

Short Communication Article

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Assessment of Parkinson's Disease-like Symptoms and Sign sin Cocaine Addicts

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Introduction

Parkinson's disease (PD) is a prevalent neurological disorder caused by dopaminergic (DA) deficiency in the striatum, which results from neuronal degeneration in the substantia nigra [1,2]. Clinically, PD is characterized by motor impairments such as resting tremor, rigidity, bradykinesia, postural instability, and other related symptoms [3]. Drug addiction, on the other hand, is a chronic brain disorder involving substances such as heroin, cocaine, and others [4]. An extensive body of research has documented the impact of addictive drugs on the dopaminergic system in both animal models and humans. Postmortem analyses of chronic cocaine users have revealed decreased levels of striatal dopamine, dopamine transporter (DAT), and vesicular monoamine transporter 2 (VMAT2). Furthermore, neuropathological specimens from such individuals showed increase in alpha-synuclein levels within dopaminergic cell groups of the substantia nigra/ventral tegmental complex compared to normal subjects.

This elevation in alpha-synuclein may reflect a compensatory mechanism against altered dopamine homeostasis induced by chronic cocaine exposure; however, its accumulation could simultaneously elevate the risk of developing Parkinson's disease-like motor abnormalities in affected individuals. Supporting this notion, neuroimaging studies have found that abstinent methamphetamine users, similar to Parkinson's patients, exhibit significantly reduced DAT density in the caudate and putamen-a finding that suggests a heightened vulnerability to parkinsonism with advancing age. Based on findings of dopamine deficiency in both drug abusers and PD patients, this study sought to determine

whether parkinsonian signs occur in chronic drug abusers. Cocaine is one of the most widely used addictive drug in China. We systematically assessed PD-like symptoms and signs among 34 cocaine addicts and 20 age-, sex-, and education-matched control subjects using the Unified Parkinson's Disease Rating Scale (UPDRS) [5].

The addicts were selected from over a hundred drug abusers. All addicted subjects met the following criteria: 1) Presence of cocaine metabolite (morphine) on urine toxicology screens; 2) Diagnosis of dependence according to DSM-IV (Diagnostic and Statistical Manual of Mental Disorders, Fourth Edition); 3) Absence of other drugs of abuse except for ethanol and cigarettes; 4) History of cocaine abuse for at least one year and abstinence for more than three months prior to the study; 5) Absence of other neurological diseases. Among the 34 addicts, 21 were male and the rest were female, aged between 24 and 39 years (31.8 ± 6.3 years). The duration of drug abuse ranged from 2 to 8 years (4.6 ± 3.4 years). The abstinence period prior to the study was 2-5 months (4.4 ± 1.9 months). Individual daily cocaine consumption ranged from 0.01 to 0.90 (0.4 ± 0.2) grams. The maximum reported daily cocaine consumption per subject was between 0.2 and 4.5 (0.7 ± 0.7) grams. Twenty (14 male) non-drug-abusing control volunteers were also recruited, matched for age, sex, and educational level. All subjects provided written informed consent for the study.

Clinical assessment methods for PD include the UPDRS, Activities of Daily Living (ADL) scale, Mini-Mental State Examination (MMSE), etc. A medical resident assessed the symptoms of addicts

using Parts I and II of the UPDRS. Another experienced neurologist performed the motor system physical examination constituting Part III of the UPDRS. Twenty-one addicts completed all three parts of the UPDRS assessment, while 13 provided scores only for Parts I and II. All control subjects except one completed three parts of the assessment. Statistical analysis was performed using SPSS.

Addicts had significantly higher scores than the control group across all three parts of the UPDRS (4.4 vs. 1.2, 3.0 vs. 2.3, 1.6 vs. 0.7, $P_s \leq 0.05$). Longer duration of abuse, history of relapse, and intravenous administration were all associated with higher UPDRS scores. A significant correlation was found between the duration of drug abuse and UPDRS scores ($r=0.20$, $P<0.05$). Addiction causes cumulative damage to the brain, explaining why a longer addictive period leads to higher UPDRS scores. Addicts with a history of relapse had significantly higher scores across all UPDRS parts than those without a relapse history (4.3 vs. 3.2, 4.1 vs. 3.2, 1.7 vs. 1.4, $P_s<0.05$). This appears consistent with the existing hypothesis that perturbations in DA transmission are potential substrates for relapse [27]. Addicts with a history of intravenous (IV) administration had significantly higher scores across all UPDRS

parts than non-IV users (4.8 vs. 3.5, 4.1 vs. 3.2, 1.5 vs. 0.8, $P_s<0.05$). Intravenous administration can affect addicts in multiple ways. HIV infection may potentiate the severity of psychomotor symptoms associated with drug abuse. As shown in Table 1, the percentage of PD-like signs (including tremor, bradykinesia, postural instability, and gait abnormality) was much higher in addicts than in control subjects.

It has been reported that acute or chronic abuse of addictive substances, including heroin and cocaine, can induce symptoms and signs similar to PD. However, earlier publications on drug-abuse-induced motor dysfunction are sparse. For instance, although inhalation of heroin vapours is common, few cases of leukoencephalopathy have been reported to date. Furthermore, earlier reports were sometimes contradictory regarding the neurological symptomatology and pathology of drug addicts. A study [6] that examined 50 male patients with a history of chronic heavy cocaine abuse using the Unified Parkinson Disease Scale (UPDS) found no significant difference in UPDS scores compared to control subjects [7]. A limitation of that study was the small number of subjects.

Table 1: Percentage of occurrence for characteristic signs of PD.

	Tremor	Bradykinesia	Postural Instability	Gait Abnormality	Sense Abnormality
Addicts	31.3%	46.4%	18.0%	17.5%	11.8%
Controls	8.1%	1.8%	0.5%	1.2%	4.2%

Utilizing a large population sample, this study identified clear PD-like symptoms and signs among drug addicts. We postulate that addiction may have a neurotoxic impact on the striatal dopamine system, which is known to be involved in brain motor functional control. These symptoms and signs are unlikely to be due to withdrawal syndrome, as all addicted subjects had been abstinent for more than three months prior to the study, and abstinence time did not significantly contribute to UPDRS scores. Tremor and bradykinesia, which are chief symptoms of PD, appear to be predominant complications of drug addiction. However, evidence of morphological and biochemical brain tissue changes due to drug exposure is lacking in earlier clinical investigations and the current study. Therefore, further functional imaging studies are needed to explore the nature of changes in the brain motor cortex and associated neuronal pathways, as well as in subcortical structures, particularly the basal ganglia and cerebellum.

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Conflict of Interest

No conflict of interest.

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