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Case Report

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A rare presentation of uremia: akathisia

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ABSTRACT

Acute movement disorders associated with bilateral basal ganglia lesions are becoming more common in patients with diabetes mellitus and uremia. Pathophysiology is not fully known, although it is believed to be complex, with ischemic/microvascular as well as metabolic/toxic variables influencing lesions and symptoms. We have reported here a uremic diabetic patient who has sudden developed severe akathisia, in Magnetic Resonance Imaging (MRI) showed bilateral symmetric basal ganglia lesions with regression at follow-up. A condition linked with acute bilateral basal ganglia lesions in diabetic uremic individuals is uncommon, with clinical and imaging data demonstrating reversible alterations. Akathisia secondary to uremia is rarely seen in the literature. Our goal is to improve awareness of this condition among doctors and radiologists in order to identify more cases.

Keywords: Akathisia, uremia, diabetes mellitus, basal ganglia

INTRODUCTION

Uremia is a clinical and metabolic condition that develops in tandem with the decline of renal function. A well-known uremia consequence is brain involvement. The neurological effects of uremia are similar in many ways to the impact of other metabolic and toxic illnesses on the central nervous system.[1]

Acute movement abnormalities with bilateral basal ganglia involvement in diabetic uremic individuals have been reported in numerous recent case reports. The imaging features of this condition include symmetric bilateral basal ganglia lesions. The etiology behind it is yet unclear.[2-6]

Parkinsonism (bradykinesia, stiffness, postural instability, and gait abnormalities with no resting tremor) is the most frequent clinical symptom of bilateral basal ganglia lesions in uremic patients, followed by dysarthria, consciousness disturbances, dyskinesia, and dysphagia.[2,3]

Akathisia is a restless motor condition that most often occurs as a side effect of various medications in individuals receiving neuroleptic treatments. It's a neuropsychiatric disorder characterized by psychomotor restlessness. A person with akathisia will typically have a significant sense of discomfort or inner restlessness that affects their lower extremities.[7]

Akathisia secondary to uremia is rarely seen in the literature. We report here a case of sudden developed severe akathisia with basal ganglia lesions in a 63-years-old diabetic uremic patient. We discuss the possible etiology of this disease, as well as its clinical symptoms, laboratory results, Magnetic Resonance Imaging (MRI) findings, and clinical outcomes.

CASE

A 63 year old man was brought to our emergency department due to sudden acute motor restlessness, irritability, sleeplessness, dysphoria, as well as meeting the akathisia criteria. The most significant symptom was akathisia, which scored 10/14 on the Barnes Akathisia Rating Scale (BARS).[8]

Although cerebellar function tests were normal, akathisia was accompanied by minimal imbalance and extrapyramidal type dysarthria. His cranial nerves and peripheral nervous system were intact. Deep tendon reflexes were normal, and the Babinski reflexes were negative. The patient's vital signs

were normal, and there were no complaints of a headache, fever, impaired vision, or mental illness. He has never taken any medicine that has the potential to cause akathisia.

He had diabetes mellitus (DM), hypertension and uremia had received regular hemodialysis 3 times per week. There was no history of psychiatric or neurological illness in the family, including Huntington's disease.

Blood urea nitrogen (BUN:21.9 mg/dl) and creatinine (6.05 mg/dl) levels were high in the blood chemistry examination, but all other values were within reference limits. His blood glucose level was 120 mg/dl.Bilateral basal ganglia lesions were hyperintense on FLAIR-T2 weighted images according to brain MRI (Figure 1).

