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Case Study

A Case Series of Delusional Infestation in Huntington's Disease

Wenxin Song[a,*], Lauren Daneman[b], Alexis Cohen-Oram[b] and Stephen Aradi[b]
[a]*USF Health Morsani College of Medicine, Tampa, FL, USA*
[b]*University of South Florida, Tampa, FL, USA*

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Abstract. Huntington's disease (HD) is an autosomal dominant disorder that affects the basal ganglia, caused by CAG repeats in the huntingtin gene. Delusional infestation (DI) is a rare psychotic manifestation of the disease. This report presents two cases of HD patients with DI, both middle-aged females. The first patient achieved remission of DI with olanzapine, later cross-tapered to risperidone, but had spontaneous relapses. The second experienced gradual resolution of DI with risperidone in the setting of iron repletion and amantadine discontinuation, although her other psychotic symptoms remained. These cases shed light on an uncommon condition and may help guide understanding of the most effective treatment for it.

Keywords: Huntington's disease, delusion, infestation, parasitosis, psychiatry, neurology

INTRODUCTION

Huntington's disease (HD) is an autosomal dominant disorder caused by CAG repeat expansion in the huntingtin gene. HD affects the basal ganglia, impairing motor function and causing chorea. Neuropsychiatric symptoms include apathy, depression, and irritability [1].

Psychotic symptoms appear in about 17% of HD patients and are associated with poorer cognitive outcomes [2]. The most common psychotic symptom in HD is paranoia; hallucinations and delusions are less common [3]. Current data on delusional infestation in the context of Huntington's disease is limited to single cases or small sample sizes.

Delusional infestation (DI), the persistent belief of skin-dwelling bugs/creatures, lacks a clear etiology. Propositions include dysfunction of the striatal dopamine transporter [4] or variations in cortical thickness involving perception and visuospatial awareness [5]. There is no consensus on a standardized treatment [6] but pharmacological intervention may increase symptom remission [7]. The first-line therapy is second-generation antipsychotics, mainly risperidone and olanzapine [6].

In our local HD clinic, we recently treated two patients with genetically confirmed Huntington's disease and delusional parasitosis.

CASE 1

Ms. K, a white female, first experienced mild and non-impairing chorea in her mid-40 s, exacerbated under emotional stress. Genetic testing showed 42 CAG repeats in the huntingtin gene. Five years later, she presented to neurology with cognitive decline, depression, and anxiety and began to follow up regularly. She was initially on doxepin 100 mg at bedtime and a 9 : 1 CBD:THC gel, both for sleep. Doxepin was initially effective but was later discontinued,

*Correspondence to: Wenxin Song, BS, USF MCOM, 560 Channelside Dr, Tampa, FL 33602, USA. Tel.: +1 617 955 6652; E-mail: songw@usf.edu.

due to recurrence of poor sleep and dizziness, and replaced with nortriptyline 25 mg. She continued to take the 9 : 1 CBD:THC gel nightly for sleep. She first reported delusions of infestation shortly after a resolved pest infestation in her home. Nortriptyline was discontinued and she was started on quetiapine. Despite this, her delusions continued to worsen in intensity and frequency. At follow up visits, she was preoccupied with the delusions and reported anxiety, diffuse pruritis, dyspnea, nausea and insomnia due to the infestation. Quetiapine was discontinued, and she was started on olanzapine 5 mg. The CBD:THC gel was replaced with 100% CBD gel. Due to ongoing problems with insomnia, doxepin 25 mg at bedtime was added. She began to report that she was unable to exercise and was not engaging in her usual social due to the infestation. Olanzapine was slowly increased to 40 mg daily with partial improvement after each increase. She had no side effects, including extrapyramidal, and continued reporting significant insomnia.

Notably, there was an 8-month period where she reported the infestation had resolved and resumed her previous activities. She then spontaneously became delusional again and informed the team that she had lied, the infestation had never actually gotten better. Olanzapine was cross tapered to risperidone, which was slowly increased to 8 mg total per day with partial improvement. She had a 3-month period where she reported resolution of the infestation, and then after 3 months became overtly delusional, saying she had been lying to her providers. Insomnia remained a significant complaint, and she informed providers she was taking risperidone, doxepin, hydroxyzine, and mirtazapine from various providers for sleep. On recommendation, she discontinued hydroxyzine and doxepin. On follow up, she reported resolution of the infestation.

CASE 2

Ms. M, a 38-year-old female with IgA vasculitis and Hashimoto's, was evaluated two years after HD symptom onset. At that visit, she had chorea, irritability, low mood, and cognitive problems meeting criteria for major neurocognitive disorder. Initial treatment involved sertraline and valproic acid, leading to improved irritability and mood. Carbidopa/levodopa was prescribed for chorea. A year later, it was replaced with risperidone 0.5 mg BID for chorea/mood.

Over six months, amantadine was added for chorea, doxepin for sleep, and risperidone was increased to 1.5 mg BID. After 3 weeks, Ms. M reported several months of sensations of bugs crawling over her body at night. She maintained insight into the symptoms and denied illusions. She worsened over 3 months, finally presenting to the ED for severe anxiety, reporting bugs on her head. As an outpatient, risperidone was slowly increased to 2 mg BID. She was tapered off of amantadine and prescribed a benzodiazepine PRN for anxiety.

Around the same time, iron deficiency anemia was detected and iron was repleted. At the next visit, patient reported resolved crawling sensations and no concern about an infestation. Family continued to report behaviors related to bug concerns, and risperidone was increased to 6 mg total per day. At follow up about a month later, patient denied concerns related to bugs or crawling sensations on skin, although her spouse reported sporadic comments about bug concerns. In the next 9 months, Neurology and Neuropsychiatry notes indicate a variety of problems related to progression of HD, but no recurrence of DI is noted.

DISCUSSION

These two cases illustrate the presence of delusional parasitosis in HD patients. Both were seen at the same HD-specialized outpatient clinic within 2-3 years, suggesting that delusional parasitosis might be more common in HD patients than previously thought. Some aspects align with general literature on DI. For example, Ms. K and Ms. M were both middle-aged females who responded to atypical antipsychotics. Unfortunately, response was limited or non-sustained.

Ms. K achieved remission on 4 mg of risperidone but had symptom recurrence 4 months later. One explanation may be progressive neurodegeneration due to HD. Ms. M did not respond to high-dose risperidone initially, but her DI symptoms eventually resolved. It is unclear whether discontinuing amantadine and supplementing iron attributed to this, as iron deficiency can cause skin crawling sensations. Interestingly, her other psychotic and affective symptoms did not resolve.

In summary, DI is an uncommon but impairing condition in HD patients. The most effective treatment is unknown as there are few documented cases.

Treatment remains informed by our understanding of DI in patients without comorbid HD and further information is needed to determine if patients would be better served by a different therapeutic approach.

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CONFLICT OF INTEREST

The authors have no conflict of interest to report.

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