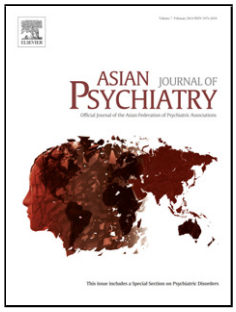

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Conversion Parkinson's Disease with Levodopa Abuse and Psychosis

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Keywords:Parkinson's; Dependence; Conversion Parkinson's Disorder; Levodopa; Factitious Disorder.

Conversion disorder (CD) now recognised as functional neurological symptom disorder (DSM V) presents with physical symptoms that are not well explained by organic aetiology. We describe a unique case of Conversion Parkinson's Disease (PD).

Ms D is a 34-year-old single woman. The salient points in her background included physical and emotional abuse by her father. She was an anxious child with tendency to somatise distress. She developed chronic unexplained fatigue with muscle aches that led to a diagnosis of fibromyalgia by a rheumatologist when she was 24. There is also a family history of bipolar disorder in her mother and schizophrenia in in second and third degree relatives. Her personality style was characterised by borderline and dependant traits, with a history of affective dysregulation and defence mechanisms of idealization and devaluation, splitting and projection evident during her hospitalisation.

She met her recently deceased partner, who was 30 years older when she was 27. He was the leader of a religious cult group whom she strongly idealised as a paternalistic figure. Her partner was diagnosed with PD and treated with L-dopa. In the context of being with a charismatic and persuasive individual, Ms D started developing somatic symptoms including tremors, unsteady gait, varying degrees of paralysis and neuropathic pain, which he attributed to PD and encouraged her to use his L-dopa. She had no prior history of levodopa-induced euphoria or psychosis. With self-reporting of unconfirmed PD, Ms D procured constant supply of L-dopa through various GPs. She was taking Levodopa-Benserazide (Madopar)200mg-50mg capsules every 6 hours and 4mg transdermal Rotigotine patches. She gradually increased the dose over five years and rationalised the dose increases as preventing withdrawal symptoms like spasms, agitation and fatigue.

She presented to local Emergency Department following an acute conscious collapse. No medical cause could be identified for her collapse. Persecutory delusions and auditory hallucinations were noted during her medical admission, requiring transfer to a psychiatric ward. She was diagnosed with L-dopa induced psychosis. Her atypical presentation of PD was formulated to be a conversion disorder with predominantly unconscious motivation and symptom production, on the background of emotional vulnerability secondary to her early trauma history, past history of somatisation and borderline/dependant traits. Her unique parkinsonian presentation was speculated to be influenced by the symptomatic template of her partner with PD, whom she idealised as a father figure.

Following review by a neurologist, PD was excluded and Dopamine Replacement Therapy (DRT) was ceased, which caused distress. Subsequent psychiatric review offered her an alternative explanation of her symptoms, which she found acceptable. Ms D was prescribed Aripiprazole, and psychosis resolved within a week. Following cessation of L-dopa, her non-apparent parkinsonian features further substantiated our formulation of conversion PD.

This is the first reported case of Conversion PD with a previous report where the patient embellished her existing PD symptoms in order to obtain a higher dose of L-dopa and gain was to obtain the sick role[1]. It is interesting that despite the higher prevalence of PD in comparison to epilepsy[2,3], the former provides a more common symptomatic template for abnormal illness behaviour.

L-dopa has intrinsic reinforcement properties and is closely linked to the reward pathways of the brain[4]. It is a drug that stimulates both direct and indirect pathways in the striatum, therefore has high dependence potential. Its abuse in PD patients is a well-recognized phenomenon and has been referred to as hedonistic homeostatic dysregulation[5] and dopamine dysregulation syndrome by various authors to avoid the stigmatizing label of addiction[6]. Its prevalence in PD is thought to be around 4%[7] in tertiary care setting. It has been discussed and debated whether it can actually be a drug of abuse given its dependence is largely

iatrogenic rather than recreational[8.] There have only been a few reports of L-Dopa abuse in non-PD setting which is rather surprising, given its potential addictive properties[9], and might indicate the complexities that underlie that neurobiology of addiction.

L-Dopa induced psychosis in the setting of PD is common with an incidence of 22%[10]. It is because while trying to stabilize the nigrostriatal dopaminergic system, levodopa destabilizes the mesolimbic system. It usually resolves following cessation of the drug but sometimes requires the addition of an antipsychotic medication if L-Dopa is unable to be ceased.

Whilst the addiction construct and L-dopa abuse is an area of controversy in PD, clinical suspicion should be raised in young onset PD with atypical features. Specialist neurologist advice should be sought before consideration of L-dopa therapy and screening for psychological factors be done in such cases. Despite the reinforcing effect of L-dopa, it is surprising that there have not been many cases of recreational abuse of this drug, and it does not form part of the restricted formulary. The authors believe that it is under-reported, which is worthy of further clinical and biological research.

Conflict of interest

The authors declare that there is no conflict of interest. The authors have not received any funding for this manuscript. No part of the manuscript has been published elsewhere.

(799 words)

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