes are seen almost exclusively in patients with diabetes mellitus and renal failure. Previously reported cases have described diabetic men with uremia on dialysis.

Here, we report a case of uremic encephalopathy with lesions of the basal ganglia in a diabetic predialysis patient. A 44-year-old man with uremic encephalopathy presented with dysarthria, chorea, and right upper extremity paresthesia. Magnetic resonance imaging of the brain showed classic findings of hyper intensity in the bilateral basal ganglia. The patient had no family history of psychiatric or neurological disease. Laboratory findings revealed elevated levels of blood urea nitrogen, creatinine, and glucose. Haloperidol and ropinirole therapy was continued, resulting in significant improvement without dialysis. The patient recovered from his episode without apparent sequelae.

**Keywords:** *Diabetes mellitus; uremic encephalopathy; basal ganglia; predialysis.*

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## **1. INTRODUCTION**

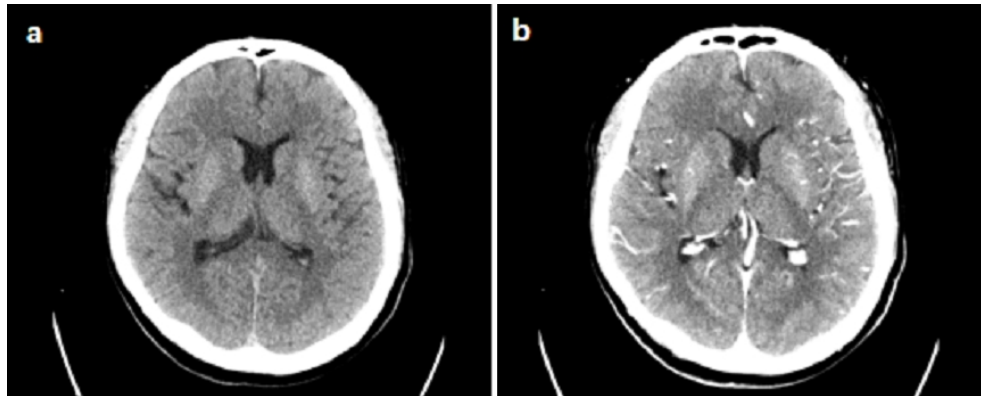
Uremic encephalopathy is one of many manifestations of uremia [1,2]. There are two types of radiological manifestations of uremic encephalopathy: those showing cortical involvement [1,3-4] and those showing basal ganglia involvement [5]. No pathophysiological associations of these two types have been determined. Uremic encephalopathy is typical of patients showing cortical involvement, with symptoms including confusion, seizures, tremors, or myoclonus [1,3-4]. Cases of acute extra pyramidal movement disorders, especially parkinsonism, have been associated with bilateral basal ganglia lesions in uremic patients [5-9]. A syndrome caused by acute bilateral basal ganglia lesions has also been reported in patients with diabetes and uremia [5,6], with symptoms including those seen in parkinsonism, disturbance of consciousness, dysarthria, dysphagia, dyskinesia, ataxia, and seizures [5-11]. Herein, we report a diabetic predialysis patient who developed acute chorea associated with bilateral basal ganglia lesions.

## **2. CASE**

A 44-year-old man with a history of hypertension and diabetes mellitus presented with diabetic nephropathy. In August 2011, he had an episode of dysarthria that lasted for 2-3 days. One day prior to his hospital admission, he suddenly developed abnormal involuntary movements, for which his family brought him to the emergency room. He denied any exposure to antipsychotics or antiemetics. The patient had no family history of psychiatric or neurological diseases.

