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Case Report

Sudden Akathisia and Parkinsonism in a Uremic Patient

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Abstract

The authors report here a case of sudden developed severe akathisia and parkinsonism with basal ganglia lesions in a 52-years-old diabetic uremic patient. Magnetic resonance of brain showed hyperintensities on T1 weighted images on bilateral basal ganglia.

Keywords: Acute akathisia, üremi, MRI

Üremik Bir Hastada Ani Gelişen Akatizi ve Parkinsonizm

Özet

Burada, ani gelişen şiddetli akatizi, parkinsonizm tablosu ve bazal gangionlarında lezyonları olan 52 yaşındaki bir hastayı sunduk. Beyin manyetik rezonans incelemesinde iki taraflı bazal ganglionlarda hiperintensite tesbit edildi.

Anahtar Kelimeler: Akut akatizi, parkinsonizm, uremia, MR

INTRODUCTION

The involuntary movements seen in uremic patients are mostly myoclonus and asterixis[(5)]. Acute parkinsonian feature in uremic patients have been rarely described, and the pathophysiology of this acute syndrome is not fully understood[(3,8,9)]. Akathisia is a motor syndrome of restlesness, occur mostly in patients during the neuroleptic treatment as side effect of some drugs. The clinical feature of akathisia are varied and may range from subtle to lethality, in the form of suicide. Patients have restlessness and an inability to keeping stil, and also they may show complex or stereotyped movements[(4)]. Here, we report a patient, who has chronic renal failure with diabetes mellitus, and developed suddenly isolated severe akathisia and mild parkinsonism with bilaterally basal ganglia lesions. The difference between our case and the cases in literature[(3,8,9)] is; our patient was presented with prominent akathisia conversely parkinsonism.

CASE PRESENTATION

A 52-year-old men admitted to our clinic because of sudden severe body restlessness, irritability, insomnia, dysphoria, also fullfilling the criteria of akathisia. Akathisia was most prominent feature, with BARS (Barnes Akathisia Rating Scale) score of 11/14, which was consistent with severe akathisia. Additional to these findings, the patient was alert, irritable, speeking loudly, and was in conflict with his environment. Mild imbalance and extrapiramidal type dysarthria accompanied to akathisia, but cerebellar function tests was normal. There was mild parkinsonian feature; rest tremor,

limb rigidity, bradykinesia and cogwheel phenomenon was observed. He had no history of using any drug could lead to akathisia. His mental functions was normal, there were no aphasia, apraxia and agnosia. He had diabetes mellitus for one year, chronic renal insuffiency for 6 years and until two months before admission he received regular hemodialysis 3 times per week. His blood glucose was not regular with insulin and oral antidiabetics. There was no hypertension and other any systemic illness or intoxication history.

Blood chemistry analysis revealed elevated blood urea nitrogen (BUN; 44 mg/dL), serum creatinin (8,8 mg/dL) levels. His blood glucose level was 138 mg/dL and aluminum level was within normal levels (53 µgr/L). Supportive management and hemodialysis were administered. His irritability gradually improved. Within two weeks all the akathisia symptoms disappeared with oral anticolinergic (Biperidene) treatment.

Imaging

Brain Magnetic Resonance (MR) imaging showed bilateral basal ganglia lesions that were hypointense on T1 weighted images and hyperintense on T2 weighted images (Fig.1). On diffusion-weighted image (DWI) and the apparent diffusion coefficient (b-values=0 and 1000 s/mm^2, ADC) map the corresponding areas had increased signal intensities (Fig.2). Follow-up brain MRI one month later showed diminution of basal ganglia lesions (Fig.3), One month after admission, 99m Tc-ethyl cysteinate dimer was given intravenously and SPECT images were obtained using a CamStar AC/T one head gama camera equipped with low enegry all purpose (LEAP) collimators (GE, Milwaukee, Wisconsin, USA). This images revealed markedly decreased regional cerebral blood flow in the right basal ganglia (Fig.4).

