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Atypical psychiatric symptoms in the presence of Huntington disease

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Abstract

Huntington Disease (HD) is an inherited neurodegenerative disorder characterized by choreiform movements, psychiatric symptoms, and dementia. There is a subset of common psychiatric symptoms seen in HD that has been outlined in the literature, preceding the onset of motor dysfunction. However, we present a patient whose illness demonstrated unique psychiatric symptoms, warranting further exploration on the relationship between substance use and HD.

Introduction

The pathophysiology of Huntington disease (HD), an autosomal dominant, slowly progressive neurodegenerative disorder characterized by psychiatric, cognitive, and motor symptoms, is related to toxicity of the mutant huntingtin protein caused by CAG trinucleotide repeats on chromosome 4 [1]. Progression occurs gradually along a continuum but in expected stages. Typically, psychiatric symptoms (irritability, disinhibition, depression) present prior to motor symptoms (chorea, imbalance), and the individual eventually is unable to perform activities of daily living [1].

While the prevalence of depression, irritability, apathy, anxiety, and suicide is common in HD patients, less frequent symptoms can exist, such as obsessive-compulsive behavior and psychosis [2]. Even less commonly documented are cases of new-onset substance use disorders in HD. While some studies suggested an increased incident of alcohol use in HD due to self-medicating or lack of impulse control, others report an incidence of alcohol use comparable to the general population [3].

Here, we outline a case in which a patient with HD presents with new-onset stimulant use disorder after initial psychiatric symptoms of HD presented.

Case

Mr. C, A 48-year-old Caucasian male, was admitted to the hospital after being found incompetent to stand trial on charges of possession of illegal substances resulting from major neurocognitive disorder due to Huntington disease. Ten years prior, this hospital admitted Mr. C voluntarily for suicidal ideation and recent suicide attempts of varying methods. He exhibited signs of depression without evidence of involuntary movements or cognitive decline. He cited a family history of HD and related suicides. Mr. C began citalopram and discharged ten days later

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to receive psychiatric care as an outpatient. He did not yet carry a diagnosis of HD, but the psychiatrist suggested genetic testing as an outpatient given his family history.

On the most recent admission to the hospital, Mr. C's presentation differed drastically. Though he exhibited occasional irritability disproportionate to the situation, Mr. C's affect was primarily euthymic. He was alert but oriented to person only, not fully comprehending his reason for or place of admission. He denied symptoms of depression, mania, or psychosis. Notably, he also denied past or present suicidal or homicidal ideations, seemingly forgetting his prior attempts ten years prior. Mr. C evidenced impairments in receptive and expressive language, complex attention, executive function, memory, and social cognition. In addition to these cognitive symptoms, he presented with significant choreiform movements diffusely and gait disturbance, impairing his activities of daily living function. According to his report and record review, he was formally diagnosed with HD five years prior to the second admission with confirmatory genetic testing.

Regarding substance use, Mr. C first began using methamphetamine approximately five years prior. Secondary to his recent and persistent use, Mr. C was arrested multiple times for charges related to methamphetamine since that time. Given his presentation, Mr. C received diagnoses of major neurocognitive disorder secondary to Huntington disease and methamphetamine use disorder.

Mr. C's presentation of HD appeared to be rather typical, with depression and suicidality preceding motor symptoms, but the treatment team noted the new onset of methamphetamine use after the onset of HD symptoms to be a unique aspect to his presentation.

Discussion

We present a case of persistent methamphetamine use occurring after onset of psychiatric HD symptomatology, potentially hastening the onset of motor symptoms. Some studies indicate an increased incidence of alcohol use in HD due to self-medicating or impulsivity, yet other studies report the incidence comparable to controls [4]. In HD, regional thinning of the cerebral cortex is present [5], underlying the clinical heterogeneity of the disease and explaining addictive behaviors through executive function disinhibition [6]. This idea supports an increased incidence of illicit substance use in HD resulting from poor impulse control.

Drug abuse alone causes neurotoxicity by effecting brain-derived neurotrophic factor and calcium signaling, which are also involved in the neurodegenerative process of HD [4]. When controlling for CAG trinucleotide repeat length, illicit substances hasten the onset of motor symptoms in HD by 3.3 years [7] likely by this neurotoxic mechanism [4]. Furthermore, amphetamines increase dopamine in the nucleus accumbens and caudate, also prominently affected in HD, leading to another possibly explanation for hastened onset of motor symptoms [7]. Our patient's presentation reflects this suggested mechanism.

Ultimately, further research is needed to better explain both the effects of substance use (particularly methamphetamine) on HD as well as if illicit substance abuse occurs secondary to the pathophysiology of HD.

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