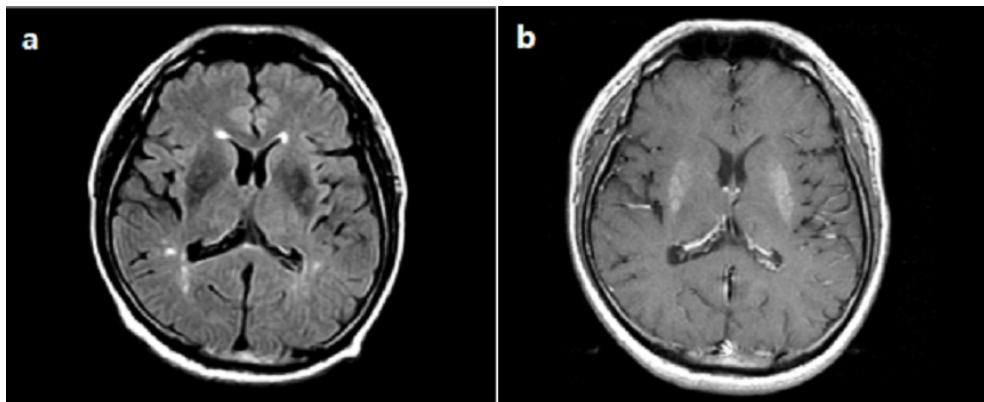
The patient's vital signs included a blood pressure of 110/70 mmHg, a heart rate of 98 bpm, a respiratory rate of 28 breaths/minute and a temperature of 36.9°C. His blood sugar was 530 mg/dL and his HbA1c was 16.4% A1c. Laboratory tests showed the following concentrations: 15.2 g/dL hemoglobin, 6,200/ $\mu$ L leukocytes, 208,000  $\mu$ L platelets, 28 mg/dL blood urea nitrogen, 1.9 mg/dL creatinine, 41 ml/min estimated glomerular filtration rate (eGFR) by MDRD (Modification of Diet in Renal Disease study) equation, 132 meq/L sodium, 4.1 meq/L potassium, 108 meq/L chloride, 9.3 mg/dL calcium, 3.6 mg/dL phosphorus, 1.7 mg/dL magnesium, 20 IU/L AST, 16 IU/L ALT and 0.8 mg/dL total bilirubin. Arterial blood gas analysis was not performed and Urinalysis showed negative finding for ketones. All other laboratory findings, including thyroid hormone levels (T3,T4,thyroid stimulating hormone) and thiamine levels, were within normal limits. After the symptoms disappeared, laboratory parameters showed the following concentrations: 21 mg/dl blood urea nitrogen, 1.6 mg/dl creatinine, 48 ml/min eGFR by MDRD equation, 404 mg/dl blood sugar. An initial brain computed tomography (CT) scan, performed at the emergency room, showed no abnormal findings of enhancing lesions (Fig. 1).

During the patient's neurological examination, irregular, arrhythmic, continuous, and rapid involuntary movements were noted. These were especially noticeable in the right hemibody, as is characteristic of chorea. The patient could not suppress these involuntary movements, which sometimes interfered with voluntary movements such as standing and walking. The involuntary movements disappeared during sleeping. The patient also had right upper extremity paresthesia. He did not have any cortical dysfunctions, and his motor systems were normal and symmetrical. Because of patient's non-compliance, we could not perform cognitive tests.



**Fig. 1. An initial brain computed tomography (CT) scan, performed at the emergency room, did not show abnormal findings. (a: non-enhanced image, b: enhanced image)**

A T1-diffusion-weighted magnetic resonance imaging (MRI) scan was taken the day the patient was admitted. It showed a hypointensity in the both basal ganglia and no evidence of acute ischemia or intracranial hemorrhage (Fig. 2, a). A brain MRI scan was performed five days after admission. It showed bilateral basal ganglia signal changes that were hyperintense on a T1-gadolinium image (Fig. 2, b).



**Fig. 2. The T1-diffusion-weighted magnetic resonance imaging (MRI) scan (a), taken the day the patient was admitted, showed no evidence of acute ischemia or intracranial hemorrhage. The brain MRI scan (b), taken five days after the patient was admitted, showed bilateral hyperintensity in the basal ganglia on a T1-gadolinium image**

The patient's choreic movements began to slowly improve. His neurologic symptoms also improved gradually with a conservative management course that included haloperidol and ropinirole. He did not receive any dialysis. On the eighth day of his admission, he was transferred to the Endocrinology Department for BST control. His symptoms resolved completely within ten days, and he was discharged in a stable condition. During the eight months of follow-up, there were no signs of symptom recurrences. We could not recheck MRI due to patient' denial.