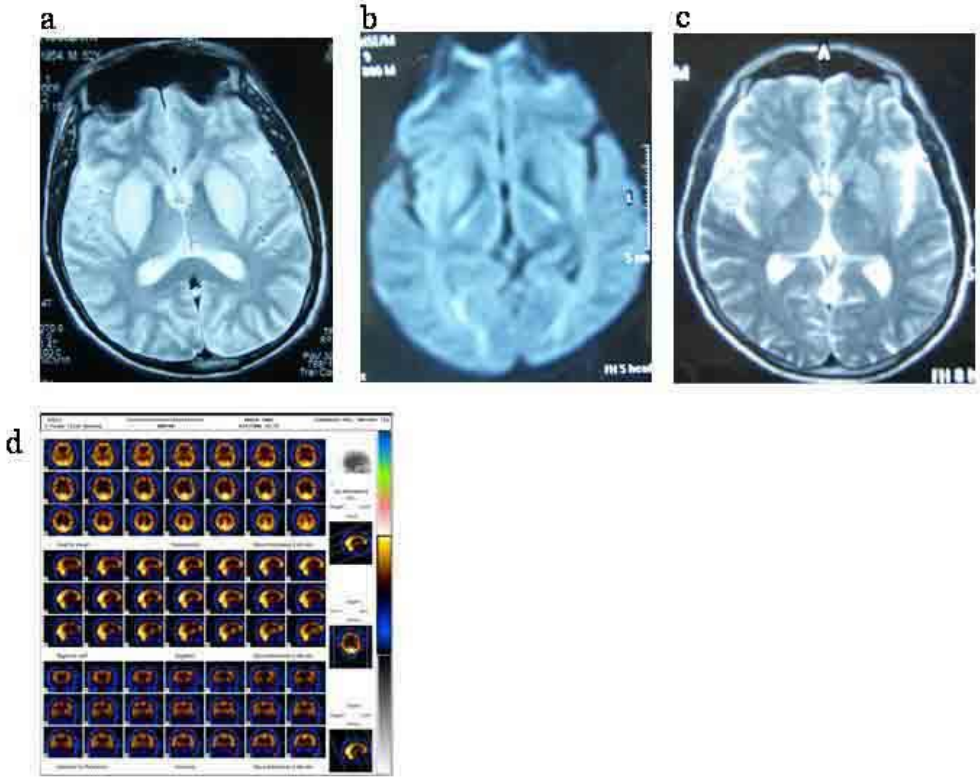

Figure 1: *(a) T2 weighted magnetic resonance image of brain showed hyperintensities on basal ganglia. (b) Diffusion weighted image (DWI) showed limited increased signal intensity on basal ganglia. (c) Follow-up MRI one manth later showed diminution of basal ganglia lesions. (d) Brain SPECT images one month later showed decreased regional blood flow in the rigt basal ganglia.*

DISCUSSION

Acute akathisia occur mostly during the neuroleptic and antidepressant treatment, but some other drugs like metoclopramide, some calcium channel blockers, dopamine agonists, antimalarials, buspirone and amphetamine may cause akathisia[4]. Stuppaeck et al. reported akathisia associated carbon monoxide intoxication[7], Carrazzana et al. reported akathisia with subtalamic toxoplasmosis abscesses[1].

Although the pathogenesis of this syndrome is still obscure, PET studies revealed D2 receptor blockade in striatum may play major role and noradrenergic and seronergic systems appear to be involved[4,9] This would explain akathisia and EPS induced by antidepressants and the positive response to 5-HT2 antagonists and beta blockers. Additionally there appears to be dopamine receptor blockade in the mesocortical dopamine system[2]. The mesocortical pathway has an inhibitory effect on motor effect. Akathisia is thought to be a product of postsynaptic blockade of this pathway. The association between low serum iron level and akathisia has been not exactly showed[2]. Drugs which have been found to have some efficacy in the treatment of akathisia are: Anticholinergics, Beta blockers, Benzodiazepines, Cyproheptadine, Clonidine, Mianserin. anticholinergics are probably most useful when akathisia is accompanied by parkinsonian side effects, thereby we treated the patient with Biperidene[4].

In our case; the initial brain MR images, high intensity images on diffusion-weighted MR and decreased blood flow in basal ganglia by SPECT showed that the lesions were associated with focal ischemia and edema. Ischemia and edema were limited to basal ganglia which may seen in cerebral hypoxic events[6]. However, our patient has no history of hypotension or anoxia, and he had rapid improvement that may not seen by hypoxic events. DWI analysis revealed that alterations of the bilateral basal ganglia in the acute stage are not the result of extracellular edema, but rather the result of intracellular edema. As we know, uremic toxins impair basal ganglia metabolism such as dopamin turnover. This may be facilitated by uncontrolled hyperglicemia. Impaired dopamin turnover and increased sensitivity of postsynaptic dopamine receptors could lead to akathisia[2,4]. Similar to our case, the neuroimaging changes of the syndrome regressed or disappeared in the majority of reported cases, after a period of several weeks.

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