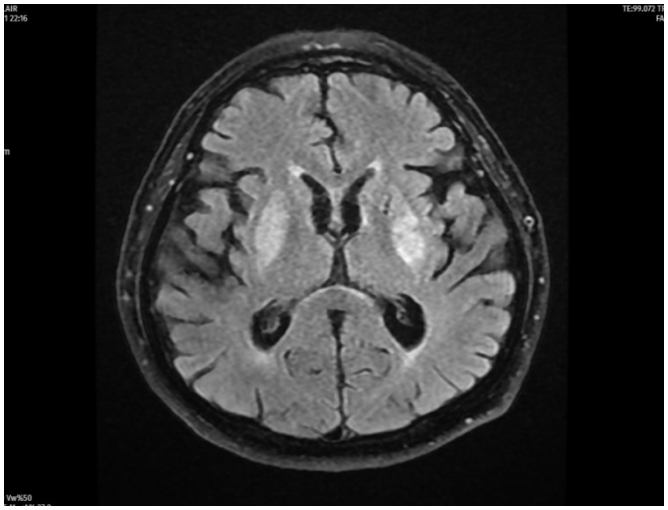

Figure 1. FLAIR image shows increased signal intensity in both basal ganglia lesions (white arrows indicate lesions).

He was not given any specific medication aside from hemodialysis. Hemodialysis was used more frequently. His irritability gradually improved in five days .One month following the initial MRI, a follow-up MRI of the brain was done, which revealed full remission of the basal ganglial abnormalities bilaterally (Figure 2). His neurological exam was normal.

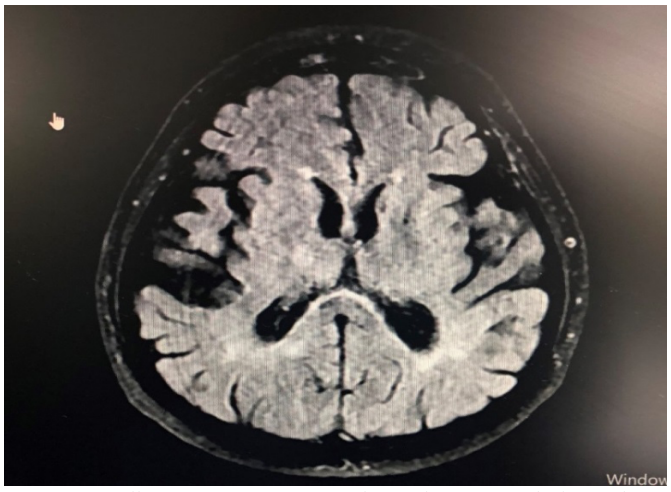

Figure 2. Follow-up FLAIR image obtained one month later shows significant regression of the lesions.

DISCUSSION

Acute movement disorders associated with bilateral lesions are increasingly described in patients affected by diabetes and uremia, it was first described by Wang et al.[3] The syndrome's most prevalent clinical symptoms are parkinsonism, gait abnormalities, dysarthria, and bradykinesia.[3,4,6] In our case the patient had akathisia.

The exact etiology of this condition is uncertain, and different researchers have suggested different theories. Uremic toxins, metabolic acidosis, and diabetic microangiopathy are only a few of the reasons that have been linked to it.[9]

Lee et al.[4] revealed the vasogenic origin of bilateral basal ganglia oedema, attributed it to localized hyperemia caused by abnormal small artery dilation, in 2006. Furthermore, they have demonstrated that diffusion-weighted imaging (DWI) may reveal tiny areas of cytotoxic oedema within confluent lesions of vasogenic oedema. Some areas of the basal ganglia lesions may undergo irreversible cytotoxic damage.

Furthermore, sympathetic dysautonomia, cerebrovascular reactivity impairment, and endothelial dysfunction in cerebral arteries may arise in people with long-standing diabetes. Hyperglycemia impairs endothelium-dependent vasoreactivity of cerebral arterioles, causes localized damaging endothelial lesions, leads to blood–brain barrier collapse, and increases free radical release.[10,11]

As a result, the cellular activity of the basal ganglia in these diabetic uremic individuals had already been impaired by long-term diabetes mellitus, either through microangiopathic alterations or energy usage failure. Furthermore, when the basal ganglia were exposed to significantly higher levels of uremic or metabolic toxins, regional cellular metabolism may have been disrupted, or a functional disturbance in smooth muscle cells of the vessels of the basal ganglia may have been induced, leading to vascular autoregulatory dysfunction and, ultimately, vasodilatation and focal hyperaemia.[2,4,12,13] Significant changes in cell metabolism and the collapse of vasogenic autoregulation occur as a result of this prolonged metabolic and toxic stress, resulting in tissue damage and oedema. Movement abnormalities are caused by alterations in physiological processes.[3]