### **3. DISCUSSION**

Here we describe a diabetic male patient with chronic renal failure who suddenly developed choreic movements associated with bilateral basal ganglia lesions. No cases of bilateral basal ganglia changes in diabetic predialysis patients have been previously reported. The most commonly reported clinical manifestation of bilateral basal ganglia lesions in uremic patients is parkinsonism (bradykinesia, rigidity, postural instability, and gait disturbance with no resting tremor), followed by dysarthria, consciousness disturbance, dyskinesia, and dysphagia [5,9]. It is possible that the right upper extremity paraesthesia is a sign of cortical lesion, although MRI did not show abnormal signal intensities on cortical areas of brain.

Although the exact pathogenesis of uremic encephalopathy is unknown, multiple chemicals have been implicated as toxins [1]. However, the serum toxin levels are not elevated in uremic patients with acute movement disorders [5]. Another possible cause of extrapyramidal symptoms is thiamine deficiencies [12]. Due to blocking of the citric acid cycle, it is possible for a thiamine deficiency to lead to cellular hypoxia of the basal ganglia and extrapyramidal motor dysfunctions [12]. However, in this case, the serum thiamine level was within the normal range. Usually, basal ganglia lesions in uremia develop in patients who are on inadequate hemodialysis [5-7] or in patients who are not on hemodialysis [13]. Our choreic patient was not on hemodialysis.

In terms of the clinicoradiologic aspects of this case, the acute onset and signal changes on the MRI (hyperintensity on a diffusion T1-weighted image) suggested an ischemic insult. There was no hyperintensity was evident at FLAIR (data was not shown). However, it would be unusual to see ischemic results in a thromboembolic disease [5] for two reasons [1]. First, bilateral basal ganglia lesions in this case showed hyperintensities on the diffusion T1-weighted image [1]. These findings suggest that the pathomechanism of the perifocal edema might be vasogenic edema [1]. A recent report [9] used MR angiography and SPECT to demonstrate that vasogenic edema in the basal ganglia resulted from focal hyperemia secondary to abnormal vasodilatation. Second, the basal ganglia lesions described here were symmetrical. Symmetrical signal changes on brain MRIs can be seen in patients with hypoxia or metabolic disorders such as hyperglycemia, liver cirrhosis and Wilson's disease [1]. However, there was no history of hypoxic brain damage in our patient's case [1]. During the patient's neurological examination, irregular, arrhythmic, continuous, and rapid involuntary movements were noted on whole body. These were especially noticeable in the right hemibody, as is characteristic of chorea.

It has been shown that vascular autoregulatory dysfunction, as may be seen in diabetic patients, can interfere with dopamine metabolism in the basal ganglia [14]. Since most patients with the syndrome have a long history of diabetes, it may be possible that basal ganglia injuries occur more easily when the adverse effects of diabetes and uremia are combined [16]. Our patient had both diabetes and uremia. Hyperglycemia-induced brain lesions and potential underlying mechanisms, such as increased blood-brain barrier permeability, have been shown to cause such effects [17,18]. After the symptoms disappeared, the level of blood sugar was high as symptoms were present. So, we thought uremia may be main cause of encephalopathy, although it cannot be ruled out that hyperglycemia was preceded by hypoglycemia in this patient with poorly controlled diabetes. In a patient who is both diabetic and uremic, diabetes mellitus may make the basal ganglia more vulnerable and exacerbate damages caused by uremic toxins [19]. His neurologic symptoms improved gradually with a conservative management course that included haloperidol and ropinirole for dopaminergic and antidopaminergic effect.

#### **4. CONCLUSION**

Our case contributes to evidence for the risk of basal ganglia injury in predialysis patients with diabetes [5].

In conclusion, bilateral basal ganglia lesions in diabetic uremic patients may have been caused by the combined input of microvascular dysfunction and uremic toxin accumulation. Larger epidemiologic studies should be conducted to establish the prevalence of this disorder in chronic kidney disease patients and to explore causative associations between basal ganglia injuries, diabetes and uremia-related risk factors.

#### **CONSENT**

In this paper, only published data from the literature were used for description. Thus, a statement of patient consent is not applicable for this paper.

#### **ETHICAL APPROVAL**

Since data from the literature, only, were used for description, ethical approval is not applicable to this paper. This study is not against the public interest. All authors hereby declare that all description have been performed in accordance with the ethical standards laid down in the 1964 Declaration of Helsinki.

#### **COMPETING INTERESTS**

Authors have declared that no competing interests exist.

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