In our patient rather than hyperglycemia, the most evident metabolic abnormality was significantly increased blood urea nitrogen and creatinine levels. The basal ganglia are especially vulnerable to a wide range of toxins and metabolic abnormalities. The acute exposure of the basal ganglia to uremic toxins was most likely caused by the worsening of the renal condition.[2-5] In our case, applying this hypothesis implies tissue oedema including the bilateral basal ganglia, and therefore full resolution with no lasting alterations. After increasing the number of hemodialysis treatments, our patient's neurological problems were completely resolved within 5 days. A month follow-up MRI revealed that the bilateral basal ganglia lesions had completely resolved.

The clinical result differs depending on the case series. According to a study of the literature, clinical abnormalities

may be resolved completely in one-fifth of the cases, partially in half of the cases, and not at all in 30% of the instances.[14] In over 90% of the instances, the radiologically detected abnormalities are resolved.[14,15]

Both basal ganglia are involved in many other disorders at the same time, including vascular abnormalities, toxic agent ingestion or inhalation, metabolic diseases, neurodegenerative diseases, demyelination, haemorrhage, infectious encephalitis, developmental anomalies, and neoplastic disease.[16-18] Our patients clinical symptoms, were not indicative of any of these diseases.

Acute akathisia is most common with neuroleptic and antidepressant therapy.[7] Akathisia is a disorder characterized by extreme restlessness. It consists mostly of two parts: i) a sensory component that includes feelings of inner restlessness, a desire to move, distress, and ii) a motor component, expressed as sensation-induced motions.[19]

Akathisia is diagnosed only on the basis of clinical observation and patient description because no confirming blood test, imaging examination, or neurophysiological investigation is available. The BARS, a 4-item scale in which the subjective and objective components of the disease are scored individually, then combined, is the most often used instrument for assessment.[8]

A movement disorder is the objective symptom of akathisia. When the condition is mild to moderate, the lower extremities are frequently the first to be affected. From the hips to the ankles, the motions are in the shape of standing in different postures and swaying or moving while sitting, the feet around. The fact that akathisia is more likely to affect the lower extremities than other antipsychotic-induced side effects that affect other body regions is sometimes useful in distinguishing it from other antipsychotic-induced side effects that affect other body regions. Although it is usually thought of as a sort of movement disorder or extrapyramidal system (EPS), akathisia is more of a sensorimotor disorder due to the strong sensory component that is a distinguishing feature of the illness. In reality, the sensory component might be the main issue, with motor indications emerging as a result of the restlessness and urge to move.[19]

Our patient have severe body restlessness, irritability, sleeplessness. And he said readily eased by altering posture or moving a limb. His motor symptoms were a result of his restlessness and need to move.

Although the etiology of akathisisa is unknown, positron emission tomography (PET) investigations have indicated that D2 (dopamine) receptor blockage in the striatum may play a significant role, and noradrenergic and serotonergic systems appear to be implicated.[5,7]

There appears to be dopamine receptor blockage in the mesocortical dopamine pathway as well the motor effect is inhibited by the mesocortical circuit. This pathway's postsynaptic blockage is considered to be the cause of akathisia.[20] Uremic toxins affect basal ganglia metabolism, including dopamine turnover. This might be aided by uncontrolled hyperglycemia. Akathisia might be caused by impaired dopamine turnover and increased sensitivity of postsynaptic dopamine receptors.[7,20] In literature there is only one case reported akathisia secondary to uremia.[21]

CONCLUSION

We provide a very uncommon case, it is the second case of reversible acute symmetrical basal ganglial lesions detected on MRI, which were linked to diabetic uremia and characterized as acute onset of akathisia. Our aim is to raise awareness of this disease among clinicians and radiologists so that more instances may be identified.

ETHICAL DECLARATIONS

Informed Consent

The patient signed and free and informed consent form.

Referee Evaluation Process

Externally peer-reviewed.

Conflict of Interest Statement

The authors have no conflicts of interest to declare.

Financial Disclosure

The authors declared that this study has received no financial support.

Author Contributions

All of the authors declare that they have all participated in the design, execution, and analysis of the paper, and that they have approved the final version.

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